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Epidemiological, Clinical, Endoscopic and Histological Aspects of *Helicobacter pylori* Chronic Gastritis in the Southern Region of Senegal

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Abstract

Introduction: *Helicobacter pylori (H. pylori*) chronic gastritis is an infectious disease causing chronic histological changes in the gastric mucosa. It is a ubiquitous condition, but is more prevalent in developing countries. The aim of our study was to determine the epidemiological, clinical, endoscopic and histological aspects of chronic *H. pylori* gastritis at the digestive endoscopy centre at the Ziguinchor Peace Hospital. **Materials and Methods:** This was a prospective, descriptive and analytical study conducted over a period of 7 months (July 2021 to January 2022). All patients aged 18 or over who underwent Oeso-Gastro-Duodenal Endoscopy (OGDE) and had histologically confirmed chronic *H. pylori* gastritis were included. **Results:** Of 298 patients who underwent OGDE with biopsies, 200 cases of chronic *H. pylori* gastritis were identified, representing an endoscopic prevalence of 67.1%. The mean age of the patients was 40 years [18 - 79 years]. Females predominated, with a sex ratio of 0.46. The indications for OGDE were dominated by epigastralgia (93.8%). The main lesions found on endoscopic examination were erosions of the gastroduodenal mucosa in 53.5% of cases, atrophic gastritis in 28% and ulcerations in 16.5%. Gastritis was antifundial in 91.5% of cases. Bacterial density was low in the majority of cases (57%). Histology showed mucosal atrophy in 88.5% of cases. Intestinal metaplasia (IM) was present in only 19 patients (9.5%). Only 3% of patients were classified as OLGA (Operative Link for Gastritis Assessment) III/IV and none as OLGIM (Operative Link for Gastritis Intestinal Metaplasia) III/IV. Low-

grade dysplastic lesions were noted in 5 patients (2.5%). There was no relationship between age, bacterial density and OLGA/OLGIM classifications. There was no endoscopic or histological correlation for the diagnosis of *H. pylori* chronic atrophic gastritis. **Conclusion:** The pandemic nature of *H. pylori* chronic gastritis and its impact on the genesis of gastric adenocarcinoma are a worldwide concern, making its treatment and the monitoring of high-risk patients a real public health challenge.

Keywords

Chronic Gastritis, *Helicobacter pylori*, Olga, Olgim, Gastric Cancer

1. Introduction

Chronic gastritis is a persistent inflammatory disease of the gastric mucosa. It is a histological entity defined by the presence of an abnormally dense infiltrate of inflammatory cells (lymphoplasmocytes, sometimes neutrophils) in the chorion of the gastric mucosa [1].

There are many causes of chronic gastritis, dominated by *Helicobacter pylori* (*H. pylori*) infection, particularly in Sub-Saharan Africa [2].

Chronic *H. pylori* gastritis is usually asymptomatic and is diagnosed with certainty on histological examination of gastric biopsies.

The Sydney classification is a recognised tool for characterising histological lesions in *H. pylori* chronic gastritis [3]. It is used in conjunction with the OLGA (Operative Link for Gastritis Assessment) and OLGIM (Operative Link for Gastritis Intestinal Metaplasia) prognostic classifications to stratify the risk of cancer and plan surveillance of chronic gastritis [4] [5]. Eighty-five percent of gastric adenocarcinomas are secondary to infection with *H. pylori*, which is the world's leading cause of neoplasia of infectious origin (810,000 cases/year) [6]. *H. pylori* was declared a class I carcinogen by the WHO in 1994 [7].

The general objective of our study was to contribute to a better understanding of chronic *H. pylori* gastritis in southern Senegal.

2. Materials and Methods

This was a prospective descriptive and analytical study of the epidemiological, clinical, endoscopic and histological aspects of *H. pylori* chronic gastritis. The study covered the period from July 2021 to January 2022, for a period of 7 months. The study population consisted of patients who underwent Oeso-Gastro-Duodenal Endoscopy (OGDE) at the digestive endoscopy centre of the Hôpital de la Paix in Ziguinchor during the study period.

