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Prevalence and Factors Associated with Intestinal Metaplasia in Chronic *Helicobacter pylori* Gastritis in a Country with High Endemicity: Ivory Coast Case

Hatrydt Guillaume Dimitri Kouamé[1,2], Lisiane Mewetieh[2], Kouassi Olivier Claver Koffi[2], Bathaix Fulgence Mamert Yao[1,2]

[1]Medical Sciences Training and Research Unit of Abidjan, Félix Houphouët-Boigny University, Abidjan, Ivory Coast
[2]Hepatology and Gastroenterology Unit, Yopougon University Hospital, Abidjan, Ivory Coast
Email: *hatry333@gmail.com

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Abstract

Context/Objective: Few studies have been carried out in a country with high endemicity for Helicobacter pylori (*H. pylori*) infection in Sub-Saharan Africa looking for the association of intestinal metaplasia (IM) with chronic gastritis. We hypothesize that IM is correlated with the intensity of *H. pylori* infection in a country with high endemicity, Ivory Coast. The objective of this study was to determine the prevalence of intestinal metaplasia in chronic *H. pylori* gastritis in Ivory Coast. **Methods:** This was a prospective, cross-sectional, multicenter study, carried out over a period of 5 months, in the reference hospital centers of Abidjan, specialized in Gastroenterology. All patients who had undergone Gastroscopy with biopsies according to the criteria of the Sydney System for the anatomopathological study, those with chronic gastritis and/or *H. pylori* intestinal metaplasia on histology and in whom all the parameters of the Sydney system classification had been well informed. The quantitative variables were expressed by their means accompanied by their standard deviations and the qualitative variables by their numbers and percentages. Chi-square and Fischer tests were used to look for associations between variables. The significance level was set at 5%. **Results:** 152 patients were retained. The mean age was 44.9 ± 12.9 years. The prevalence of intestinal metaplasia was 11.8%. In univariate analysis, no significant association was found between clinical and pathological sociodemographic factors (age, sex, ethnicity, educational level, profession) and intestinal metaplasia in chronic Helicobacter pylori gastric cases. In multivariate analysis we found that prolonged use of Proton Pump Inhibitors (PPIs) and a history of Gastroesopha-

geal Reflux Disease (GERD) were significantly associated with the absence of IM. **Conclusion:** Chronic *H. pylori* gastritis is the main risk factor for intestinal metaplasia. Prolonged use of PPIs and a history of GERD were significantly identified as factors that would protect against intestinal metaplasia.

Keywords

Helicobacter Pylori, Chronic Gastritis, Intestinal Metaplasia, Ivory Coast

1. Introduction

Chronic gastritis (CG) is considered a precancerous stage which has been the subject of several studies and classifications, the most recent of which is the coding according to the Sydney system [1]. It has a histological definition: it is a persistent inflammatory state of the gastric mucosa, diffuse or localized, associated with epithelial alterations which can progress towards atrophy and/or intestinal metaplasia [2]. In many patients, it is associated with Helicobacter pylori infection. Gastric carcinogenesis is a multi-step process with a multitude of histological lesions which can either stabilize or evolve slowly according to the sequence gastritis - atrophy - intestinal metaplasia (IM) - dysplasia - cancer [3]. IM is an intermediate step in the carcinogenesis process of intestinal-type adenocarcinoma [4]. It is defined as the replacement of gastric columnar epithelial cells by cells of intestinal morphology with the presence of goblet cells, Paneth cells and absorptive cells [5]. *Helicobacter-pylori* infection affects half of the world's population, mainly in developing countries where the infection reaches 80% of the population [6]. It is responsible for the majority of chronic gastritis ranging from minimal gastritis to severe gastritis. IM during chronic gastritis in Africans is rarely reported in the literature. Indeed, T. Darre *et al.* reported 134 cases of IM out of 247 cases of histologically proven chronic gastritis [1]. Studies including a high number of patients suffering from chronic gastritis have nevertheless been rarely reported in Ivory Coast.

On the other hand, although suggested in the world literature that the intensity of pylori infection is strongly correlated with the occurrence of IM [7], the study by Theresa H Nguyen *et al.* in the USA does not did not show a causal link between the intensity of *H. pylori* infection and the occurrence of IM [8]. Furthermore, this study included more Hispanic than African-American patients. In addition, other factors have been implicated, such as alcohol and tobacco consumption, in the occurrence of IM in chronic *H. pylori* gastritis [9]. However, no study has been done in a country with high endemicity in sub-Saharan Africa for *H. pylori* infection looking for the association of IM with chronic gastritis. We hypothesize that MI is correlated with the intensity of *H. pylori* infection in a country with high endemicity, Côte d'Ivoire [6]. The primary objective was to determine the prevalence of IM in *H. pylori* GC and specifically to determine the factors associated with IM in chronic *H. pylori* gastritis.

