RESEARCH ARTICLE

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Helicobacter pylori Infection Promotes Gastric Premalignancies and Malignancies Lesions and Demotes Hyperplastic Polyps: A 5 Year Multicentric Study among Cameroonian Dyspeptic Patients

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Abstract

Background: *Helicobacter pylori* infection is the most well-known risk factor for gastric mucosa abnormalities. However, some geographic regions with persistent high *H. pylori* infection rates do not suffer from high gastric mucosa lesions incidence. The aim of the study was to establish the relationship between *H. pylori* infection and gastric pathological features in Cameroon. **Methods:** We performed a retrospective study, collecting data from the University Teaching Hospital and the Cameroon Pasteur institute on 1290 patients (mean age 46.31 ± 16.45 years, sex ratio 1.19:1) for whom histological features of the gastric mucosa and *H. pylori* infection were investigated from 2014 to 2019. Data were extracted from the medical records; hospital computerized databases; or clinical charts of these patients and reviewed according to gender and age of participants. The study was approved by the Ethical Committee of Medical Sciences. **Result:** Approximately 3% (2.56%) of the sample population were with normal gastric mucosa whereas chronic gastritis, atrophic gastritis, intestinal metaplasia, dysplasia, carcinoma, hyperplastic polyps and MALT lymphoma was found in 75.35, 8.2, 7.7, 2.8, 9.3, 1.55 and 0.8% of cases respectively. Unlike hyperplasia (OR= 0.3838), infected participants were in a high risk to develop gastric lesions with an odds ratio of 1.1775, 1.4866, 1.4415, 1.2088, 0.9408 and 0.9075 for gastritis, atrophic gastric, dysplasia, carcinoma, intestinal metaplasia and MALT lymphoma respectively. **Conclusion:** our finding showed that chronic gastritis, gastric premalignancies and malignancies are positively link to *Helicobacter pylori* infection and that hyperplastic polyp is inversely associated with *H. pylori* infection in our milieu.

Keywords: *H pylori* infection- Gastric precancerous and cancerous lesions- Cameroon.

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Introduction

Helicobacter pylori is a gram negative, spiral shaped micro-organism that inhabits and colonizes the harsh acidic environment of the gastric mucosa. More than 50% of the world's wide population is chronically infected by this bacterium. However, a great discrepancy exists in the prevalence of *H. pylori* worldwide; 20 to 50% of the population seems to be infected in the developed countries and up to 90% in the developing countries (Alkout et al., 2000). This steep decline of *H. pylori* infection between developed countries and developing ones is mainly due to the improvement in personal hygiene and community sanitation that prevent re-infection, since the prevalence of this infection varies according to the socioeconomic conditions and the living standard of the populations. This disparity may also be due to the widespread use of *H. pylori* eradication therapy that reduced the prevalence of this infection in developed countries.

Infection due to this bacterium can be life-long in the host unless it is eradicated (Amieva and El-Omar, 2008). Although 85% of infected patients have only mild asymptomatic gastritis, gastric colonization with this bacterium in some cases can cause several gastrointestinal diseases such as, chronic gastritis, gastro-duodenal ulcers and gastric malignancies. It is reported that 15% of *H. pylori* infected patients can develop peptic ulcer disease (PUD) and less than 1% can develop gastric cancer (Amieva and El-Omar, 2008; Niyaz and Leonardo, 2005). Many epidemiological surveys conducted in Africa where incidence of infection is higher have revealed that 70–90% of duodenal and gastric ulcer cases is due to this bacterium (Alkout et al., 2000; Eyoum et al., 2020).There are two form of gastric malignancies or neoplasia; adenocarcinomas which represents 95% of the stomach cancers and Mucosa-associated lymphoid tissue lymphoma (MALT lymphoma) which accounts for up to 3 % (Freeman et al., 1972). Both the

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two form of gastric neoplasia are related to *H. pylori* infection. In fact, *Helicobacter pylori* provokes a local inflammation in almost all host, a continuous process which increases the risk of developing atrophic gastritis, intestinal metaplasia, dysplasia and non cardia gastric adenocarcinoma (Correa, 2004). On the other hands, long time gastric colonization by *H. pylori* induce local humoral and T-cell response in the gastric mucosa, this persistent stimulation of activated B cells by T cells lead to the development of lymphoid follicles which predisposes the development of MALT lymphoma (Katharina et al., 2015). Hyperplastic polyps (HPs) result from hyper-regenerative epithelium in response to an underlying chronic inflammatory stimulus (Carmack et al., 2009). It account for approximately 75 percent of gastric polyps in geographic areas where *H. pylori* is common. Over time, polyps may remain stable, increase in size, or regress following *H. pylori* eradication (Ljubicic et al., 1999). Polyps have malignant potential, malignancy develops in hyperplastic polyps through a dysplasia/carcinoma sequence (Ahm et al., 2014). Between 1 and 20 percent of hyperplastic polyps have been reported to harbor foci of dysplasia (Ahm et al., 2014). The risk of malignancy in hyperplastic polyps is increased in polyps >1 cm (Ahm et al., 2014). Globally, gastric cancer is the leading cause of infection-associated cancers. According to GLOBOCAN 2018 data, gastric cancer is the third leading cause of cancer deaths worldwide, following only lung and colorectal cancer in overall mortality. About 1 in 12 of all oncological deaths is attributable to gastric cancer. Gastric cancer has the fifth highest incidence among cancers, with 5.7% of all new cases attributable to the disease and over a million new cases of gastric cancer are diagnosed, worldwide each year (Bray et al., 2018).

Several diagnostic methods have been developed in order to detect *Helicobacter pylori* accurately. These tests include non-invasive methods and invasive methods which require upper gastrointestinal endoscopy to obtain gastric biopsy samples (Kouitcheu et al., 2020). Among these, histological examination is one of the most useful invasive diagnostic tests for *H. pylori* infection (Kouitcheu et al., 2020). Histology plays a pivotal role in detecting *H. pylori* and it also provides more information about the degree of inflammation and associated pathologies, such as atrophic gastritis (AG), intestinal metaplasia (IM), MALT lymphoma, hyperplasia and gastric cancer.

As expected in developing country, Cameroon is an endemic area for *H. pylori* infection. Prevalence of infection ranges from 92.2% among apparently healthy children in the Southern region in 2004 to 64.34% among patients with gastro-duodenal disorders aged 35 years and above in the Littoral region in 2018 (Kouitcheu et al., 2018). All these previous studies on *H. pylori* prevalence in Cameroon were investigated on a specific age group and used a non-invasive test which is not reliable for a current infection. Moreover, studies concerning the simultaneous evaluation of *H. pylori* colonisation assayed histologically (an accurate invasive diagnostic test for current *H. pylori* infection) in association to gastric histopathological features such as hyperplastic polyps, MALT lymphoma, precancerous and cancerous lesions among population are not available in the country to our knowledge. Therefore, this study was investigated in a Cameroonian population characterised by its high incidence of *H. pylori* infection in order to visualise the morbidity associated with this bacterium.

