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Helicobacter pylori Infection and Gastroduodenal Lesions: Prevalence and Associated Factors in Cote d'Ivoire

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Abstract

Objective: The objective is to determine the prevalence of *Helicobacter pylori* (*Hp*) infection and highlight the determinants of the infection as well as the gastric histopathological lesions associated with this infection. **Methods:** This is a retrospective study carried out from August 01, 2015 to December 31, 2020 in Bouaké. It included all patients with gastric and/or duodenal lesion on upper gastrointestinal endoscopy in whom gastric biopsies and histopathology results are available. **Results:** The study involved 510 patients (301 men and 209 women). The prevalence of *Hp* was 66.47% (339/510 patients). The presence of *Hp* was not significantly related to age and gender. Epigastralgia was the most common indication with no significant difference between the positive and negative *Hp* groups (65.37% vs 34.63%, $p = 0.35$). A significant difference was only observed for duodenal ulcers (84.31% *Hp*+ vs 15.69% *Hp*–, $p = 0.004$). Regarding histological lesions: Chronic and active gastritis was strongly related to the presence of *Hp* ($p < 0.05$). Intestinal metaplasia and gastric atrophy were not significantly associated with the presence of *Hp*. These precursor lesions of gastric cancer (metaplasia and atrophy) were, however, significantly related to chronic gastric disease with $p = 0.02$ and $p < 0.001$, respectively. **Conclusion:** The prevalence of *Hp* is high in Bouaké. Our study confirms the link between *Hp* infection and chronic and active gastritis.

Keywords

Helicobacter Pylori, Gastritis, Atrophy, Intestinal Metaplasia

1. Introduction

Since its discovery in 1981 by Barry Marshall and Robin Warren, *Helicobacter pylori* (*Hp*) established itself as a bacterium of major importance in the genesis of pathologies in gastroenterology [1]. The links between chronic gastritis (CG) due to *Hp*, peptic ulcers (PU) and some gastric cancers (adenocarcinomas and *MALT* gastric lymphomas) are well established [2]. Its global prevalence is in the order of 50%, predominant in Africa, Asia, Central and South America [1]. In developing countries, it affects about 80% of the population with transmission occurring very early in childhood [3]. The factors that influence the incidence and prevalence of *Hp* infection are age, gender, geographic and socio-economic factors [1] [3]. Among the diagnostic methods of *Hp*, the pathological examination of gastric biopsies performed during digestive endoscopy is most commonly used because of its high specificity and sensitivity that are greater than 90% [4]. Despite the improvement in living standards and the introduction of new *Hp* eradication protocols, the prevalence of this bacterium and its involvement in the genesis of gastric lesions remain a concern as well in Africa, in Asia and in the low-prevalence countries [5] [6]. In Côte d'Ivoire, few studies exist on the relationship between *Hp* and gastric lesions since the advent of quadruple therapy [7]. The purpose of this study is to determine the prevalence of *Hp* infection and to identify the determinants of infection as well as the gastroduodenal histopathological lesions associated with this infection.

2. Methods

This was a retrospective study covering the period from 1 August 2015 to 31 December 2020. Our study was carried out on the basis of digestive endoscopy reports from 3 digestive endoscopy centres in Bouaké (Bouaké Teaching Hospital, Holy Fraternity Clinic and Notre Dame des Apôtres Clinic). The study population consisted of patients who performed upper digestive endoscopy during the study period in one of the 3 centres.

We included in the study, patients in whom gastric biopsies (fundal and antral) had been performed and with pathological result from the available biopsies. For patients who performed multiple upper digestive endoscopies, only the results of the initial endoscopy and histological examination were considered for the study. On the upper gastrointestinal endoscopy reports, we collected data on age, gender, occupation, examination indications, description of gastric and duodenal lesions. The histological gastric lesions in accordance with Sydney's categorization criteria (chronic gastritis, active gastritis, glandular atrophy, intestinal metaplasia, dysplasia and gastric cancer) and the presence or absence of *Hp* were also collected from the histological report of the gastric biopsies.

Epi info 7 software was used for data entry and analysis. Differences in the distribution of variables were evaluated by the Chi-square test and those with a p-value of <0.05 were considered statistically significant. Odds ratios (OR) and respective 95% confidence intervals (95% CI) were calculated for each gastric cancer precursor lesion in relation to the presence of H. pylori infection.

Ethics

The study was carried out with the approval of the Scientific Medical Director of the Bouaké's Teaching Hospital. Confidentiality was respected by assigning an anonymity number to each investigation report.

3. Results

A total of 510 patients who met the criteria were considered in our study. The average age was 51.40 years with extremes ranging from 13 to 89 years, divided into age groups of less than 30 years (8.43%), 30 to 50 years (40.20%), 51 to 70 years (42.75%) and 71 to 90 years (8.63%). The majority of patients included were male at 59% (sex ratio 1.44). The 510 patients resided in 70% urban areas and 30% in rural areas. More than half of the patients were unemployed (25.10%) and in the informal sector (26.85%).

