

Chronic Gastritis: Evaluation of the OLGA and OLGIM Systems at CNHU-HKM in Cotonou, Benin: A Retrospective Analysis of 600 Cases

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Abstract

Methodology: This retrospective cross-sectional analysis included 600 histologically confirmed cases (April 2023-December 2024), irrespective of *Helicobacter pylori* status. Agreement was assessed with quadratic-weighted Cohen's kappa; an exact McNemar test compared advanced-stage classifications. Eight secondary grade-by-stage comparisons used Pearson's chi-square or Fisher-Freeman-Halton exact tests, as appropriate, with Holm correction. **Results:** Mean age was 47.70 years $\pm$ 17.21 years. OLGA classified 332 patients (55.3%) in stages 3 - 4, versus 7 (1.2%) by OLGIM; 565 (94.2%) were OLGIM stage 0. Severe antral inflammation differed across OLGA stages (1.2% at stage 0 vs 86.0% at stage 4; $P < 0.001$). Activity distributions were associated with OLGA and OLGIM (Holm-adjusted $P < 0.001$ for both), although OLGIM strata were small and non-monotonic. Follicles were absent in 85.3%; the nominal OLGA association did not persist after correction (adjusted $P = 0.062$). Across OLGA stages, *H. pylori*-negative proportions declined from 50.6% to 1.8%, while severe density rose from 3.6% to 29.8%; density was not associated with OLGIM (adjusted $P = 1.000$). Weighted agreement was negligible ($\kappa = 0.0043$; 95% bootstrap CI, -0.007 to 0.015), with 329 versus 4 discordant advanced-stage classifications. **Conclusion:** The systems yielded different histological classifications, consistent with their distinct target lesions and the low reported prevalence of intestinal metaplasia. This does not establish clinical progression or

cancer risk. Reporting both may provide complementary histological information; prospective outcome studies are needed.

Keywords

OLGA, OLGIM, Chronic Gastritis, Sydney System, *Helicobacter pylori*

1. Introduction

Chronic gastritis is defined histologically by an abnormally dense, predominantly mononuclear inflammatory infiltrate in the lamina propria of the gastric mucosa, which may coexist with atrophic or metaplastic lesions. Eosinophils and, more rarely, neutrophils may also be present and reflect lesion activity. Gastric cancer remains one of the leading causes of cancer-related death worldwide, with an estimated 659,853 deaths in 2022 [1]. Advanced OLGA/OLGIM stages have been associated with higher gastric cancer risk [2]. Progression to gastric adenocarcinoma follows a well-established histological sequence, the Correa cascade, which runs from chronic gastritis through atrophy, metaplasia and dysplasia to invasive cancer [3].

Helicobacter pylori infection is the main etiological factor of chronic gastritis and a major driver of this cascade [4]. Not all gastritis, however, whether *H. pylori*-associated or not, progresses to cancer. Risk stratification tools are therefore needed to identify patients who require close endoscopic monitoring.

Historically, the Updated Sydney System has been used to classify gastric lesions, but its descriptive nature and lack of risk stratification limit its clinical utility for targeted screening [5]. In response to these limitations, two staging systems have been developed: OLGA (Operative Link on Gastritis Assessment) and OLGIM (Operative Link on Gastric Intestinal Metaplasia Assessment). OLGA assesses the severity and topographic distribution of gastric atrophy, whereas OLGIM is based on the extent and topography of intestinal metaplasia, which is generally more reproducible histologically [6] [7].

Both systems stratify patients into five stages (0 to 4); stages 3 and 4 are commonly treated as advanced histological categories and have been associated with higher gastric cancer risk [2]. Comparative differences between OLGA, OLGIM and related systems have been described in cohorts, including men with atrophic gastritis [8], raising questions about their interchangeability and complementarity [9] [10]. Hospital-based histopathological data are available from Benin [11], but direct comparisons of these staging systems in this setting remain limited. The present work is a secondary analysis of the same 600-case cohort previously reported for clinical, endoscopic and histological correlations [12]; it focuses on OLGA/OLGIM staging.

The present study therefore aims to compare the OLGA and OLGIM staging systems in patients with chronic gastritis seen at the Hubert Koutoukou Maga National

University Hospital Center in Cotonou, and to describe the Sydney System parameters associated with each stage. The comparison is deliberately limited to differences in histological staging, without inference about clinical progression, to describe their implications for histological reporting in this hospital.