2. Methods

Type of study and period:

This was a prospective, cross-sectional, multicenter study, carried out over a period of 5 months (March-July 2022) in Abidjan (Ivory Coast).

Study framework:

Outpatient consultation services specialized in hepato-gastroenterology:

- Public structures: Cocody and Angré university hospitals, Abobo hospital in Ivory Coast.
- Private structures: Farah Polyclinic, II Plateaus Polyclinic.

Study population:

Our study concerned:

- Patients who had performed an Gastroscopy with associated gastric biopsies (2 fundus, 01 angulus, 2 antrum).
- Patients who had consulted in the various centers in the city during our study period.

Selection criteria:

Patients who had chronic *H. pylori* gastritis with or without intestinal metaplasia on pathological analysis of the stomach biopsy sample.

Inclusion criteria:

Had been included:

- All patients who had performed a gastroscopy with biopsies for anatomopathological study.
- Patients who had chronic gastritis and/or *H. pylori* intestinal metaplasia on histology.
- Patients in whom all the parameters of the Sydney system classification had been correctly informed and who consented to the study.

Non-inclusion criteria:

Not included:

- Patients who had CG not associated with *H. pylori* infection.

- Patients in whom the elements of the Sydney classification were incomplete on the pathological report.

- Patients who did not consent to the study.

The parameters studied:

Descriptive variables:

- Age, sex, level of education, profession, presence of comorbidities.

- History of alcoholism, smoking, notion of triple therapy, notion of taking PPI medications (proton pump inhibitors), antacids, alginates, Anti-H2.

- The notion of gastric cancer in the family.

- Indications for Gastroscopy: epigastralgia, nausea, vomiting, heartburn, GERD, dyspepsia.

Histological variables linked to gastritis:

Histological variables according to the Sydney system classification:

- Topographic, morphological and etiological information linked to gastritis.

- The location of the gastritis, its focal or diffuse nature, the location of the infiltrate within the chorion, the density of the inflammation as well as its active nature or not.
- The presence or absence of glandular atrophy, intestinal metaplasia and *H. pylori.*
- The rating of parameters evaluating the degree of severity according to a scale ranging from 1 to 3 corresponding to a mild (1/3), moderate (2/3) and severe (3/3) stage.

Data collection and analysis:

A pre-established survey sheet presenting the different variables served as a data collection support. The data was recorded in Cspro version 7.2 then exported to SPSS 26.0 for statistical analysis. The quantitative variables were expressed by their means accompanied by their standard deviations and the qualitative variables by their numbers and percentages. Chi square and Fischer tests were used to look for associations between variables. The significance threshold was set at 5%.

Ethical consideration:

Data confidentiality was respected. The managers of the structures in which the studies were carried out were informed and gave their agreement.

3. Results

During our study period, 261 patients were eligible, of whom we retained 152, 18 of whom had intestinal metaplasia, or 11.8% of our study population (**Figure 1**). The average age of our study population was 44.9 ± 12.9 with extremes of 16 and 72 years. There was a female predominance of 77.8% (n = 14) among patients with intestinal metaplasia and the majority (50%) of patients who had IM belonged to the age group of [50 - 60] with a mean age of 50.9 ± 8.1 years. Chronic *H. pylori* gastropathy was more common in the age group of [40 - 50 years].

The location of intestinal metaplasia was almost always antral (11.2%) compared to 2% at the fundal level.

There was no significant association between metaplasia and the intensity of *H. pylori* infection (**Table 1**). There was also no significant association between intestinal metaplasia and the degree of severity of chronic gastritis (**Table 2**).

We did not find a significant association between intestinal metaplasia and the sex of the patients (p value = 0.59), nor between intestinal metaplasia and the age of the patients (p value = 0.1).

Our work found a significant association between the absence of intestinal metaplasia and the use of PPIs (p value = 0.04) (**Table 3**).

Our study found a significant association between GERD and the absence of intestinal metaplasia (p value = 0.04) (**Table 4**).

Taking PPIs (p value = 0.01) and GERD (p value = 0.01) remained significantly associated with the absence of intestinal metaplasia after logistic regression (**Table 5**).

