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Correlation between Clinical, Endoscopic, and Anatomopathological Aspects of Chronic Gastritis: Retrospective Study of 600 Cases at CNHU-HKM Cotonou (Benin)

Falilatou Seidou[1*], Dominique Raoul Hebel Gomez[1], Antoine Houndonougbo[1,2], Martin Sokpin[3], Fidelia Nihad Da Silva[1], Alexis Gédéon Houessou-Yovo[1], Déo-Gracias Houmènou[1], Maimounatou Atindehou[1], Jude Agonhoundjè[1], Luc Valère Codjo Brun[4], Marie Thérèse Akele-Akpo[1]

[1]Clinique Universitaire d'Anatomie, de Cytologie Pathologiques (CU-ACP), Centre National Hospitalier Universitaire Hubert Koutoukou Maga (CNHU-HKM) de Cotonou, Cotonou, Bénin
[2]Laboratoire des Fièvres Hémorragiques Virales et des Arbovirus, Cotonou, Bénin
[3]Clinique Universitaire d'Hépato Gasto Entérologie du CNHU HKM, Cotonou, Bénin
[4]CHU Borgou/Alibori, Parakou, Bénin
Email: *sfalilatou@ymail.com

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Abstract

Introduction: Chronic gastritis is a multifactorial condition whose management requires clinical, endoscopic, and histological analysis. This study aimed to evaluate the relationships among these three aspects in 600 patients, highlighting the dominant characteristics and their associations. **Methodology**: Data from 600 cases of chronic gastritis were collected, including epidemiological, clinical, endoscopic, and histological characteristics (Sydney System). Statistical analyses were performed to assess correlations between these parameters. **Results:** This study included as many women (52.33%) as men (47.0%). Clinically, epigastric pain (one-third of patients) and dysphagia (one-sixth of patients) were the most common symptoms. Endoscopy revealed antral or fundal mucosal congestion in approximately half of the patients, while a quarter presented with specific lesions such as erythema, nodules, or ulcers. Histologically, the majority of cases presented with mild activity and moderate or severe inflammation, regardless of endoscopic findings. Atrophy was moderate, while metaplasia and lymphoid follicles were rare. *H. pylori* density was generally mild. Despite these observations, no significant correlation was found between clinical, endoscopic, and histological findings ($p > 0.05$). **Conclusion:** This study highlights the lack of a statistically significant relationship between clinical, endoscopic, and histolog-

ical investigations in chronic gastritis. Endoscopic findings do not always reflect the severity of histological or clinical abnormalities. These findings highlight the importance of a multidisciplinary approach in the management of chronic gastritis.

Keywords

Chronic Gastritis, Sydney-System, Correlation, Endoscopy, Histology

1. Introduction

Chronic gastritis is one of the most common, serious, and insidious diseases in humans. Despite a significant decrease in the global incidence and prevalence of gastritis, this condition continues to impose a burden on the population, particularly in less developed regions. It can be estimated that more than half of the world's population is affected by this disease to some degree or another. Thus, several hundred million people worldwide may suffer from chronic gastritis in one form or another [1]. Chronic gastritis is a persistent inflammatory disease of the gastric mucosa, the etiology of which is dominated by *Helicobacter pylori* infection, particularly in low- and middle-income countries [2], and by an autoimmune reaction. Clinically, it manifests in a heterogeneous manner, ranging from nonspecific digestive symptoms (dyspepsia, epigastric pain, nausea) to more serious complications such as iron deficiency anemia or weight loss [3]. This polymorphic symptomatology often makes its diagnosis difficult and late.

Upper gastrointestinal endoscopy can objectify certain abnormalities of the gastric mucosa such as erythema, nodularity, erosion, or atrophy, but these images remain non-specific and their interpretation is subject to inter-observer variability [4]. Histological evaluation from biopsies therefore remains essential to characterize gastric lesions accurately. The recommendations of the Sydney System, the OLGA (Operative Link for Gastritis Assessment), and OLGIM (Operative Link on Gastric Intestinal Metaplasia) classifications make it possible to quantify inflammation, glandular atrophy, intestinal metaplasia, and *Helicobacter pylori* infection, while stratifying the risk of progression to gastric cancer [5] [6].

Several studies have highlighted poor concordance between clinical, endoscopic, and histopathological observations [7] [8]. This discordance can lead to an underestimation of the risk of progression in some patients and compromise appropriate management. In the Beninese context, and more specifically at the Hubert Koutoukou Maga National University Hospital (CNHU-HKM) in Cotonou, few studies have systematically explored this tripartite correlation, even though a better understanding of the interactions between these dimensions could strengthen diagnostic and therapeutic relevance.