Materials and Methods

Setting and study design

From March to May 2019, we performed a multicentre retrospective study based on gastric endoscopy biopsy data collected from 2014 to 2019 at the anatomo-cytopathology unit of two reference health facilities located in Yaoundé, the Centre region of Cameroon; the University Teaching Hospital (CHU) and the Cameroon Pasteur institute (CPC). The University Teaching Hospital is a tertiary hospital. It is a large teaching hospital which offers full and high-complexity service serving around 2,765,600 people from its catchment area. Its anatomo cytopathology unit receives patients referred from other hospitals in Yaoundé and also from different parts of the country. The Cameroon Pasteur Institute is a reference clinical and research Centre of the country. It has the highest technical platform that can support all types of clinical analysis. Its anatomo cytopathology laboratory receives and analyses clinical samples collected for oncology analysis all around the country, because adequate technical platform for oncology analysis are not widely available in Cameroon.

Participants and Baseline assessment

The study involved the analysis of existing clinical and laboratory data from all consecutive patients with dyspepsia whose gastric biopsies samples were brought for examination at the anatomo-cytopathology unit of either CPC or CHU from 2014 to 2019. Data were extracted from the medical records; hospital computerized databases; or clinical charts of the patients and reviewed according to a pre-established protocol including the following demographics variables (sex and age). The biopsy specimens were usually taken from the antrum and other locations if required. Biopsy sample from patient with more than one biopsy specimen collected were mixed before processing, so one biopsy sample or mixed biopsy sample were analyzed per participant and the number of biopsy specimen analyzed was equal to the number of participants. Only patients for whom histological features of the gastric mucosa and *H. pylori* were investigated were included in the study. All biopsies samples were analyzed by a dedicated anatomo pathologist in the selected health facilities. Samples were fixed overnight in a 10% buffered formaldehyde solution, processed, embedded in paraffin, cut and the slides were stained by the Hematoxylin-Eosin (HE) method for histological features detection. Histological features of the gastric mucosa included: normal gastric mucosa that is gastric mucosa without injuries, chronic inflammation, inflammatory activity, gastric atrophy, intestinal metaplasia, dysplasia, hyperplasia polyps, adenocarcinoma and MALT lymphoma. All these variables were classified as absent or present, and when present the intensity of inflammation and the neutrophil activity were ranked as mild, moderate

or severe (Price, 1991). Chronic inflammation was considered when high level of mononuclear leucocytes, including lymphocytes, plasmocytes and macrophages was found. Inflammatory activity was confirmed by the detection of polymorphonuclear cells (neutrophils) in the lamina propria, epithelium or lumen. *H. pylori* infection was established though histology examination. Participants were considered *H. pylori* positive if the bacteria could be detected histologically in any of the Giemsa-stained slides.

Ethical consideration

This study was approved by the Institutional Review Board of University Teaching Hospital (Approval no 703/AR/CHUY/DG/DGA/CAPRC) and Cameroon Pasteur institute CPC (Approval no 000378/2019/CPC/DG/DAF/SRH) and the Ethical Committee of Medical Sciences for the Centre Region of Cameroon (Approval no 0438 / CRERSHC/ 2019).

Statistical analysis

The data collected throughout this study was coded before being entered in the database and analysed using the SPSS 20 software package. The frequency of each histological feature of the gastric mucosa according to *H. pylori* infection was determined and compared to that of normal mucosa taken as the reference group. Differences in the distribution of these variables according to sociodemographic parameters were assessed by the chi-square test and those with value $P < 0.05$ were considered statistically significant. Odds ratios (OR) and 95% confidence intervals (95% CI) were calculated for each gastric lesions in relation to the presence of *H. pylori* infection using univariable and multivariable logistic regression.

Results

Characteristic of the study population according to H. pylori infection

A total of 1290 dyspeptic participants were enrolled in this study; 158 in 2014, 201 in 2015, 188 in 2016, 360 in 2017 and 383 in 2018. Among these participants, 1123 were from the CPC and 167 from the UHC. Their mean age was 46.31 ± 16.45 years (range 08 – 89 years), and the age groups from 20 to 39 (33.10%, 427/1290) and

Table 1. Characteristic and Distribution of H. pylori Infection According to Age and Sex of Participants

Variables	Number (%)	H. pylori positive (%)	H. pylori negative (%)	X^2, (P value)
Gender, Sex ratio: 1.19/1				
Female	702	309 (45.71)	279 (45.44)	0.9554
Male	588	367 (54.29)	335 (54.56)	
Total (%)	1290	676 (52.40)	614 (47.6)	
Age (years), (Mean ±SD)	46.31 ± 16.45)	48.46 ± 4.31	49.15 ± 4.07	
≤20	55	29 (4.28)	26 (4.23)	0.4664, (0.9260)
20-39	425	221 (32.69)	204 (33.22)	
40-59	484	250 (36.98)	234 (38.11)	
>60	326	176 (26.03)	150 (24.42)	
Total (%)	1290	676 (52.40)	614 (47.6)	

n, number; SD, Standard deviation; X^2, Chi-square

Table 2. Gastric Histological Features According to Age and Gender in the Sample Population

Variables	Histological features								R square (P value)
	Nor (%) n=33	CSG (%) n=972	AG (%) n=106	IM (%) n=100	DYS (%) n=36	GC (%) n=120	MALT (%) n= 10	HPs (%) n=20	
Sex									
Female	18 (54.5)	523 (53.8)	62 (58.5)	60 (60)	14 (38.8)	75 (62.5)	2 (20.0)	10 (50)	
Male	15 (45.5)	449 (46.2)	44 (41.5)	40 (40)	22 (61.2)	45 (37.5)	8 (80.0)	10 (50)	
Total number (Prevalence)	33 (2.56)	972 (75.35)	106 (8.22)	100 (7.75)	36 (2.79)	120 (9.30)	10 (0.77)	20 (1.55)	
Age (years) Mean ± SD	40.17± 9.59	41.73±15.35	46.57±16.33	54.34±15.69	55.77± 19.51	56.55 ± 16.61	47.13± 19.81	52.25±19.65	0.012 (p=0.5879)
≤20	2 (6.1)	45 (4.6)	04 (3.8)	4 (4.0)	1 (2.8)	2 (1.7)	1 (10.0)	1 (5.0)	
21-39	17 (51.5)	340 (35)	32 (30.2)	16 (16.0)	8 (22.2)	32 (26.7)	2 (20.0)	5 (25.0)	
40-59	8 (24.2)	414 (42.6)	47 (44.3)	45 (45.0)	11 (30.5)	40 (33.3)	4 (40.0)	5 (25.0)	
≥60	6 (18.2)	173 (17.8)	23 (21.7)	35 (35.0)	16 (44.5)	46 (38.3)	3 (30.0)	9 (45.0)	
Total number (Prevalence)	33 (2.56)	972 (75.35)	106 (8.22)	100 (7.75)	36 (2.79)	120 (9.30)	10 (0.77)	20 (1.55)	