Patients were included after obtaining written informed consent:

- 18 years of age or older,
- Histologically confirmed chronic *H. pylori* gastritis.

Not included:

- Patients under 18 years of age,
- Patients with chronic histological gastric disease without evidence of *H. pylori*.

We chose to specifically include chronic gastritis with evidence of *Helicobater Pylori*. Chronic active gastritis without the presence of *Helicobater Pylori* on histology was excluded, even though we are not certain that it is not linked to *Helicobater Pylori*, but in the context of our work, we only have standard histology to confirm the presence of the bacterium.

During the OGDE, after careful examination of the mucosa, biopsies were taken according to the Sydney protocol for anatomopathological study. The samples were placed in two separate jars containing formalin and sent to the pathology department of the Hôpital de la Paix in Ziguinchor. The specimens were fixed with 10% formalin and embedded in paraffin. Staining was performed with Haematein-Eosin (HE) and modified Giemsa.

Microscopy was performed by a pathologist. Chronic gastritis was classified according to the revised Sydney system and the OLGA and OLGIM classifications. The pathologists used histological evaluation of the intensity of lesions of atrophy (OLGA) and intestinal metaplasia (OLGIM) in the fundic and antral mucosa from biopsies taken (two from the antrum, one from the angulus and two from the fundus), with a grading ranging from stage 0 (no atrophy/metaplasia) to stage IV (significant atrophy or metaplasia).

For bacterial density, pathologists relied on microscopic assessment: a low density of *Helicobacter pylori* was defined as the presence of a few isolated bacteria or small groups of bacteriad in the crypts, a moderate density by the presence of more bacteria, sometimes grouped in clusters, affecting several microscopic fields, and a high density by a large number of bacteria, often on the surface and at depth.

A survey form was used to collect the following information: marital status, family and personal history, clinical aspects, OGDE results, and histology of biopsies.

Data were entered using Sphinx software version 5.1.0.2. Data analysis was performed using SPSS (Statistical Package for Social Sciences) version 18. The descriptive study involved calculating frequencies and proportions for the qualitative variables and means and standard deviations for the quantitative variables. The analytical study was carried out using cross-tabulations. To compare frequencies, we used Pearson's Chi-square test or Fisher's two-tailed exact test, depending on their applicability, with a significance threshold of $p < 0.05$.

3. Results

During the study period, 332 patients were seen at the centre for upper GI endoscopy. Of these, 298 underwent biopsy sampling according to the Sydney protocol. Histological examination revealed chronic gastritis in 262 patients (87.9%). Sixty-two patients whose histology did not show the presence of *H. pylori* were not included.

The study therefore included 200 patients with chronic *H. pylori* gastritis, giving a prevalence of 67.1%.

The mean age of the patients was 40 years, with extremes of 18 and 79 years. Women predominated, with a sex ratio of 0.46 (137 women). The majority of patients lived in the Ziguinchor region (74.5%). However, 39 patients (19.5%) lived in Guinea-Bissau. Most patients (64.5%) had no fixed income. Patients lived in families with an average of 9 people in the house.

The most frequent clinical manifestations were epigastralgia (93.8%), gastro-oesophageal reflux disease (53.7%) and dyspepsia (25.5%) (Figure 1).