The present study thus aims to evaluate the correlation between the clinical, endoscopic, and histopathological aspects of chronic gastritis treated at the CNHU-HKM in Cotonou, in order to provide an integrated and contextually relevant interpretation of this digestive pathology.

2. Methodology

This is a descriptive, retrospective, observational study conducted at the University Clinics of Hepato-Gastroenterology and Pathological Anatomy and Cytology of the Hubert Koutoukou Maga National University Hospital (CNHU-HKM) in Cotonou, a leading tertiary care facility in Benin. The study covered a 20-month period, from April 2023 to December 2024.

All patients of any age who underwent Esophagogastroduodenoscopy (EGD) during the study period and were diagnosed with chronic gastritis based on histopathological examination of gastric biopsies were included in the study. Inclusion criteria required the simultaneous availability of a complete clinical record (history, symptoms), an endoscopic report, and a pathological examination prepared according to the Sydney System recommendations. For each included patient, the biopsy was necessarily from both antral and fundic mucosa. Also, the number of samples per mucosa was at least two. Patients with unusable histological samples or incomplete clinical or endoscopic data were excluded.

The data collected included:

- Epidemiological characteristics (age, sex);
- Clinical manifestations (epigastric pain, dyspepsia, vomiting, hematemesis, haematochezia, melena, gastroesophageal reflux, belching, heartburn, etc.);
- Endoscopic characteristics (appearance of the gastric mucosa: erythema, nodularity, atrophy, erosions) and topographical features (fundus and antrum);
- Histological data (presence and intensity of chronic inflammation, inflammatory activity, glandular atrophy, intestinal metaplasia, presence of lymphoid follicles, and presence of *H. pylori*) are assessed according to the Sydney, OLGA, and OLGIM systems. Using this system, pathologists assign to each feature a score based on the visual scale, ranging from 0 (none), 1 (mild), 2 (moderate), to 3 (severe).

The data were entered using Microsoft Excel and analysed using Sigma Plot V15 software. Quantitative variables are expressed as mean ± standard deviation. Clinical variables were coded as binary (presence = 1, absence = 0) for each of the eight clinical signs. Endoscopic profiles (6 categories) were considered as dependent variables in a multiple logistic regression analysis, with each clinical sign included as an explanatory variable. The six histological parameters of the Sydney system were coded into four ordinal levels of severity (absent, mild, moderate, severe). Associations between clinical signs and histological parameters, as well as between endoscopic profiles and histological parameters, were assessed using the chi-square test with two degrees of freedom. The significance level was set at 5%.

3. Results

3.1. Epidemiological and Clinical Characteristics of Patients

The study included 600 patients admitted to the CNHU-HKM in Cotonou, with a balanced gender distribution (52.33% women and 47.67% men). The mean age was 47.7 ± 17.21 years (Table 1).

The most frequently reported symptoms were epigastric pain (32.33%), followed by dysphagia and dyspepsia (17.83%). Hematemesis and melena (7.33%), gastroesophageal reflux disease (5.17%), vomiting (3.50%), heartburn (1.33%), and belching (1%) were the least common (Table 1).

Table 1. Epidemiological and clinical characteristics of patients.

Characteristics	Mean	Deviation
Age	47.7	17.21
	Number	**Frequency (%)**
Sex		
Male	286	47.67
Female	314	52.33
Clinical Signs		
Epigastric pain (A)	194	32.33
Dysphagia et dyspepsia (B)	107	17.83
Vomiting (C)	21	3.50
Hematemesis, Haematochezia, and Melena (D)	44	7.33
Gastroesophageal Reflux (E)	31	5.17
Eructation (F)	6	1.00
Heartburn (G)	8	1.33
Others (H)	204	34.00

3.2. Endoscopic Aspects

Endoscopy revealed major abnormalities affecting both the antral and fundal mucosa. Erythema and/or petechiae were the most common lesions, occurring in 59% of the antrum and 59.83% of the fundal mucosa (Figure 1). Erosions and/or ulcers were present in 29.33% of patients in the antrum and 21.33% in the fundus (Table 2).

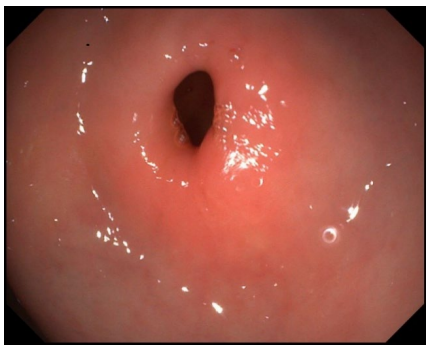

Figure 1. Endoscopic appearance of an oedematous gastric mucosa.