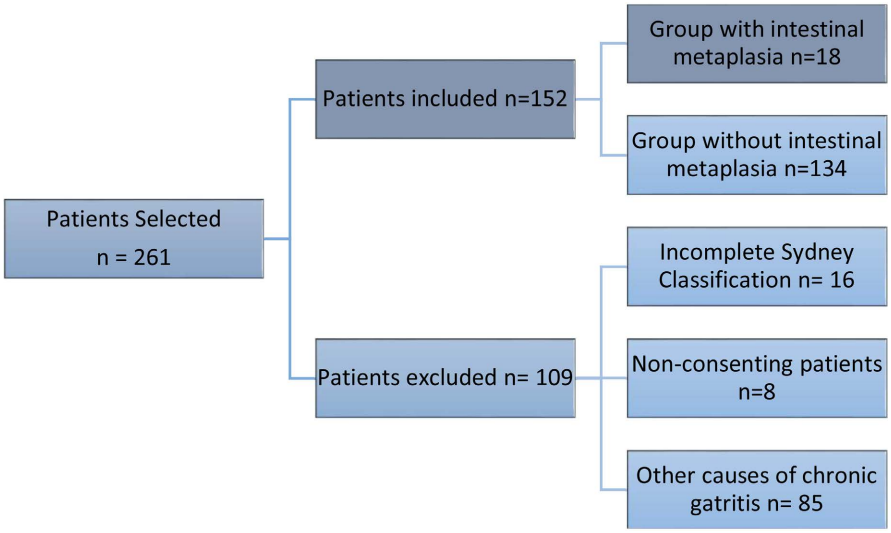

Figure 1. Flowchart.

Table 1. Association between the degree of *H. pylori* infection and the presence of intestinal metaplasia.

Degree of *H. pylori* infection	Metaplasia		p-Value
	Yes n (%)	No n (%)	
Lightweight	7 (38.9)	55 (41.0)	0.82
Average	9 (50.0)	58 (43.3)	
Severe	2 (11.1)	21 (15.7)	
Total	18 (100)	134 (100)	

Table 2. Association between intestinal metaplasia and the degree of severity of chronic *H. pylori* gastritis.

Gastric severity	Metaplasia		p-Value
	Yes n (%)	No n (%)	
Lightweight	5 (3.3)	55 (36.2)	0.08
Average	8 (5.3)	66 (43.4)	
Severe	5 (3.3)	13 (8.6)	
Total	18 (12.0)	134 (88.2)	

Table 3. Association between lifestyle and intestinal metaplasia.

Background	Metaplasia		p-Value
	Yes n (%)	No n (%)	
Alcohol	5 (3.3)	20 (13.2)	0.17
Tobacco	1 (0.7)	4 (2.6)	0.47
Traditional therapy	9 (5.9)	79 (52.0)	0.47

Continued

Coffee	2 (1.3)	22 (14.5)	0.74
Tea	7 (4.6)	32 (21.1)	0.17
Consumption of dairy products	1 (0.7)	3 (2.0)	0.40
NSAIDs	2 (1.3)	15 (9.9)	1.00
Aspirin	1 (0.7)	0 (0)	0.12
PPI	3 (2.0)	4 (2.6)	0.04*
Anti acids	4 (2.6)	17 (11.2)	0.28
Alginates	1 (0.7)	3 (2.0)	0.40

Table 4. Association between digestive signs and intestinal metaplasia.

Clinical signs	Metaplasia		p-Value
	Yes n (%)	No n (%)	
Epigastralgia	15 (9.9)	121 (79.6)	0.37
Nausea	4 (2.6)	15 (9.9)	0.24
Vomiting	3 (2.0)	9 (5.9)	0.16
Pyrosis	0 (0)	1 (0.7)	1.00
GERD	3 (2.0)	4 (2.6)	0.04*
Dyspepsia	1 (0.7)	17 (11.2)	0.70

Table 5. Factors associated with intestinal metaplasia in multivariate analysis.

Variables	Metaplasia		Adjusted p-value
	Yes n (%)	No n (%)	
PPI	3 (2.0)	4 (2.6)	0.01*
GERD	3 (2.0)	4 (2.6)	0.01*

4. Discussion

***Prevalence of Intestinal Metaplasia* (*IM*):**

This study is the first of its kind in Côte d'Ivoire. It consisted of estimating the frequency of IM in patients with chronic *H. pylori* gastritis. It took place over a period of 5 months, 261 patients were recruited and 150 were included.

In our series, the prevalence of IM was 11.8%, comparable to the data observed by Ghailane Ghizlane in Morocco [3]. Higher (54.25%) or lower (3.8%) prevalences were also found in Black Africa [1] [7]. These data demonstrate the heterogeneity of the prevalence of IM according to regions in Africa.