The overall prevalence of *Hp* infection was 66.47% (339/510). There was no significant difference between men (199/301) and women (140/209) for *Hp* prevalence (p = 0.84). There was no significant difference between *Hp* (+) and *Hp* (−) patients for different age groups (p = 0.48), gender (male/female, p = 0.84) and area of residence (Urban/rural, p = 0.19) (Table 1).

The most common indication of gastroscopy was epigastralgia (75.8%) with no significant difference between *Hp* (+) and (−) patients (Table 2).

The most common endoscopic lesions were gastric erythema (65.10%) and gastric ulcers (29.60%) with no significant difference between positive and negative *Hp* patients. A significant difference was observed only for duodenal ulcers (Table 3).

Table 1. Distribution of age, gender, place of residence and hospitalization according to *H. pylori* status.

H. pylori Status	Total n = 510	*Hp* (+) n = 339 (66.47%)	*Hp* (−) n = 171 (33.53%)	p
Gender				
Male	301 (59.01%)	199 (66.11%)	102 (33.89%)	0.84
Female	209 (40.98%)	140 (66.99%)	69 (33.01%)	
Average age		51 years old	52.6 years old	0.2
Age group				
10 - 30 years old	43 (8.43%)	31 (72.09%)	12 (27.91%)	0.48
31 - 50 years old	205 (40.19%)	138 (67.32%)	67 (32.68%)	
51 - 70 years old	218 (42.74%)	138 (63.30%)	80 (36.70%)	
71 - 90 years old	44 (8.62%)	32 (72.73%)	12 (27.27%)	
Residence				
Rural	151 (29.60%)	94 (62.25%)	57 (37.75%)	0.19
Urban	359 (70.40%)	245 (68.25%)	114 (31.75%)	
Hospitalization	57 (11.11%)	33 (57.89)	24 (42.11)	0.15

Table 2. Distribution of indications for upper gastrointestinal endoscopy in 510 patients according to the presence of *Hp*.

H. pylori Status	Total n = 510	*Hp* (+) n = 339	*Hp* (−) n = 171	p
Epigastralgia	387 (75.90%)	253 (65.37%)	134 (34.63%)	0.35
Vomiting	57 (11.17%)	35 (61.40%)	22 (38.60%)	0.39
Upper duodenal hemorrhage (UDH)	52 (10.19%)	39 (75.00%)	13 (25.00%)	0.16
Cirrhosis chech-up	16 (3.13%)	11 (68.75%)	5 (31.25%)	0.84
Weight-loss	13 (2.54%)	5 (38.46%)	8 (61.54%)	0.03
Gastro-oesophageal reflux disease (GERD)	11 (2.15%)	8 (66.67%)	3 (33.33%)	0.98
Dysphagia	12 (2.35%)	8 (66.67%)	4 (33.33%)	0.98
Anaemia	10 (1.96%)	6 (60. 00%)	4 (40.00%)	0.66

Table 3. Distribution of endoscopic lesions according to the presence of *Hp*.

H. pylori Status	*Hp* (+) n = 339	*Hp* (−) n = 171	p
Gastric erythema	245 (69.01%)	110 (30.99%)	0.06
Gastric ulcers	104 (68.87%)	47 (31.13%)	0.45
Duodenal ulcer	43 (84.31%)	8 (15.69%)	0.004
Gastric erosions	60 (67.42%)	29 (32.58%)	0.83
Gastric ulcerations	46 (71.88%)	18 (28.13%)	0.32
Gastric tumors	20 (37.04)	34 (62.96%)	0.001

For histological lesions: Chronic and active gastritis were strongly related to the presence of *Hp* (respectively OR = 77.79 [30 - 187]; p = 0.001 and 260 [106 - 640]; p = 0.001). Gastric malignancies, on the other hand, were significantly related to the absence of *Hp* (OR = 0.19 [0.1 - 0.35]; p = 0.001). Intestinal metaplasia and gastric atrophy were not significantly associated with the presence of *Hp* (**Table 4**). These precursor lesions of gastric cancer (intestinal metaplasia and glandular atrophy) were, on the other hand, significantly related to chronic gastric univariate analysis with p = 0.02 and p = 0.001 respectively (**Table 5**).

4. Discussion

The results of this work show a high frequency of *Hp* infection in our study population. In developing countries, *Hp* infections affect about 80% of the population with transmission occurring very early in childhood [1] [3].

In this study, the higher prevalence of *Hp* in men at 58.70% compared to 41.30% in women was not statistically significant. Amel and al [8] reported that it is generally accepted that men and women have the same risk of being infected at any age confirming the results of our series. However, Houria and al reported that women are the most infected with *Hp* compared to men [9]. Also other studies found male predominance [10] [11] [12] [13].

Table 4. Distribution of patients by gastric histological lesions associated with the presence of *Hp*.