In cases of erythema, the stomach lining appears red, either diffusely or more localized. It may appear thicker than normal, sometimes with an oedematous (swollen) appearance.

Table 2. Distribution according to endoscopic aspects.

Endoscopic aspects	Antral mucosa		Fundic mucosa	
	Number	Frequency (%)	Number	Frequency (%)
Congestion and/or Oedema	28	4.67	97	16.17
Erythema and/or Petechia	354	59	359	59.83
Erosion and/or Ulcer	176	29.33	128	21.33
Atrophy, effacement of folds, and/or whitish appearance	144	24	76	12.67
Nodule and/or concentration of folds	6	1	12	2
Normal	152	25.33	17	2.83

It is important to note that, since endoscopic features are not mutually exclusive, the antral or fundic mucosa of the same patient may present two or more concomitant features. This explains the total number of cases exceeding 600 patients included and the percentages exceeding 100%.

Atrophy and a whitish appearance of the folds were observed in 24% of patients in the antral mucosa and 12.67% in the fundal mucosa (Figure 2). The nodular appearance and concentration of the folds were rarer, at 1% for the antrum and 2% for the fundus (Table 2).

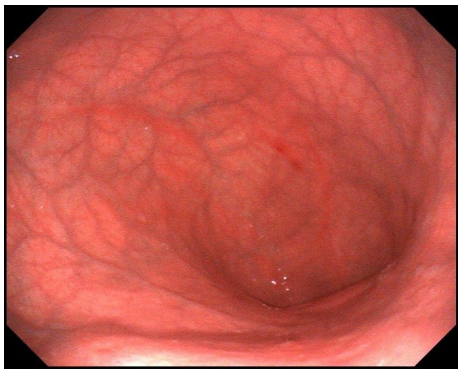

Figure 2. Endoscopic appearance of atrophic gastric mucosa.

Endoscopy with gastric atrophy may present with pale mucosa, increased visibility of blood vessels due to thinning of the mucosa, and loss of gastric folds.

3.3. Microscopic Aspects

Inflammation in the fundus was moderate in 66.8% of cases, while in the antrum, it was more often severe (45.7%). Regardless of the anatomical location, the presence of lymphoid follicles and intestinal metaplasia remained uncommon (Figure

3). As for activity, it was mostly mild, and atrophy was moderate in both mucosae (Figure 4). *Helicobacter pylori* density was low in nearly half of the samples (Table 3).

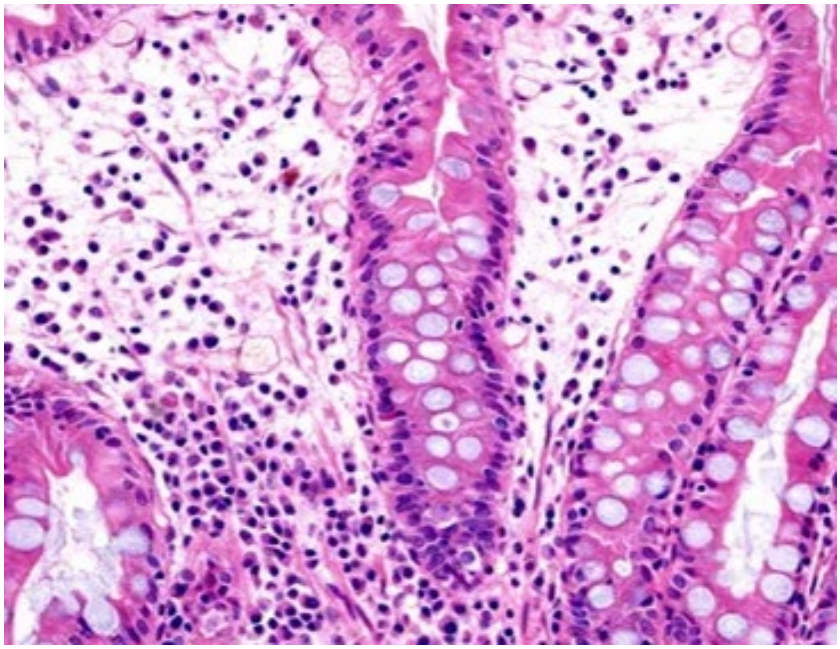

Figure 3. HEX100: Histological appearance of intestinal metaplasia of the gastric mucosa.