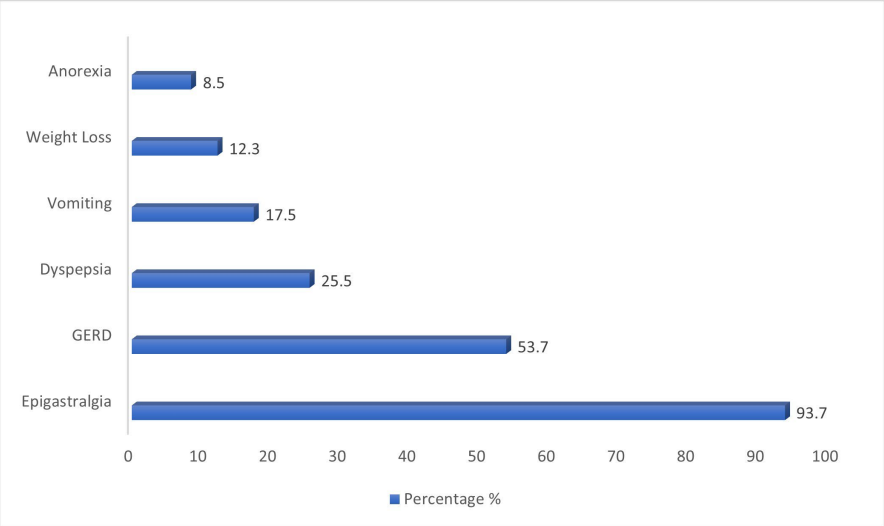

Figure 1. Distribution of patients according to clinical signs.

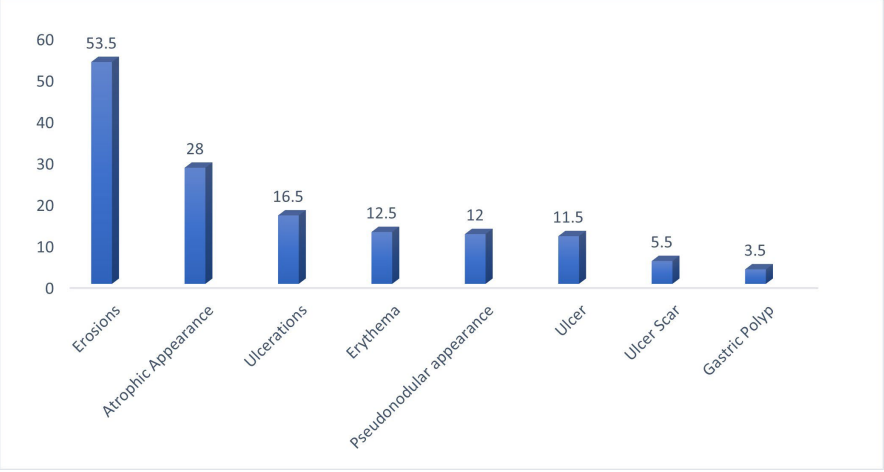

Figure 2. Frequency of different gastric lesions on OGDE.

OGDE was normal in 19 patients (9.5% of cases). Endoscopy showed various mucosal lesions, 83.8% of which were located in the antrum. Gastroduodenal erosions were the most frequently found lesions. They were present in 107 patients (53.5%). Gastric or duodenal ulcers were noted in 23 patients (11.5%). Figure 2 shows the frequency of different gastric lesions on OGDE.

Other abnormalities were detected on OGDE. Cardiac incontinence was the most common, affecting almost half the patients (43.4%).

Chronic gastritis with a lymphoplasmacytic inflammatory infiltrate was found in all patients. Pan gastritis was present in 91.5% of cases.

Gastritis was moderate in the majority of cases (63.5%). Eighteen patients had severe chronic gastritis.

Chronic gastritis was active in almost all patients (97.6%).

Bacterial density was low in 57% of cases. Chronic gastritis was atrophic in 177 patients (88.5% of cases). It was predominantly antro-fundiacular (69.5%).

According to the OLGA classification, atrophy was stage I in 56.5% of cases (Table 1).

Table 1. Distribution of patients according to OLGA stage.

OLGA Stage	Number	Percentage (%)
0	23	11.5
I	113	56.5
II	58	29
III	6	3
IV	0	0

Intestinal metaplasia was observed in 19 patients. The majority (89%) were found in the antrum.

Five patients (2.5%) had dysplastic lesions. All were of low grade.