Nor, Normal mucosa; CSG, Chronic gastritis; AG, Atrophic Gastritis; IM, Intestinal metaplasia; DYS, Dysplasia; GC, Gastric cancer; MALT, MALT lymphoma; HPs, Hyperplasia.

Table 3. Histological Features of the Gastric Lining According to H. pylori Status among the Study Population Taken Subjects with Normal Gastric Lining as Reference

Histological features	Number	H. pylori positive (%)	H. pylori negative (%)	OR (IC 95)	P value
Normal	33	03 (9.0)	30 (91.0)	Reference	
Chronic gastritis	972	519 (53.4)	452 (46.6)	0.08709 (0.02775-0.259)	P< 0.0001*
Atrophic gastritis	106	65 (61.3)	41 (38.7)	0.06308 (0.01956-0.1996)	P< 0.0001*
Intestinal metaplasia	100	51 (51.0)	49 (49.0)	0.09601 (0.02967-0.3355)	P< 0.0001*
Dysplasia	36	22 (61.1)	14 (38.9)	0.06364 (0.01857-0.2355)	P< 0.0001*
Gastric cancer	120	68 (56.7)	52 (43.3)	0.07647 (0.02378-0.2599)	P< 0.0001*
MALT lymphoma	10	5 (50.0)	5 (50.0)	0.01 (0.02273-0.6297)	P=0.0103*
Hyperplasia	20	06 (30.0)	14 (70.0)	0.2333 (0.05909-0.947)	P=0.0666

OR, odd ratio; (95% CI), 95% confidence intervals. Statistical test with subjects with normal mucosa as the reference group. *, Significant.

from 40 to 59 (37.52%, 484/1290) years old were the most represented. Fifty four percent (54.42%, 702/1290) versus 45.58% (588/1290) of participants were females, given a sex ratio of 1.19:1 (Table 1).

Among the overall 1290 dyspeptic patients enrolled in this study, 89 in 2014, 119 in 2015, 105 in 2016, 202 in 2017 and 161 in 2018 were *H. pylori* infected, giving an infection prevalence of 56.33%, 59.20%, 55.85%, 56.11% and 42.04% respectively from 2014 to 2018, and an overall prevalence of 52.40% in our sample population. Their mean age was 48.46 ± 4.31 years and 49.15 ± 4.07 for infected and non-infected patients respectively and the age group 40 to 59 (36.98%) was the most affected. According to the gender, females were more affected by *H. pylori* infection than males (54.29 versus 47.71%) (Table 1).

Frequency of gastric histological features among the study population

One thousand two hundred and ninety (1290) biopsy sample or mixed biopsy sample were collected from the overall dyspeptic participants recruited and examined for histological features. Thirty three (33) of these gastric specimen showed normal mucosa, given a proportion of participant with gastric mucosa without injury of 2.56% (33/1290) among our sample population. The mean age of participant showing normal gastric mucosa was 40.17 ± 9.59 years with 20 to 39 years old (51.5%) as the age group with the highest incidence and female was the predominant gender (54.5%). The remaining 97.44% (1257/1290) of our sample population presented histopathological features in their gastric mucosa (Table 2).

Chronic gastritis predominated and was found in 972 (75.35%) cases. The average age of patients with

Table 4. Frequency and Intensity of Inflammatory Infiltrate and Neutrophil Activity According to H. pylori Status among the Study Population

Variables	Number (%)	H. pylori positive (%)	H. pylori negative (%)	X^2 (p value)
Inflammatory infiltrate				
Yes	914 (94.03)	475 (91.34)	439 (97.12)	0.0002*
No	58 (5.97)	45 (8.66)	13 (2.88)	
Total n(%)	972 (75.35)	520 (53.50)	452 (46.50)	
Intensity of the inflammatory infiltrate				
Mild	419 (43.10)	184 (38.7)	235 (53.5)	20.12 (< 0. 0001)*
Moderate	32 (3.29)	19 (4.00)	13 (2.96)	
Severe	463 (47.63)	272 (57.3)	191 (43.51)	
Total n (%)	914 (94.03)	475 (51.96)	439 (48.04)	
Neutrophil activity				
Yes	352 (64.94)	212 (74.38)	140 (54.47)	(< 0.0001)*
No	190 (35.05)	73 (25.61)	117 (45.52)	
Total n (%)	542 (42.01)	285 (52.58)	257 (47.42)	
Intensity of neutrophil activity				
Mild	257 (47.42)	148 (57.59)	109 (42.41)	3.22
Moderate	86 (15.87)	57 (66.28)	29 (33.72)	
Severe	9 (1.66)	07 (77.77)	02 (22.22)	-0.1999
Total n (%)	352	212 (60.22)	140 (39.77)	

n, Number; X^2, Chi-square

Table 5. Histological Features Adjusted to Socio-demographic Factors and H. pylori Status among the Study Population using Univariate and Multivariate Logistic Regression Analysis