Intestinal metaplasia of the gastric mucosa is an abnormal transformation of the stomach lining into tissue resembling that of the intestine. Histologically, intestinal cells, such as goblet cells and absorptive cells, are observed in place of normal gastric cells.

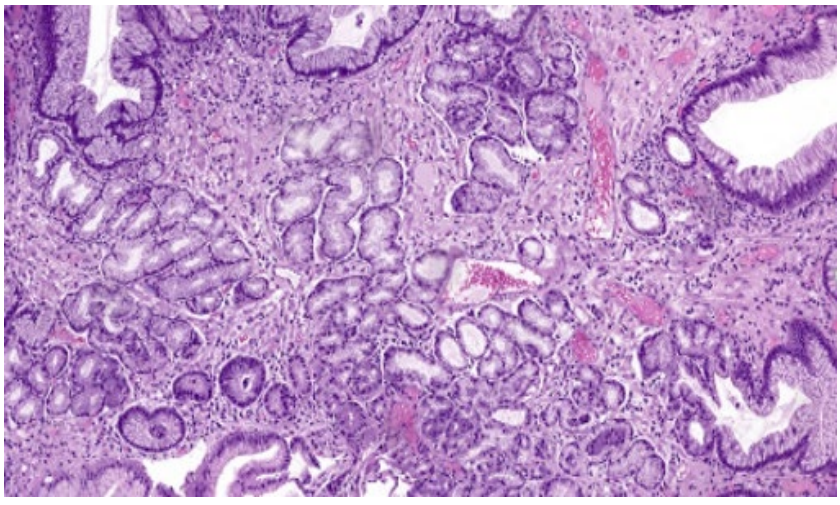

Figure 4. HEX40: Histological appearance of gastric mucosal atrophy.

Histologically, gastric mucosal atrophy is characterized by a decrease in the number of gastric glands and thinning of the mucosa.

Table 3. Distribution according to microscopic aspects.

Mucosa and Sydney System parameters	Lesion intensity							
	Absent		Mild		Moderate		Severe	
Antrum *Number* **%** *Number* **%** *Number* **%** *Number* **%**								
Activity	62	10.3	525	87.5	11	1.8	2	0.3
Atrophy	83	13.8	49	8.2	389	64.8	79	13.2
Inflammation	0	0	68	11.3	258	43	274	45.7
Lymphoid follicles	525	87.5	72	12	3	0.5	0	0
Metaplasia	571	95.2	17	2.8	5	0.8	7	1.2
H. pylori density	69	11.5	269	44.8	149	24.8	113	18.8
Fundus								
Activity	104	17.3	490	81.7	5	0.8	1	0.2
Atrophy	134	22.3	153	25.5	307	51.2	6	1
Inflammation	22	3.7	104	17.3	401	66.8	73	12.2
Lymphoid follicles	578	96.3	21	3.5	0	0	1	0.2
Metaplasia	591	98.5	7	1.2	2	0.3	0	0
H. pylori density	241	40.2	260	43.3	67	11.2	32	5.3

3.4. Distribution of Clinical Signs According to Endoscopic Features

Congestion and/or oedema were the most common endoscopic features, regardless of the clinical sign and mucosa. The majority of patients complained of dysphagia and dyspepsia when the antral mucosa was normal on endoscopy. They most often complained of epigastric pain when the fundal mucosa was normal on endoscopy (Table 4).

Table 4. Distribution of clinical signs according to endoscopic aspects.