H. pylori Status	Total n = 510	*Hp* (+) n = 339	*Hp* (−) n = 171	p	OR (IC 95%)
Malignant gastric tumor	51 (0.10%)	16 (31.37%)	35 (68.63%)	0.001	0.19 (0.1 - 0.35)
Chronic gastritis	413 (80.98%)	334 (80.87%)	79 (19.13%)	0.001	77.79 (30 - 187)
Active gastritis	363 (71.17%)	333 (91.74%)	30 (8.26%)	0.001	260 (106 - 640)
Reactive gastritis	66 (12.94%)	3 (4.55%)	63 (95.45%)	0.001	0.01 (0.005 - 0.05)
Mucosal atrophy	64 (12.54%)	45 (70.31%)	19 (29.69%)	0.46	1.24 (0.69 - 2.17)
Intestinal metaplasia	48 (9.41%)	32 (66.67%)	16 (33.33%)	0.97	1.01 (0.53 - 1.89)

OR: odds ratio; CI: 95% confidence interval.

Table 5. Association of gastric precancerous lesions and chronic gastritis.

Stomach histology	Chronic gastritis (+) n = 413	Chronic gastritis (−) n = 97	p	OR (CI 95%)
Mucosal atrophy	61 (95.31%)	3 (4.69%)	0.001	3.83 (1.66 - 17.69)
Intestinal metaplasia	45 (93.75%)	3 (6.25%)	0.01	1.01 (1.16 - 12.60)

OR: odds ratio; IC: Confidence interval 95%.

Regarding age, our results are similar to a study conducted in Côte d'Ivoire, where no significant differences in age were reported [14]. He argues that in Africa, every adult, regardless of socio-economic status, had a childhood in an environment conducive to contamination [9].

Some studies confirm the association between *Hp* and urban poverty related to hygiene conditions [15]. In our study, place of residence and occupation did not significantly influence the presence of *Hp*. Our results correspond to the work of Andoulo *et al.* [16].

Epigastralgia accounted for about two-thirds of the indications of positive *Hp* patients. However, there is no link between this indication and the presence of *Hp* in our study (p = 0.35). A significant difference was observed only for weight loss between positive and negative *Hp* patients (p = 0.03). This difference could be explained by stenosis syndrome, which is usually responsible for weight loss in gastric tumors. The link between gastric adenocarcinoma and *Hp* is well established according to the literature [17].

Although the prevalence of *Hp* in gastric ulcers is high (68.87%). There is no significant link in our study between gastric ulcer and *Hp*. The link between duodenal ulcers and *Hp* is well established according to the literature [18]. However, it should be noted that our study was retrospective. Patients taking proton pump inhibitors or antibiotics may have false-negative histological results for *Hp*. Which could probably explain our results.

Hp infection is the most important risk factor for chronic gastritis, gastric atrophy and intestinal metaplasia and is considered the precursor to gastric cancer [19]. In our work, chronic gastritis and active gastritis were significantly higher in *Hp* (+) patients and were a risk factor with an OR of 77.79 and 260 respectively in accordance with the literature data [12]. The frequencies of atrophic gastritis and intestinal metaplasia are low in our study. The studies carried out in sub-Saharan Africa also noted a low frequency of intestinal metaplasia of less than 20%. However, they showed a higher frequency of gastric atrophy of up to 75% [5] [7]. In his review of the literature, Archampong noted that precancerous lesions (gastric atrophy and intestinal metaplasia) are not uncommon in symptomatic patients undergoing endoscopy in tertiary health facilities in Africa. Most hospital-based endoscopic studies showed prevalence rates of gastric atrophy of 5% - 38% and for intestinal metaplasia of 4% - 32% among populations in sub-Saharan Africa [20]. The presence of *Hp* was not significantly associated with gastric atrophy and intestinal metaplasia in our study. These gastric histological lesions are considered by several authors to be significantly related to the presence of *Hp* [21] [22]. Inflammation and atrophy of the gastric mucosa are known to be factors that are not conducive to the development of Hp [20]. In addition, the lack of information in our study on the use of pump inhibitors or antibiotics could influence the search for hp during pathological examination [3] [20]. The absence of link between these gastric precancerous lesions and the presence of *Hp* in our study could therefore be explained by these two conditions. For tumours, *Hp* was shown to be a determining factor in the etiology of gastric cancers [17]. In our work, tumour lesions are significantly associated with *Hp* with a predominance of tumour lesions in case of *Hp* negative. Some authors would explain this result by "the African sub-Saharan enigma", because of an ancient early childhood infection in sub-Saharan Africans, the human and host response to *Hp* could be protective against a virulent organism and that, in most people, *Hp* would not cause more serious sequelae. This would suggest that there may be host-protective/inhibiting factors that would prevent the progression of *Hp*-induced active gastritis to cancer [23]. This theory is nevertheless challenged by other authors such as Agha and Graham through a multicentre study [24].

The retrospective nature of our study could underestimate the prevalence of *Hp* through selection bias. It is not known whether the patients were on antibiotic therapy and or under a proton pump inhibitor when the gastric biopsy was performed. Despite this limitation, our study gives interesting results.

5. Conclusion

The prevalence of *Helicobacter pylori* is high in Bouaké, like are other cities in developing countries. This study confirmed the involvement of *Hp* in chronic and active gastritis. Performing gastric biopsies in search of *Hp* during upper digestive endoscopies should be systematic. It would also be interesting to carry

out a prospective multicentre study to clarify the links between *Hp* and precancerous and cancerous gastric lesions.

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Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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