Variable	Total	Present	Absent	Univariate logistic regression		Multivariate logistic regression	
				OR (95% CI)	p value	OR (95% CI)	p value
Normal							
Age ≥ 20							
Yes	55	2 (6.06)	53 (4.22)	0.6808	0.6045	0.5771	0.4682
No	1235	31 (93.94)	1204 (95.78)	(0.1589- 2.9162)		(0.1307- 2.5487)	
Gender							
Female	702	18 (54.55)	684 (54.42)	0.9948	0.9882	0.9932	0.9849
Male	588	15 (45.45)	573 (45.58)	(0.4969- 1.9915)		(0.4920- 2.0051)	
H. pylori status							
Yes	676	3 (9.09)	673 (53.54)	0.0869	0.0001*	0.086	0.0001*
No	614	30 (90.91)	584 (46.46)	(0.0264- 0.2859)		(0.0261- 0.2833)	
Chronic gastritis							
Age ≥ 20							
Yes	55	45 (81.82)	10 (18.18)	0.6684	0.2574	0.6792	0.2772
No	1235	927 (75.04)	308 (24.96)	(0.3329-1.3422)		(0.3380-1.3648)	
Gender							
Female	702	523 (74.5)	179 (25.5)	11.031 (0.8548-1.4235)	0.4507	10.995	0.4662
Male	588	449 (76.32)	139 (23.68)			(0.8518-1.4193)	
H. pylori status							
Yes	676	519 (76.78)	157 (23.22)	11.775 (0.9139 -1.5171)	0.2064	10.995	0.4662
No	613	452 (73.74)	161 (26.26)			(0.8518 -1.4193)	
Atrophic gastritis							
Age ≥ 20							
Yes	55	4 (3.77)	51 (4.31)	11.478	0.7946	11.793	0.7559
No	1235	102 (96.23)	1133 (95.69)	(0.4067- 3.2398)		(0.4169- 3.3359)	
Gender							
Female	702	62 (58.49)	640 (54.05)	0.8349	0.3801	0.8331	0.375
Male	588	44 (41.51)	544 (45.95)	(0.5580- 1.2492)		(0.5565-1.2472)	
H. pylori status							
Yes	676	65 (61.32)	611 (51.60)	14.866	0.0563*	14.918	0.0544*
No	614	41 (38.68)	573 (48.40)	(0.9894- 2.2337)		(0.9925- 2.2422)	
Intestinal metaplasia							
Age ≥ 20							
Yes	55	4 (4.00)	51 (4.29)	10.725(0.3799-3.0281)	0.8948	10.596	0.9131
No	1235	96 (96,00)	1139 (95.71)			(0.3745-2.9979)	
Gender							
Female	702	60 (60.00)	642 (53.95)	0.781	0.2443	0.7816	0.2457
Male	588	40 (40.00)	548 (46.05)	(0.5152-1.1840)		(0.5156-1.1848)	
H. pylori status							
Yes	676	51 (51.00)	625 (52.52)	0.9408	0.7697	0.9429	0.7781
No	614	49 (49.00)	565 (47.48)	(0.6255-1.4151)		(0.6266-1.4190)	
Dysplasia							
Age ≥ 20							
Yes	55	1 (2.78)	54 (4.31)	15.703	0.6589	28.19	0.6271
No	1235	35 (97.22)	1200 (95.69)	(0.2118-11.6444)		(0.6777-11.7263)	

Table 5. Continued

Variable	Total	Present	Absent	Univariate logistic regression		Multivariate logistic regression	
				OR (95% CI)	p value	OR (95% CI)	p value
Dysplasia							
Gender							
Female	702	14 (38.89)	688 (54.86)	19.101	0.0618	0.6937	0.0644
Male	588	22 (61.11)	566 (45.14)	(0.9684-3.7675)		(0.4709-1.0221)	
H. pylori status							
Yes	676	22 (61.11)	654 (52.15)	14.415	0.2912	12.253	0.2945
No	614	14 (38.89)	600 (47.85)	(0.7309-2.8429)		(0.8381-1.7914)	
Gastric cancer							
Age ≥ 20							
Yes	55	2 (1.67)	53 (4.53)	27.969	0.1569	28.19	0.1542
No	1235	118 (98.33)	1117 (95.47)	(0.6734-11.6166)		(0.6777-11.7263)	
Gender							
Female	702	75 (62.50)	627 (53.59)	0.6929	0.0632	0.6937	0.0644
Male	588	45 (37.50)	543 (46.41)	(0.4705-1.0203)		(0.4709-1.0221)	
H. pylori status							
Yes	676	68 (56.67)	608 (51.97)	1.2088	0.3267	12.253	0.2945
No	614	52 (43.33)	562 (48.03)	(0.8276-1.7655)		(0.8381-1.7914)	
MALT lymphoma							
Age ≥ 20							
Yes	55	1 (10.00)	54 (4.22)	0.3964(0.0493- 3.1853)	0.3841	0.4136	0.4083
No	1235	9 (90.00)	1226 (95.78)			(0.0510-3.3518)	
Gender							
Female	702	2 (20.00)	700 (54.69)	48.198	0.0471*	47.914	0.0481*
Male	588	8 (80.00)	580 (45.31)	(1.0207-22.7606)		(1.0131-22.6620)	
H. pylori status							
Yes	676	5 (50.00)	671 (52.42)	0.9075	0.8784	0.8952	0.8619
No	614	5 (50.00)	609 (47.58)	(0.2616- 3.1476)		(0.2571- 3.1166)	
Hyperplasia							
Age ≥ 20							
Yes	55	1 (5.00)	54 (4.25)	0.8437	0.8696	0.7931	0.8234
No	1235	19 (95.00)	1216 (95.75)	(0.1109-6.4186)		(0.1035-6.0745)	
Gender							
Female	702	10 (50.00)	692 (54.49)	11.972	0.6896	12.007	0.6856
Male	588	10 (50.00)	578 (45.51)	(0.4949-2.8964)		(0.4954-2.9104)	
H. pylori status							
Yes	676	6 (30.00)	670 (52.76)	0.3838	0.0512*	0.3822	0.0503*
No	614	14 (70.00)	600 (47.24)	(0.1466-1.0051)		(0.1459-1.0013)	

N or n, number; OR, Odd ratio; (95% CI), 95% confidence intervals; *Significant.

chronic gastritis was 41.73± 15.35 years, and the most represented age of incidence of chronic gastritis was 40 to 59 years (42.6%) with female predominance (53.8%, 523/972) (Table 2). Nine hundred and fourteen (914) versus 58 patients had inflammatory infiltrate. Among inflammatory infiltrate, 419 (45.8%) were mild, 32 (3.6%) were moderate, and 463 (50.6%) case were markedly intense (Table 4). Neutrophil activity was noticed in 542 patients. Of these, 190 (35.05%) had no activity while 257 (47.42%), 86 (15.87%) and 9 (1.66) were with mild, moderate and markedly intense neutrophil activity respectively (Table 4).