	Clinical signs	A	B	C	D	E	F	G	H
Endoscopic appearances of the antral mucosa	Normal	7 (2.6)	11 (7.3)	1 (3.2)	3 (5)	1 (2.1)	0 (0)	1 (8.3)	6 (2)
	Congestion and/or Oedema	121 (44.6)	63 (41.7)	9 (29)	21 (35)	19 (39.6)	4 (80)	5 (41.7)	118 (39.7)
	Erythema and/or Petechia	48 (17.7)	28 (18.5)	7 (22.6)	12 (20)	12 (25)	0 (0)	3 (25)	68 (22.9)
	Erosion and/or Ulcer	48 (17.7)	22 (14.6)	5 (16.1)	19 (31.7)	6 (12.5)	0 (0)	1 (8.3)	46 (15.5)
	Atrophy*	2 (0.7)	2 (1.3)	0 (0)	0 (0)	0 (0)	0 (0)	1 (8.3)	2 (0.7)
	Nodule and/or concentration of folds	45 (16.6)	25 (16.6)	9 (29)	5 (8.3)	10 (20.8)	1 (20)	1 (8.3)	57 (19.2)
Endoscopic appearances of the fundic mucosa	Normal	32 (14.4)	13 (10.2)	1 (5.3)	9 (16.7)	7 (20.6)	2 (50)	1 (12.5)	49 (14.6)
	Congestion and/or Oedema	122 (55)	72 (56.3)	11 (57.9)	23 (42.6)	16 (47.1)	2 (50)	4 (50)	170 (50.7)
	Erythema and/or Petechia	31 (14)	25 (19.5)	6 (31.6)	8 (14.8)	6 (17.6)	0 (0)	3 (37.5)	72 (21.5)
	Erosion and/or Ulcer	27 (12.2)	12 (9.4)	1 (5.3)	13 (24.1)	4 (11.8)	0 (0)	0 (0)	27 (8.1)
	Atrophy*	4 (1.8)	1 (0.8)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	8 (2.4)
	Nodule and/or concentration of folds	6 (2.7)	5 (3.9)	0 (0)	1 (1.9)	1 (2.9)	0 (0)	0 (0)	9 (2.7)

Keys: A: Epigastric pain; B: Dysphagia and Dyspepsia; C: Vomiting; D: Hematemesis, Haematochezia, and Melena; E: Gastroesophageal Reflux; F: Eructation; G: Heartburn; H: Others; *Atrophy, effacement of folds, and/or whitish appearance.

3.5. Distribution of Endoscopic Findings According to Histological Features

While in the majority of cases, regarding the antral mucosa, inflammation was moderate, it should be noted that it was more often severe when endoscopy revealed congestion, oedema, nodules, or concentration of folds. Similarly, the density, more often light, was found to be moderate, particularly in cases of atrophy, effacement of folds, or a whitish appearance (Table 5).

Table 5. Endoscopic and microscopic aspects of the antral mucosa.

	Endoscopic appearances of antral mucosa					
	Normal	Congestion and/or Oedema	Erythema and/or Petechia	Erosion and/or Ulcer	Atrophy, effacement of folds, and/or whitish appearance	Nodule and/or concentration of folds
Number of cases	28/4.67	354/59	176/29.33	144/24	6/1	152/25.33
Mean Age (years)	44.75 ± 18.43	47.91 ± 16.69	44 ± 16.81	53.66 ± 17.07	53.33 ± 18.89	45.01 ± 17.30
Sex (H/F)	9/19	177/177	66/110	66/78	5/1	76/76
Inflammation						
Absent (case/%)	0/0	0/0	0/0	0/0	0/0	0/0
Mild (case/%)	4/14.29	38/10.73	24/13.64	12/8.33	1/16.67	14/9.21
Moderate (case/%)	16/57.14	142/40.11	76/43.18	67/46.53	4/66.67	57/37.5
Severe (case/%)	8/28.57	174/49.15	76/43.18	65/45.14	1/16.67	81/53.29
***H. pylori* density**						
Absent (case/%)	5/17.86	39/11.02	21/11.93	12/8.33	0/0	16/10.53
Mild (case/%)	12/42.86	151/42.66	84/47.73	69/47.92	2/33.33	64/42.11
Moderate (case/%)	7/25	91/25.71	40/22.73	37/25.69	4/66.67	41/26.97
Severe (case/%)	4/14.29	73/20.62	31/17.61	26/18.06	0/0	31/20.39

As for the fundic mucosa, while inflammation was moderate in the majority of cases, endoscopic aspects of congestion, oedema, erythema, or petechiae were more often noted in severe inflammation. Also, the density was more often moderate for endoscopic aspects of nodules or concentration of folds, while it was mostly light in other endoscopic aspects (Table 6).

3.6. Distribution of Microscopic Features According to Clinical Signs

Inflammation was more often severe when patients complained of epigastric pain, dysphagia, dyspepsia, or hematemesis. It was more often moderate in cases of vomiting, haematochezia and melena, gastroesophageal reflux, or eructation (Table 7).

Table 6. Endoscopic and microscopic aspects of the fundic mucosa.