Antral location was more frequent than fundal location (11.2% versus 2%). This preferential distribution of IM was usual [3] [4] [7] [10] [11] [12]. Indeed, *H. pylori* infection is considered by the WHO to be the main factor involved in gastric carcinogenesis, particularly IM [6].

Factors associated with Intestinal Metaplasia:

Histological factors:

Chronic gastritis (CG) was strongly associated with *H. pylori* infection in our study. Which corroborates the results of several studies carried out in black Africa in this direction [13] [14] [15]. *H. pylori* is the main risk factor in the development of CG. On the other hand, this was part of the selection criteria for the patients in our sample. We also noted a significant association between the severity of chronic gastritis and the intensity of *H. pylori* infection, contrary to certain data reported in the literature [7] [16] [17]. Choudhary *et al.* in India did not show a correlation between the intensity of *H. pylori* infection and the severity of chronic gastritis. However, our study did not find a significant association between the degree of *H. pylori* infection and the occurrence of IM. The same was true for the search for the association between IM and the severity of chronic gastritis. Data in the literature on the correlation between the degree of *H. pylori* infection and gastric intestinal metaplasia are discordant. Thus Ghasemi *et al.* [18] noted a correlation between the degree of *H. pylori* infection and IMD, contrary to the results of Tanko *et al.* in Niger [19].

This apparent contradiction between the promoting role of *H. pylori* in the genesis of precancerous lesions, particularly IM, can be explained by several factors.

It has been proven that when a large part of the gastric mucosa was the site of the IM, the possibilities of identifying the bacteria were reduced even if the samples taken complied with the sampling protocol recommended by the Sydney system [20]. In addition, it is also known that in gastric IM, other factors could be linked to the occurrence of IM, notably the mucosal microbiota [21]. The most commonly reported factors were environmental factors, age, alcohol, tobacco, and diet [3] [4] [8] [17] [22]. Some of these factors were also studied in our study.

Epidemiological factors:

In the present study the average age of patients with IM was 50.9 ± 8.1 years with a high prevalence in patients belonging to [50 - 60] years. Theresa Nguyen *et al.* in the USA had reported a high prevalence of IM at 70 years old unlike our study where the patients were relatively younger in age.

Several data in the literature have revealed a correlation between certain socio-environmental factors and the appearance of IM. These included the advanced age of the patients, tobacco and alcohol consumption, ethnicity, race and low educational level, a family history of gastric cancer [3] [8] [9]. These data were different from those collected in our study, because we did not find an association between these different factors and the appearance of IM. This could be explained by several factors, the study contexts were not identical, the risk factors to which our patients were exposed would be different, our sample was relatively younger (average age of patients = 44.9).

Clinical factors:

Epigastralgia was a frequent reason for consultation in accordance with sever-

al data in the literature [8] [23] [24]. Indeed, epigastralgia was the main indication for gastroscopies. However, nowhere has an association between gastric clinical manifestations and IM been reported. However, our study found a significant association between the absence of IM and GERD (p = 0.004). The same was true for patients who declared having consumed PPIs over the long term (p = 0.004).

Comparison of samples:

The comparative study of sociodemographic and clinical parameters at inclusion of patients with IM and the group of patients without IM allowed us to establish that these two samples had similar exposure factors, with the exception of age of patients which was relatively higher in the group of patients with IM. Indeed, IM is a precancerous lesion which intervenes in the process of gastric carcinogenesis. It therefore appears several years after an *H. pylori* infection.

Clinically, the frequency of patients having taken long-term PPIs was lower in the group of patients with IM than in patients without IM. PPIs would certainly protect against IM. Paradoxically, it was the same for GERD. Indeed, hypochlorhydria would modify the conditions of the gastric environment, less favorable to the development of *H. pylori* which would have a promoting role in the genesis of gastric precancerous lesions, particularly IM. Our results were different from some data in the literature which reported that chronic use of PPIs masked *H. pylori* infection, increased the rate of inactive GC and increased the occurrence of IM [19].

5. Conclusion

In our study, the prevalence of intestinal metaplasia is relatively high at 11.8%. Chronic *H. pylori* gastritis is the main risk factor for intestinal metaplasia. Prolonged use of PPIs and a history of GERD were significantly identified as factors that would protect against intestinal metaplasia.

Authors Contribution

Hatrydt Guillaume Dimitri Kouamé wrote the article, made conception and design, data acquisition. Lisiane Mewetieh made data analysis and interpretation. Kouassi Olivier Claver Koffi filled and analyzed the database. Bathaix Fulgence Mamert Yao motivated the study and made the critical revision of the article.

Conflicts of Interest

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

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