For the premalignant and malignant lesions: the prevalence of atrophic gastritis was 8.2% (106/1290), prevalence of intestinal metaplasia was 7.7% (100/1290), prevalence of dysplasia was 2.8% (36/1290) and prevalence of carcinoma was 9.3% (120/1290). The average age of patients with atrophic gastritis, intestinal metaplasia, dysplasia and gastric carcinoma was 46.57 ± 16.33, 54.34 ±15.69, 55.77 ±19.51 and 56.55 ± 16.61 respectively compared with 40.17 ± 9.59 and 41.73 ± 15.35 for normal mucosa and chronic

gastritis. The most represented age of incidence was 40-59 years old for atrophic gastritis (47/106, 44.3%) and intestinal metaplasia (45/100, 45%), and up to 60 years old for dysplasia (16/36, 44.5%) and gastric carcinoma (46/120, 38.3%). As gender is concerned, females were predominantly affected among patients with atrophic gastritis, intestinal metaplasia and gastric carcinoma; male among those with dysplasia.

For the others histological lesions, the prevalence of hyperplastic polyps was 1.55% (20/1290) with 52.25 ±19.65 years and up to 60 years old respectively as the average age and the most represented age of incidence (9/20, 45.0%) among patients with these type of gastric lesion. Only ten (0.8%) cases of MALT lymphoma were noticed. Their average age was 47.13 ±19.81 years old with 40-59 years old as the most represented age of incidence for this lesion (4/10, 40.0%). Females were predominantly affected among patients with MALT lymphoma, while no difference between the two sexes was observed concerning those with hyperplasia (Table 2).

Frequency of histological features according to H. pylori infection

H. pylori was detected in 3 patients with normal gastric mucosa, given an infection rate of 9% (3/33) among patients with normal gastric mucosa. This difference was significant when comparing to *H. pylori* negative patients with normal mucosa (p= 0.0001). *H. pylori* was detected in 519/972 (53.4 %) specimens of gastritis, in 51/100 (51.0 %) specimens of intestinal metaplasia, in 22/36 (61.1 %) specimens of dysplasia, in 68/120 (56.7 %) specimens of carcinoma, in 6/20 (30.0%) specimen of hyperplasia, in 65/106 (61.3%) of atrophic gastric and in 5/10 (50.0%) of lymphoma. Considering participants with normal mucosa as the reference group, *H. pylori*-positive participants had statistically significant high prevalence for histological lesions such as gastritis (p <0.0001), atrophy (p <0.0001), metaplasia (p <0.0001), dysplasia (p <0.0001), carcinoma (p <0.0001) and MALT (p=0.0337) (Table 4). Among patients with hyperplasia, *H. pylori*-negative participants were highly affected by this gastric lesion and the difference was not significative compared to patient with normal mucosa (P=0.0666) (Table 3).

Adjustment of the relationship between *H. pylori* infection and histological features of the gastric mucosa to confounding factors such as age and sex of the participants was also evaluated using logistic regression (Table 5). Age group less than 20 years old was taken as reference since this age group was the least affected for each histological feature (Table 5). Compared to infected patients, non-infected patients were more protected from gastric lesions (OR= 0.0869, IC 95 (0.0264- 0.2859). Unless for hyperplasia where non-infected patients were more affected than infected ones (OR= 0.3838 (0.1466-1.0051), *H. pylori* infected patients were more subjected to develop histopathological features compared to non-infected ones. In fact *H. pylori* infected subjects were 1.1775 (0.9139 -1.5171) times more prone to develop gastritis, 1.4866 (0.9894- 2.2337) times to develop atrophic gastric, 1.4415 (0.7309-2.8429) times to develop dysplasia, and 1.2088 (0.8276-1.7655) times to develop carcinoma. For intestinal metaplasia (OR= 0.9408, IC 95=0.6255-1.4151) and MALT lymphoma (OR = 0.9075 IC 95=0.2616- 3.1476), infected patients were slightly affected than non-infected ones. This positive relationship between *H. pylori* infection and histopathological features of the gastric mucosa persisted even after adjustment with confounding factors such as age and sex.

H. pylori infection and inflammatory infiltrate

H. pylori was detected in 91.34% of biopsies samples with inflammatory infiltrate versus 8.66% with no inflammation. This difference was significant (P= 0.0002). Regarding the intensity of inflammation, 38.7%, 4% and 57.3% of specimens with mild, moderate, and marked inflammation respectively were *H. pylori* positive, while 53.5, 2.96 and 43.51% showed respectively the same characteristic among non-infected samples. This difference was also significant (P <0.0001) (Table 4).

H. pylori infection and neutrophil activity

Neutrophil activity and the presence of *H. pylori* were significantly associated (P <0.0001). *H. pylori* was detected in 74.38% (212/285) of specimens showing neutrophil activity, while in specimens with no neutrophil activity, 25.61% and 45.52% were respectively *H. pylori* positive and *H. pylori* negative. Among *H. pylori* positive specimens, 57.59%, 66.28%, 77.77% were with mild, moderate and markedly intense neutrophil activity, while 42.41%, 33.72% and 22.22% of sample revealed the respective characteristic in *H. pylori* negative ones. However, this difference was not significant (p = 0.1999) (Table 4).

Discussion

As reported in previous epidemiological investigations on *H. pylori* infection, Cameroon is an endemic area of infection by this pathogen (Kouitcheu et al., 2018). Also, *H. pylori* clinical isolates circulating in Cameroon has been shown to be highly resistant against antibiotics currently used in the eradication of this pathogen (Kouitcheu et al., 2019). Despite the high prevalence of *H. pylori* infection in the Cameroonian population and the broad spectrum of resistance of circulating strains, this is the first study characterising the association between current *H. pylori* infection assayed by histology and severe gastric lesions in a large Cameroonian population who underwent upper endoscopy for gastric complaints.

The prevalence of *H. pylori* infection was 52.40% among the overall 1290 dyspeptic patients enrolled in this study. A comparison with previous studies carried out in Cameroon shows that this prevalence is lower than the 92.2%, 79.3% and 64.34% reported in previous studies performed in Cameroon using different methods of H pylori detection (Kouitcheu et al., 2018). It is also lower that the prevalence of 73.2% reported in a study performed in the Littoral region of Cameroon using histological examination (Eyoum et al., (2020). The discrepancy between the present prevalence and the previous ones may be due to a rise in the socioeconomic

status and residential conditions of the target population since the prevalence of infection parallels improvement of economy and sanitary standards and is also related to the risk factors exposition of the sample population (Kouitcheu et al., 2019). However, the prevalence we found correlates with the results of some studies done in other developing countries within and out of Africa; 50.6% in the North of South Africa (Venda) (Samie et al., 2007) and about 58% in Guatemala (Dowsett et al., 1999) among the examined individuals.

It has been reported that in developing countries most individuals were infected during childhood and continued to be infected as adults, with a peak prevalence of 80% just before 50 years old (Torres et al., 1998). In the present study, the prevalence of infection according to age exhibited a usual trend as reported in developing countries. It increases with age, from less than 20 years old, reaches a peak at 40 years old and remains almost the same as you grow older.