Of the patients who did not have an atrophic appearance on OGDE (119), histology allowed the diagnosis of atrophic gastritis in 110 patients (92.4%). There was no correlation between endoscopy and histology (Table 2).

Table 2. Distribution of patients according to atrophic appearance on OGDE and atrophy on histology.

Atrophic appearance		Atrophy		Total	P
		Yes	No		
Yes	Number	63	10	73	0.167
	%	36.4%	52.6%	38.0%	
No	Number	110	9	119	
	%	63.6%	47.4%	62.0%	

4. Discussion

The digestive endoscopies in our study are not performed every day, and there are frequent stoppages due to technical factors, which may explain the size of our sample.

The prevalence of chronic *H. pylori* gastritis was 67.1% in our study. High prevalences were found in Africa and Asia, in contrast to northern countries [8]-[18]. This prevalence of chronic gastritis is similar to that of *H. pylori* infection, which

is higher in developing countries. This can be explained by the low standard of living (promiscuity, lack of drinking water) in these countries. Most of our patients had no fixed income (64.5%) and lived in overcrowded conditions with an average of 3 people per room.

The average age was 40 and 70.7% of patients were under 50. This is comparable to the results reported elsewhere in Senegal (40 years), Togo (45.5 years) and Côte d'Ivoire (37.5 years). Infection with *H. pylori* occurs early in Africa, from childhood, via the faecal-oral route, which may explain the predominance of infection in young adults. On the other hand, in developed countries, due to improved living conditions, the prevalence is very low in young subjects (less than 10%) [19].

A predominance of females was observed in our study, with a sex ratio of 0.46. Bamba *et al.* in Senegal and Doffou *et al.* in Côte d'Ivoire reported a sex ratio of 0.63 and 0.79, respectively [11] [12]. However, a male predominance has been reported in other studies in Africa and the West [13] [20].

These results show that gender is not a factor in *H. pylori* chronic gastritis. Infection with the bacterium is not linked to individual characteristics but mainly to hygiene and socio-economic conditions.

Epigastralgia was the main indication for upper GI endoscopy (93.8%). Dyspepsia was present in 25.5% of cases. Our results are in line with those found in previous African studies [10]-[12] [14]. Chronic gastritis is usually asymptomatic. However, it is generally associated with various lesions related to *H. pylori* infection, which may explain this symptomatology.

The majority of patients (53.7%) complained of symptoms of GERD and were therefore subject to prolonged use of PPIs. Chronic *H. pylori* gastritis associated with long-term PPI use has been shown to be a risk factor for gastric cancer [21]. It is therefore essential to always perform biopsies during EOGD in patients with GERD to look for *H. pylori* in order to eradicate it.

In our study, the prevalence of GDU was 11.5%. In Senegal, recent studies reported a prevalence of GDU of 19.8% and 6% [10] [11]. These data confirm the clear decline in the prevalence of GDU that was demonstrated in 2010 by Diouf *et al.*, with a 12% drop over a decade [22]. This downward trend in PUD in Africa is probably linked to improved hygiene and better access to *H. pylori* eradication treatment. The use of PPIs prior to OGDE, which is now almost routine in patients (85.5%), leads to temporary healing of lesions and false negatives.

In our study, as in that of Jmaa *et al.* in Tunisia [23], all patients had an inflammatory infiltrate which was moderate in the majority of cases (63.5%) and antro-fundial in location (91.5%). However, an antral predominance was found by other authors [11] [24]. The difference in biopsy sites could explain this discrepancy. In these studies, only antral biopsies were usually performed, in contrast to our own study in which the Sydney protocol was followed.