	Endoscopic appearances of the fundic mucosa					
	Normal	Congestion and/or Oedema	Erythema and/or Petechia	Erosion and/or Ulcer	Atrophy, effacement of folds, and/or whitish appearance	Nodule and/or concentration of folds
Number of cases	97	359	128	76	12	17
Mean Age (years)		47.43 ± 17.16	47.53 ± 16.27	51.05 ± 15.15	60.08 ± 15.68	54.29 ± 18.19
Sex (H/F)		174/184	57/71	38/38	5/7	6/11
Inflammation						
Absent (case/%)	0/0	0/0	0/0	0/0	0/0	0/0
Mild (case/%)	14/14.43	35/9.75	21/16.41	8/10.53	1/8.33	0/0
Moderate (case/%)	47/48.45	147/40.95	44/34.38	41/53.95	6/50	9/52.94
Severe (case/%)	36/37.11	177/49.3	63/49.22	27/35.53	5/41.67	8/47.06
***H. pylori* density**						
Absent (case/%)	10/10.31	38/10.58	20/15.63	7/9.21	0/0	1/5.88
Mild (case/%)	49/50.52	148/41.23	55/42.97	36/47.37	7/58.33	6/35.29
Moderate (case/%)	24/24.74	92/25.63	29/22.66	19/25	4/33.33	7/41.18
Severe (case/%)	14/14.43	81/22.56	24/18.75	14/18.42	1/8.33	3/17.65

Table 7. Microscopic aspects of lesions according to clinical signs.

	Clinical signs							
	A	**B**	**C**	**D**	**E**	**F**	**G**	**H**
Number of cases	194	107	21	44	31	5	8	204
Mean Age (years)	43.01±17.18	50.77±15.37	46.61±19.21	53.65±17.82	44.12±15.35	40.4±4.92	38.25±17.67	50.88±16.82
Sex (H/F)	80/114	51/57	10/11	27/17	17/14	2/3	4/4	100/104
Inflammation								
Absent (case/%)	0/0	0/0	0/0	0/0	0/0	0/0	0/0	0/0
Mild (case/%)	21/10.82	4/3.74	4/19.05	5/11.36	6/19.35	1/20	2/25	25/12.25
Moderate (case/%)	83/42.78	46/42.99	14/66.67	17/38.64	16/51.61	3/60	4/50	83/40.69
Severe (case/%)	90/46.39	57/53.27	3/14.29	22/50	9/29.03	1/20	2/25	96/47.06

Keys: A: Epigastric pain; B: Dysphagia and dyspepsia; C: Vomiting; D: Hematemesis, haematochezia and melena; E: Gastroesophageal reflux; F: Eructation; G: Heartburn; H: Others.

3.7. Correlation between Clinical, Endoscopic, and Histological Features

In most cases, the distributions across the different aspects appeared to be independent. Therefore, the analysis of correlations between clinical and endoscopic findings, between endoscopic and histological findings, and between clinical and histological findings did not reveal any statistically significant relationship ($p > 0.05$). However, some convergences did emerge, as mentioned above (see also Ta-

bles 8-10).

Table 8. Association between clinical signs and endoscopic findings. Only the p-values from statistical tests are shown (multiple logistic regression).

Clinical signs	Endoscopic appearances					
	Congestion and/or Oedema	Erythema and/or Petechia	Erosion and/or Ulcer	Atrophy, effacement of folds, and/or whitish appearance	Nodule and/or concentration of folds	Normal
				Antral mucosa		
A	0.473	0.855	0.663	0.739	0.746	0.467
B	0.547	0.583	0.482	0.771	0.614	0.899
C	0.191	0.695	0.516	0.998	**0.012***	0.994
D	0.14	0.947	0.018	0.992	0.224	0.995
E	0.327	0.172	0.678	0.992	0.694	0.975
F	0.998	0.998	0.998	1	0.874	0.999
G	0.701	0.998	0.998	1	0.998	0.35
H	0.698	0.199	0.795	0.955	0.532	0.971
				Fundic mucosa		
A	0.5	0.195	0.646	0.97	0.92	0.711
B	0.299	0.565	0.899	0.441	0.324	**0.005**
C	0.092	0.144	0.995	0.998	0.998	**0.006**
D	0.499	0.795	**0.014**	0.992	0.964	0.431
E	0.405	0.385	0.843	0.995	0.879	0.524
F	0.998	0.998	0.998	0.999	0.999	0.998
G	0.998	0.998	0.998	0.999	0.999	0.998
H	0.78	0.096	0.47	**0.027**	0.854	0.324

Keys: A: Epigastric pain; B: Dysphagia and dyspepsia; C: Vomiting; D: Hematemesis, haematochezia, and melena; E: Gastroesophageal reflux; F: Eructation; G: Heartburn; H: Others.