Prevalence of *H. pylori* infection was higher among women than men (54.29 versus 45.71%). This observation may simply reflect the high number of females in our sample population due to the fact that women are more prompt for clinical examination than men. However, the results of some previous studies carried out in Cameroon showed that because males are naturally more active and less hygienic than females, they were associated with a higher risk of acquiring *H. pylori* infection than the females (Kouitcheu et al., 2018).

Regarding histologic features of biopsy samples, 2.56% of this dyspeptic population was with normal gastric mucosa and the remaining 97.44% presented various pathological features (Table 2). Such observations indicated that clinical signs of dyspepsia are commonly correlated with gastric mucosa lesions or pathological features.

Chronic gastritis with the prevalence of 75.35% was the most frequent histological lesion found among the overall samples. This result is considerably lower than that of Bravo et al. in Colombia who found the prevalence of gastritis as 83.6% (Bravo et al., 2003), but similar regarding the age group of 40 to 59 years as that with the highest incidence of gastritis (Bravo et al., 2003). The majority of biopsies with chronic gastritis were found to be *H. pylori* infected (53.4%), a result that match reports in the literature that this infection is a major cause of gastritis (Gonzalez-Carbajal et al., 2005). Our results were concordant with figures of 50.4% (Dooley et al., 1989), 48% (Satoskar and Vora 1994), 46.5 % (Maitra and Ghosh, 1991), and 45.6% (Parvez et al., 2015) of *H. pylori* associated gastritis detected in gastric specimens and less than those showing *H. pylori* associated gastritis in 66.9% from India (Akanda and Rahman, 2011), in 62.5% from Pakistan (Dandin et al., 2012) and in 67% from Jordan (Yakoob and Hussainy, 2010).

When examining inflammatory infiltrate with respect to *H. pylori* infection, we realised that biopsies with inflammatory infiltrate (P= 0.0002) and mostly those with marked inflammation (P <0.0001) were significantly colonised with the bacterium. This finding is concordant with that of previous studies reporting that the presence of a marked infiltrate of chronic inflammatory cells usually indicates the presence of *H. pylori* and that the degree of chronic inflammation is directly related to the intensity of colonization by this bacterium (Abu-Ahmad et al., 2011).

According to our data, neutrophil activity and the presence of *H. pylori* were significantly associated (P <0.0001), with a higher prevalence of infection but non-significant in biopsies having marked neutrophil activity (p = 0.1999). This coincides with the findings of Tanko et al. (2008) and Sasa et al. (2002) showing a positive relationship between the degree of *H. pylori* colonisation and the intensity of neutrophil activity. As a consequence of *H. pylori* interactions with the epithelium, pro-inflammatory chemokines and cytokines, including IL-8, IL-1β, tumour necrosis factor alpha (TNFα), IL-6, IL-12, CCL2-5, CCL20, and CXCL1-3 are up regulated in the infected gastric mucosa (Reymunde et al., 1993). The presence of these chemokines leads to the recruitment of immune cells (Reymunde et al., 1993). So, *H. pylori* infection leads invariably to an inflammatory process in the stomach which is characterised by an increase in the number of lymphocytes, macrophages and plasmocytes in the lamina propria in variable degrees. This inflammation may be accompanied by neutrophils which contribute to gastritis by secreting inflammatory cytokines and releasing tissue damaging factors from neutrophilic granules such as neutrophil-derived reactive oxygen radicals and proteases indicating inflammatory activity (Evans et al., 1995; Tanko et al., 2008).

It is generally believed that the development of gastric cancer is a multistep process involving sequential changes of the gastric mucosa from non-atrophic gastritis to atrophic gastritis, intestinal metaplasia (IM), dysplasia also known as precancerous or premalignant lesions and finally carcinoma (Correa, 2004). Several studies demonstrated this multistep process in the development of gastric cancer has been reported: in a nationwide cohort study performed in the Netherlands to evaluate the risk of gastric cancer associated with the various premalignant gastric lesions, it was revealed that, atrophic gastritis, intestinal metaplasia, mild-moderate dysplasia, and severe dysplasia were associated with annual incidences of gastric cancer of 0.1%, 0.25%, 0.6%, and 6.0%, respectively (De Vries et al., 2008); in systematic reviews, the incidence of gastric cancer associated with IM ranges from 0% to 10%, with the variable range attributable to various sample sizes and follow-up periods (Kim et al., 2016).

Prevalence of 9.3% was noticed in the current study for carcinoma, 8.2%, 7.7% and 2.8% for atrophic gastritis, intestinal metaplasia and dysplasia (Table 2). This prevalence follows the same tendency observed in a previous study performed in Cameroon reporting intestinal metaplasia rate of 6.3% (5/79) among patients with chronic antral gastritis in Yaoundé, Cameroon, but differ regarding the rate of atrophic gastritis of 74.7% (Firmin et al., 2015). The limited number of samples (79 patients) in the above previous study compared to the current one could explain such a difference. The current prevalence of gastric precursors is less or close than that of some previous studies: prevalence of gastric IM of 7% (Sonnenberg et al., 2010) and 15% (Almouradi et al.,

2013) was found in studies conducted among dyspeptic patients in the United States respectively in 2010 and 2013 and 8% in Netherlands (DeVries et al., 2008). Also, prevalence of 10.3% and 14.7%, 3% and 15%, 11.2% and 21.6% respectively for atrophy and metaplasia was found among dyspeptic patients from South Eastern Brazil (Rodrigues et al., 2019), from the Southern region of Brazil (Muller et al., 2007), and from the Brazilian Northeast region (Motta et al., 2008). However, this prevalence of precancerous states is far lower than that reported in the meta-analysis by Marques-Silva et al. (2014) revealing an atrophic gastritis prevalence of 33.4% and increased to 42% in countries with high incidence of gastric cancer, an intestinal metaplasia (IM) prevalence of 25% and extensive IM of 13% in the worldwide population in general. We think that the low prevalence of precancerous lesions in our sample population compared to that in the above meta-analysis may be due to the fact that our region is an area with low incidence of gastric cancer.