Chronic gastritis was active in almost all patients (97.5%). This activity noted on histology is related to the presence of *H. pylori* on the gastric mucosa. Once acquired, the bacterium is thought to be responsible for the development of defense

mechanisms, including the proliferation of neutrophils. PNNs in the chorion and/or epithelium define the activity criterion constantly associated with the presence of the germ [25]. In our study, 62 patients who had chronic active gastritis with the presence of PNN in the mucosa, but no germ were included. However, 54.8% of these patients had severe chronic gastritis. In accordance with the Maastricht VI recommendation, Immunohistochemical (IHC) staining is required before concluding that *H. pylori* is not present [26], which was not the case in our series, where only standard and modified Giemsa stains were used to test for *H. pylori*. Thus, a higher prevalence of *H. pylori* gastritis would probably have been found with IHC. Immunohistochemistry would allow us to detect the presence of very small quantities of bacteria in the mucosa, which simple histology cannot do.

In our series, almost 10% of patients had normal gastric mucosa on OGDE. However, histology was consistent with chronic *H. pylori* gastritis. Doh *et al*. found chronic *H. pylori* gastritis in 61% of patients with normal GI endoscopy [9]. Furthermore, our study showed that there was no concordance between the results of OGDE and histology for the diagnosis of chronic atrophic gastritis. There is little correlation between endoscopy and histology findings [9] [11] [27] [28]. This underlines the importance of always taking gastric biopsies during digestive endoscopy, even in the absence of mucosal lesions.

Bacterial density was mild in the majority of cases (57%). Only 8.5% of patients had severe density. Tagzout *et al*. had similar results [29]. This could be explained by the almost constant use of PPIs before EOGD found in our patients.

Gastric atrophy and IM are precancerous lesions that constitute links in the Corréa cascade leading to gastric adenocarcinoma [30]. In our study as well as in those of Diallo in Dakar and Doffou in Ivory Coast, a high prevalence of gastric atrophy was observed with 88.5%, 84.5% and 81.5% respectively [10] [12]. This atrophy was more frequent in elderly subjects in our series. Previous studies have shown that there is a significant correlation between atrophy and age over 50 years, regardless of *H. pylori* infection [31]. In reality, with age, histological changes are observed with rarefaction of the gastric glands which are replaced by fibrosis.

This chronic atrophic gastric disease requires monitoring, especially since it is predominantly antrofundic in our series. Indeed, atrophic pangastritis constitutes a higher risk of gastric cancer compared to antral involvement alone [30].

Gastric IM is significantly associated with chronic *H. pylori* gastritis. A Japanese study of 2445 patients showed that IM was 43.1% in *H. pylori*-positive patients versus 6.2% in *H. pylori*-negative patients [32]. However, the prevalence of MI varies between countries. In our series, 19 patients or 11% had mild MI, mainly of antral location. These results are similar to those obtained in other countries with a low risk of gastric cancer [10] [11] [23]. On the other hand, in populations at high risk of gastric cancer, such as Japan or China, the frequency of gastric MI is on average 30% [13] [32] [33].

Patients classified as OLGA/OLGIM 0, I, II were considered at low risk for gastric cancer, and those classified as OLGA/OLGIM III, IV were at high risk [15]

[16]. In our study, only 6 patients were classified as high risk (stage III) according to the OLGA classification and none according to the OLGIM classification.

Five patients presented with low-grade dysplasia. This is consistent with the results obtained in other African series [11] [29] where low rates of dysplasia in chronic *H. pylori* gastritis were noted. In Africa, despite the high presence of the carcinogenic bacterium *H. pylori* [2] [34], the incidence of gastric cancer remains low (3%): this is the "African enigma" [35].

5. Conclusion

Chronic *H. pylori* gastritis represents a major public health issue due to its high prevalence and its involvement in the pathogenesis of numerous gastroduodenal diseases, including gastric cancer. Upper gastrointestinal endoscopy has low sensitivity for diagnosing chronic *H. pylori* gastritis. It is therefore imperative to always perform gastric biopsies. In Senegal, as in several African countries, there is a paradox between the high prevalence of chronic *H. pylori* gastritis and the low prevalence of precancerous lesions and gastric cancer. Further studies appear necessary to understand this phenomenon.

Conflicts of Interest

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

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