Table 9. Associations between Sydney System parameters and endoscopic findings. Only p-values are shown. (χ^2 test, df = 2)

Sydney System parameters	Endoscopic appearances					
	Congestion and/or Oedema	Erythema and/or Petechia	Erosion and/or Ulcer	Atrophy, effacement of folds, and/or whitish appearance	Nodule and/or concentration of folds	Normal
				Antral mucosa		
Activity	0.428	0.432	0.325	0.458	0.211	-
Atrophy	0.442	0.239	0.164	0.337	0.276	-
Inflammation	0.109	0.316	0.316	0.835	0.056	-
Lymphoid follicles	0.670	0.655	0.823	0.453	0.428	-
Metaplasia	0.923	0.914	0.294	1.00	0.845	-
H. pylori density	0.665	0.802	0.480	0.196	0.664	-

Continued

			Fundic mucosa			
Activity	0.536	0.348	0.558	0.542	0.461	-
Atrophy	0.151	0.259	0.570	0.549	0.337	-
Inflammation	0.082	0.096	0.672	0.838	0.237	-
Lymphoid follicles	0.230	0.185	0.970	0.932	0.751	-
Metaplasia	0.840	0.468	0.646	0.224	0.093	-
H. pylori density	0.264	0.465	0.906	0.581	0.471	-

Table 10. Associations between Sydney System parameters and clinical signs. The values shown represent only the p-values from the statistical tests performed (χ^2 test, df = 2).

Clinical signs	Sydney-System parameters					
	Atrophy	Activity	Inflammation	Lymphoid Follicles	Metaplasia	*H. pylori* density
A	--	--	--	--	--	--
B	0.111	0.026	0.089	0.498	0.282	0.624
C	0.075	0.136	**0.018**	0.856	0.784	**0.016**
D	0.634	0.895	0.880	0.886	0.907	0.488
E	0.342	0.460	0.141	0.821	0.473	0.219
F	0.401	0.460	0.955	0.680	0.886	0.628
G	0.521	0.460	0.329	0.605	0.823	0.139
H	0.553	0.725	0.865	0.992	0.667	0.757

Keys: A: Epigastric pain; B: Dysphagia and Dyspepsia; C: Vomiting; D: Hematemesis, Haematochezia, and Melena; E: Gastroesophageal Reflux; F: Eructation; G: Heartburn; H: Others.

4. Discussion

This retrospective study conducted at the CNHU-HKM in Cotonou, involving a sample of 600 patients, explored the relationship between clinical data, endoscopic observations, and pathological findings in the context of chronic gastritis. This multidimensional approach reinforces the relevance of cross-diagnosis in a context where field data from sub-Saharan Africa remains limited.

Clinically, epigastric pain emerged as the main symptom, reported by 32.33% of patients, followed by dysphagia and dyspepsia (17.83%) and, to a lesser extent, hematemesis. This symptom profile is consistent with that described in several African and Asian studies. For example, Zuzek and Bamba reported a comparable prevalence of epigastric pain in their series, suggesting a dominant symptomatic phenotype of chronic gastritis across continents [4] [9]. These findings support the idea that, despite cultural and dietary variations, certain digestive symptoms remain constant in the clinical presentation of this condition.

Analysis of endoscopic data revealed that the most frequently observed lesions were diffuse erythema and mucosal petechiae (nearly 60% of cases), followed by erosions and ulcerations. This topographic distribution is consistent with the ob-

servations made by Bamba [9], who also reported a preponderance of these endoscopic features. However, an antral predominance of lesions might be expected due to the colonization dynamics of *Helicobacter pylori* and the particular vulnerability of this region to acid-peptic aggression.

Intestinal metaplasia was found in less than 5% of cases, which remains a relatively low rate compared to Asian or European series. This low frequency could be interpreted in two ways: on the one hand, it could reflect a diagnosis made at an early stage of the pathological continuum, before the onset of deep glandular changes; on the other hand, it could reflect lower cumulative exposure to *Helicobacter pylori* or other environmental factors known to promote progression to metaplasia. This observation deserves further investigation, particularly in longitudinal studies integrating infectious status and dietary habits.

Cross-sectional analysis of clinical, endoscopic, and histopathological data in our cohort highlighted a convergence between the severity of certain symptoms and the importance of histological lesions. Moderate to severe chronic inflammation was observed predominantly in patients with suggestive clinical manifestations. This mucosal inflammation, detected histologically, was often associated with an increased density of mononuclear cells in the lamina propria and active neutrophilic infiltration, reflecting ongoing inflammatory activity [10] [11].