The distribution of gastric precancerous and cancerous lesions according to the age and sex of participants showed that the prevalence of these gastric lesions had an upward trend with aging from chronic gastritis to precancerous and cancer states, while for normal mucosa, it had a downward trend with aging (Table 2). This observation is in accord with previous studies reporting that severe gastric lesions are predominantly pathologies of the middle aged and elderly (Ebili et al., 2015), and those revealing that age ≥50 years is an independent risk factor for IM (Hirota et al., 2006; Correa, 1992). There is a fact that helps to understand the increase of mucosa pathological severity with age. In fact, the degradation of the gastric mucosa is a chronic process starting with a local inflammation of the gastric mucosa, the continuity of this process progressively leads to chronic superficial gastritis, atrophic gastritis, intestinal metaplasia, dysplasia and finally gastric adenocarcinoma (Correa, 1992; Correa, 2004). Since this progression is related to chronic inflammation of the gastric mucosa, there is an age-associated increase in the prevalence of precancerous and cancerous states.

Our data as gender is concern showed that dyspeptic women were at a slightly higher risk of acquiring atrophic gastritis, intestinal metaplasia and gastric carcinoma than males and this is the inverse for dysplasia (Table 2). Such observation may simply reflect the higher proportion of female in our sample population since it is documented that oestrogen is protective, as both delayed menopause and increased fertility lower the risk of gastric cancer (Derakhshan et al., 2009). Moreover, men currently have been shown to have an increased risk of developing gastric pathologies than women due to the fact that they are more exposed to risk factor such as acquiring *H. pylori* infection, poor eating habits, active or passive tobacco consumption, drug abuse (Montalban et al., 1999).

When the prevalence of precursor lesions and cancer were evaluated in relation to *H. pylori* infection, we found a positive relationship between *H. pylori* infection and both precancerous and cancerous lesions. In fact, *H. pylori* was detected in 61.3%, 51.0 %, 61.1 % and 56.7 % of specimens with atrophic gastritis, intestinal metaplasia, dysplasia, and carcinoma respectively with a statistically significant prevalence rate for atrophy ($p < 0.0001$), metaplasia ($p < 0.0001$), dysplasia ($p < 0.0001$) and carcinoma ($p < 0.0001$) among infected subjects compared to infected subjects with normal mucosa (Table 3). Moreover, *H. pylori* infected subjects were 1.4866 (0.9894- 2.2337) times more prone to develop atrophic gastritis, 0.9408 (0.6255-1.4151) times to develop intestinal metaplasia, 1.4415 (0.7309-2.8429) times to develop dysplasia, and 1.2088 (0.8276-1.7655) times to develop carcinoma, suggesting that the presence of this bacteria is a risk factor for these gastric lesions (Table 5). This positive relationship between *H. pylori* infection and both precancerous and cancerous lesions persist even after adjustment with confounding factors such as age and sex. These results are in accordance with several studies ((Almouradi et al., 2013; Olmez et al., 2015; Uemura et al., 2001) and also with the findings of the meta-analysis in which the prevalence of atrophy and intestinal metaplasia was higher in individuals infected by *H. pylori* than in uninfected individuals (Marques-Silva et al., 2014). In the current study, the probability to develop atrophy or dysplasia was higher than metaplasia between *H. pylori* (+) patients and *H. pylori* (-) ones. Such observation indicates that bacterial factors seem to exert a higher influence on the development of atrophy and dysplasia, whereas environmental and host factors would play an important role in the development of intestinal metaplasia (Kim et al., 2008). In fact, it is reported that although the risk developing gastric cancer is increased in patients with gastric intestinal metaplasia, and that gastric intestinal metaplasia (GIM) is an intermediate precancerous gastric lesion in the gastric cancer cascade of chronic gastritis, atrophic gastritis, intestinal metaplasia (IM), dysplasia, and adenocarcinoma (Correa, 2004), the absolute risk is modest and specific subsets of patients with gastric intestinal metaplasia may be at higher risk for progression (Correa, 1992). Such an observation may be also due to the loss of current *H. pylori* infection in intestinal metaplasia (Prashanth and Adam, 2019).

Regarding gastric cancer, our finding is in accordance with previous studies reporting that *H. pylori* is a risk factor in the development of gastric cancer. In fact, *H. pylori* has been found to increase the odds ratio of stomach cancer by 5.9 fold within ten years of infection (Prashanth and Adam, 2019). Also, a recent meta-analysis showed that eradication of *H. pylori* resulted in 37% reduction in the incidence of gastric cancer (Li et al., 2014) and that *H. pylori* eradication is one of the most promising approach in gastric cancer prevention (Li et al., 2014). Adenocarcinomas are divided into cardia and non-cardia cancer based on their anatomical site. Non-cardia cancers are brought about by chronic gastritis and inflammation of the stomach lining and are associated with *H. pylori* infection in 90% of cases (Ford et al., 2014). The pathogenesis of cardia cancer remains unclear, although two distinct aetiologies have been proposed: one is associated with gastro-oesophageal reflux disease and resembles oesophageal adenocarcinoma, and the other is associated with *H. pylori* atrophic gastritis and resembles non-cardia cancer (Mukaisho et al., 2015). Koreans reported that *H. pylori* infection was associated with a

2.88-fold higher risk of gastric cardia adenocarcinoma Bae and Kim (2016), and another meta-analysis reported a positive association between *H. pylori* infection and gastric cardia adenocarcinoma in geographic regions with a high incidence of gastric cardia cancer (Cavaleiro-Pinto et al., 2011). However, there are contradictory studies where the association of *H. pylori* with gastric precancerous and cancerous lesions tended to be null or inverse. In fact, Kato et al. (2006) documented no difference in the presence of intestinal metaplasia among the study group of children with and without *H. pylori* infection; no child in a Brazilian cohort of 96 children with *H. pylori* gastritis were found to have gastric intestinal metaplasia (Carvalho et al., 2012). One explanation for the geographic variation in the association between *H. pylori* and gastric cancer could be the differences in the genetics of *H. pylori* or host genetics in populations from different geographic regions. It has been shown that the presence of certain *H. pylori* genotype (vacA m1) and polymorphisms of the host inflammatory cytokines (interleukin-1β, IL-10 and IL-17) were associated with the presence of gastric precancerous lesions (Wroblewski et al., 2010). Bacterial virulence factors such as cytotoxin-associated gene A (CagA), vacuolating cytotoxin A (VacA) and the adhesion protein BabA2 likely play an important role in determining the outcome of *H. pylori* infection (Cover and Blanke, 2005). A longitudinal cohort study following 4655 healthy asymptomatic subjects for 7.7 years, with gastric cancer development in 45 patients showed that the risk of gastric cancer increased progressively from *H. pylori*–negative/cagA-negative to *H. pylori*–positive/cagA-negative, and finally *H. pylori*–positive/cagA-positive (Ohata et al., 2004).

Prevalence rates of MALT lymphoma was 0.8% in the sample population. This prevalence was significantly elevated (p=0.0337) among *H. pylori* infected patients compared to infected subjects with normal mucosa (Table 3). Moreover, infected patients were slightly more affected by this type of gastric lesions compared to non-infected ones (OR = 0.9075, IC 95 = 0.2616-3.1476), suggesting a positive relationship between *H. pylori* infection and MALT lymphoma (Table 5). *H. pylori* infection leads to chronic gastritis which involves both T cells and B cells arising from the mucosal associated lymphoid tissue (MALT). The recognition of the *H. pylori* antigen by the immune system leads to T cell activation, lymphoid follicle formation, and B cell proliferation (Katharina et al., 2015). It is thought that the *H. pylori* antigen-presenting cells interact with CD4-expressing cells. This CD4 cell then binds to a B cell in the marginal zone causing hyperproliferation of B cells (D'Elios et al., 1999), also known as a MALT lymphoma. The involvement of *Helicobacter pylori* in MALT lymphoma is now well established through the following observations; *H. pylori* eradication allows lymphoma regression in 60% to 90% of patients (Wotherspoon et al., 1993), *H. pylori* eradication therapy is now considered as the first therapeutic approach for low grade gastric mucosal lymphoma (Cammarota et al., 1995), if a reinfection occurs, gastric mucosal lymphoma reappears and evolves more rapidly because neoplastic cells are already sensitized to *H. pylori* antigens (Kim et al., 2016).

The average age of patients affected by MALT lymphoma was 47.13 ±19.81 years old with 40-59 years as the most represented age group of incidence for this lesion type (4/10, 40.0%) and males were more affected than women with a significant risk (P= 0.0471). Our results coincide with literature reporting that approximately 70% of severe gastric lesions are diagnosed between the ages of 55 and 84 years and that men have a 5- and 2-fold increased risk of cardia cancer and non-cardia types, respectively (Sonnenberg and Genta, 2015). The mechanism by which men are predisposed to developing gastric cancer is likely multi-factorial. Currently, men have been shown to have an increased risk of developing gastric pathologies than women due to the fact that they are more exposed to risk factor such as acquiring *H. pylori* infection, poor eating habits, active or passive tobacco consumption, drug abuse (Montalban et al., 1999).

Gastric polyps are usually found incidentally on upper gastrointestinal endoscopy and they are generally asymptomatic. However, this type of gastric lesions should be diagnoses and management as some polyps have malignant potential. The prevalence of hyperplastic polyps (HPs) was 1.55% (20/1290) in our sample population and 70.0% (14/20) of biopsies with this lesion were *H. pylori* negative. This difference was significant (P= 0.0503) (Table 5), indicating a negative association between *H. pylori* infection and hyperplasia. These results provide further evidence for the inverse association between *H. pylori* infection and hyperplastic polyps. In fact, several studies had demonstrated that *H. pylori* infection is inversely associated with the occurrence of hyperplastic polyps (Carmack et al., 2009; Xinjuan et al., 2020). The possible reasons for the negative association between the infection and hyperplastic polyps remain controversial. It has been suggested that the increased prevalence of hyperplastic polyps is caused by IM of gastric mucosa, rather than the decreased prevalence of the infection (Carmack et al., 2009). However, this explanation was not supported by other studies reporting that hyperplastic polyps are widely found in geographic areas where *H. pylori* is common and that polyps may regress following *H. pylori* eradication (Ljubicic et al., 1999; Kyburz and Muller, 2017).

The average age of patients with hyperplasia was 52.25 ±19.65 compared to 40.17 ± 9.59 in those with normal mucosa. Moreover, the most represented age of incidence of hyperplasia (9/20, 45.0%) was up to 60 years old. This finding agrees with other studies, reporting that older age appeared to be associated with an increased risk for hyperplasia polyps (Kyburz and Muller, 2017; Takeuchi et al., 2017; Stolte et al., 1994).

As gender is concerned, there was no difference between the two sexes. This is in accordance with studies from other geographical regions, according to which gender has no significant influence on the risk of hyperplastic polyps (Kyburz and Muller, 2017; Dore et al., 2017). However, studies carried out among the western and Chinese populations have shown that, females have an increase risk in the development of gastric polyps (Dore et al., 2017). These conflicting results may be attributed to

the differences in ethnicity or differences in the prevalence of *H. pylori* (Hooi et al., 2017).

It should be noted that in this study 52.4% of patients evaluated were currently *H. pylori* infected whereas gastric pathological features was detected in 97.44% cases. So, we believe that H pylori infection alone cannot explain these overall gastric lesions and that there must be other critical cofactors affecting the risk of *H. pylori* infection in the infection-precancerous and cancer relationship such as sex, age as well as life style. Another reason which could explain such situation may be the underestimation of the true risk of *H. pylori* infection in the development of precancerous and cancerous lesions due to loss of *H. pylori* as the mucosa undergoes malignant transformation. In fact, although, there is evidence that *H. pylori* is frequently found in gastric biopsy specimens from individuals with atrophic gastritis, intestinal metaplasia and gastric cancer, it is reported that with the development of advanced gastric tumours, the bacteria can be lost from the stomach (Prashanth and Adam, 2019) and that with the loss of infection, patients with gastric cancer may be *H. pylori* negative even though they have been infected in the past (Karnes et al., 1991).

In conclusion, our findings revealed that the prevalence of *H. pylori* among our sample population is high as is the case in most of the African countries. It was also shown that degeneration of the gastric lining which results in pathologies such as chronic gastritis, precursors of malignancy, adenocarcinomas and MALT are mainly caused by *Helicobacter pylori* and that hyperplastic polyp is inversely associated with *H. pylori* infection. *H. pylori* eradication, early detection and treatment of precursor lesions could significantly impact these outcomes in our milieu. Further studies have to be carried out in order to find out the various strains which in our context is responsible for this degeneration.

Author Contribution Statement

KMLB conceived of the study, designed the experiments and supervised the work. FNGF extracted data from the medical records, hospital computerized databases and clinical charts of the patients. KMLB provided the facilities for the study and drafted the manuscript. All the authors read and approved the final manuscript.

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Approved by scientific Body

No applicable.

Ethical consideration

This study was approved by the Institutional Review Board of University Teaching Hospital (Approval no 703/AR/CHUY/DG/DGA/CAPRC) and Cameroon Pasteur institute CPC (Approval no 000378/2019/CPC/DG/DAF/SRH) and the Ethical Committee of Medical Sciences for the Centre Region of Cameroon (Approval no 0438 /CRERSHC/ 2019).

Availability of data

The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

Conflict of interest

The authors declare that they have no competing interests.

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