Our study revealed that inflammation is more often severe when endoscopy reveals an aspect of congestion, oedema, nodule, or concentration of folds at the level of the antral mucosa. In the same dynamic, fundic endoscopic aspects of congestion, oedema, erythema, or petechiae were more often noted in severe inflammation. This concordance between endoscopic lesions and microscopic damage underlines the interest of endoscopy as a diagnostic orientation tool, although it remains limited in the evaluation of histological severity when not accompanied by targeted biopsies. It also confirms the conclusions of previous studies, which reported a relationship between the severity of visible mucosal lesions and the evolution towards more advanced stages of chronic atrophic gastritis [10].

Overall, these data confirm that an integrated and systematic approach, combining clinical history, endoscopy, and histological analysis, remains essential to accurately assess the severity of chronic gastritis. They highlight the complementary diagnostic value of each modality, particularly in resource-limited settings where the use of molecular techniques or advanced imaging remains marginal.

The lack of a statistical link between these aspects underlines the importance of staging the Sydney System parameters in the assessment of cancer risk. Our results corroborate those of Dave and Nieminen, who demonstrated the value of the OLGA (Operative Link for Gastritis Assessment) and OLGIM (Operative Link on Gastric Intestinal Metaplasia Assessment) classification systems for stratifying the progressive risk of gastric lesions [12] [13]. These systems allow grading intestinal atrophy or metaplasia on the basis of their location and extent in the gastric mucosa, conferring a useful prognostic value in the context of gastric cancer prevention. In our context, these tools could offer a standardized method of assessment,

which is currently not widely used in clinical routines due to a lack of specific training and institutional appropriation.

The use of the OLGA and OLGIM classifications is all the more relevant, since several studies have demonstrated an association between advanced stages of these scores and an increased risk of developing gastric adenocarcinoma, particularly of the intestinal type. The absence of such an assessment system in current practices limits the possibility of effectively monitoring high-risk patients and proposing targeted surveillance or eradication strategies.

This study presents several methodological strengths that reinforce its validity. The sample size, consisting of 600 patients, gives the analysis sufficient statistical power to highlight possible correlations between clinical, endoscopic, and histological data. This volume of data is all the more remarkable given that it is part of an African hospital context, where large-scale studies are still relatively rare. The systematic triangulation of these three dimensions constitutes another major asset. This integrative approach allows a more nuanced reading of the diagnosis of chronic gastritis, breaking with the too-often compartmentalized practice that prevails in many departments. It thus contributes to reducing the risk of underdiagnosis or overinterpretation of certain forms of gastritis, particularly those with a frustrating clinical expression or a misleading endoscopic appearance.

The study setting, centered on a tertiary referral hospital in Benin (CNHU-HKM), also provides realistic anchoring, reflecting the conditions of care in a resource-limited environment. The results obtained are therefore particularly useful for the development of clinical recommendations adapted to French-speaking African contexts.

However, this study is not without limitations, which must be acknowledged to accurately interpret the results. The retrospective design is the main methodological constraint, as it can lead to selection bias and variability in the quality of medical documentation. Certain potentially influential clinical or environmental parameters, such as socioeconomic status, medication history, or diet, could not be analysed due to their absence in the records. As limitations, we can also note that the status of *H. pylori* eradication was not clearly indicated in the patient files. This could confound observed correlations.

Furthermore, the lack of longitudinal follow-up prevents us from documenting the evolution of gastric lesions over time and the response to treatments, particularly *H. pylori* eradication. This lack of temporal perspective limits the ability to identify risky pathological trajectories, particularly those leading to precancerous or neoplastic lesions.

Finally, since the study population was drawn exclusively from a tertiary-level hospital, there is a risk of recruitment bias, with the most severe or symptomatic cases being overrepresented compared to the general population. This could limit the ability to generalize these results to all patients with chronic gastritis, particularly those treated in primary care or community healthcare facilities.

In conclusion, our results highlight the value of an integrated diagnostic ap-

proach in the management of chronic gastritis. Endoscopy, although essential, alone cannot predict the severity of histological lesions. Histopathological analysis therefore remains essential, particularly to identify forms at risk of progression. Systematic screening and eradication of *H. pylori* must be strengthened. Finally, multicenter and prospective studies would allow us to better understand the evolutionary trajectories of this pathology and to validate the use of the OLGA and OLGIM classification systems in an African context.

Ethical Considerations

The study was conducted in accordance with medical ethics. Patient data were anonymized and treated confidentially, in accordance with the principles of the Declaration of Helsinki.

Conflicts of Interest

The authors declare that they have no conflicts of interest in relation to this article.

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