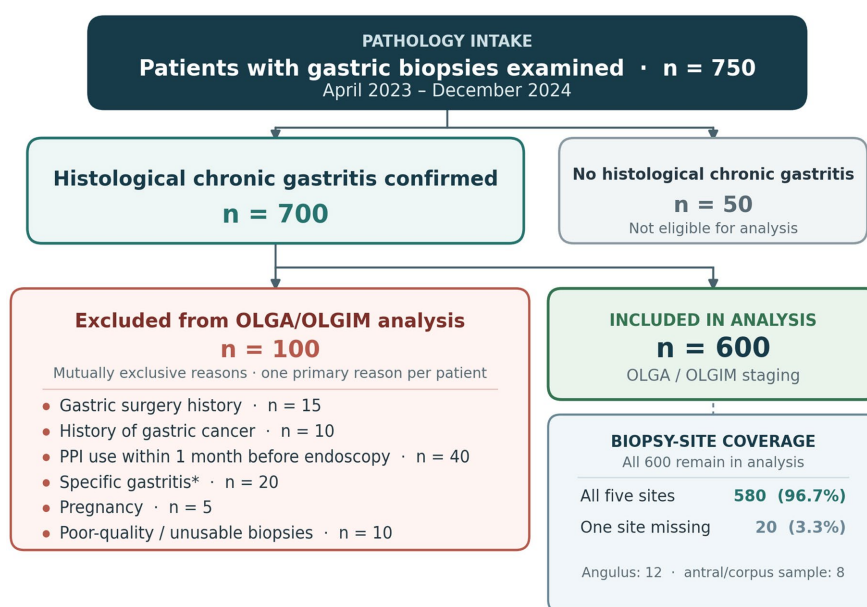
2. Methodology

2.1. Study Design and Setting

We conducted a retrospective, descriptive, cross-sectional study in the Gastroenterology Department and the Pathology Department of the Hubert Koutoukou Maga National University Hospital Center (CNHU-HKM) in Cotonou, Benin, from April 2023 to December 2024. All eligible hospital records for this period were included consecutively; the retrospective, hospital-based design remains susceptible to selection bias. This analysis concerns the same cohort of 600 patients as our previous report of clinical-endoscopic-histological correlations [12], but addresses OLGA and OLGIM staging.

2.2. Study Population and Patient Flow

During the study period, gastric biopsy specimens from 750 patients were submitted to and examined by the Pathology Department. The participant flow in **Figure 1** begins at this pathology intake ($n = 750$); EGDs without gastric biopsy are outside the diagram. Histological examination identified chronic gastritis in 700 patients; 50 without histological chronic gastritis were not eligible for this analysis. The following 100 patients with chronic gastritis were excluded:



PPI, proton-pump inhibitor. *Specific gastritis includes autoimmune, granulomatous, or lymphomatous gastritis.

Pathology intake is the flow entry ($n = 750$); 100 of 850 EGDs had no gastric biopsy and are outside this diagram.

Figure 1. Participant flow from pathology intake to analysis. The 100 EGDs without gastric biopsy are outside this pathology-based flow.

- Patients with a history of gastric surgery (n = 15);
- Patients with a known history of gastric cancer (n = 10);
- Patients treated with proton pump inhibitors (PPIs) within one month prior to endoscopy (n = 40);
- Cases of specific gastritis (autoimmune, granulomatous, or lymphomatous gastritis) (n = 20);
- Pregnant women (n = 5);
- Poor-quality or unusable samples (unusable fragments, lack of representative mucosa) (n = 10).

Each excluded patient was assigned one primary reason; the six exclusion categories are mutually exclusive and sum to 100.

In all, 600 patients with histologically confirmed chronic gastritis were analyzed (**Figure 1**). Consent for endoscopy and gastric biopsy had been obtained in the parent study; the ethics committee waived consent for this secondary analysis of anonymized records (see Section 2.7).

Chronic gastritis was defined histologically according to the Updated Sydney System as a chronic inflammatory infiltrate in the lamina propria, irrespective of clinical symptoms; upper gastrointestinal symptoms were the indication for endoscopy, not a diagnostic criterion. *Helicobacter pylori* status was assessed histologically on modified Giemsa staining; a case was considered positive when curved bacilli were seen on at least one biopsy. Density was graded from 0 (absent) to 3 (severe) on the Sydney visual analogue scale.

2.3. Biopsy Protocol and Handling of Missing Sites

For each included patient, biopsies were obtained from both the antral and corpus mucosa, as recommended by the Updated Sydney System:

- Two biopsies from the gastric antrum (greater and lesser curvatures);
- Two biopsies from the gastric body (greater and lesser curvatures);
- One biopsy from the angulus.

All five biopsy sites were available in 580 cases (96.7%). One site was missing in 20 cases (3.3%): the angulus in 12 and one antral or corpus sample in 8. No case lacked an entire compartment. The angulus was assigned to the antral compartment. For atrophy and intestinal metaplasia, compartment grades were calculated as the mean of available biopsies; the corresponding OLGA/OLGIM stage was then assigned using the standard staging matrix. Excluding these 20 cases yielded 54.8% advanced OLGA stages, compared with 55.3% in the full cohort.

2.4. Histological Processing and Assessment

All biopsies were immediately fixed in 10% buffered formalin. After dehydration, paraffin embedding and sectioning, 3 - 5 µm sections were prepared and stained with the following techniques:

- Hematoxylin-eosin (HE) for general morphological analysis;
- Modified Giemsa staining for the detection of *Helicobacter pylori*.

Histological grading followed the Updated Sydney System. All biopsy samples from the 600 included cases were evaluated independently by two pathologists. Discordant assessments were reviewed jointly and resolved by consensus; the reconciled assessments were used in the study analyses. The presence and intensity (0 = absent, 1 = mild, 2 = moderate, 3 = severe) of the following features were graded:

- Chronic inflammation;
- Activity (neutrophilic infiltrate);
- Glandular atrophy;
- Intestinal metaplasia (graded by severity; the OLGIM staging system does not distinguish complete from incomplete subtypes);
- Lymphoid follicles;
- *H. pylori* density.

For associations with stages, the case-level scores recorded in the study dataset following independent review and consensus resolution of discordant readings were cross-tabulated separately with OLGA and OLGIM. The antral grade was used for chronic inflammation; activity, lymphoid follicles and *H. pylori* density were analyzed using their recorded patient-level scores.

Each case was then staged using two systems:

OLGA (Operative Link on Gastritis Assessment): based on the severity and location of gastric atrophy, with a stage ranging from 0 to 4 [7];

OLGIM (Operative Link on Gastric Intestinal Metaplasia Assessment): based solely on the extent and severity of intestinal metaplasia, also assigned a stage from 0 to 4 [6].

Stages were then grouped into two categories: lower histological stages (0 - 2) and advanced histological stages (3 - 4). These categories do not represent observed cancer outcomes in this cohort.

2.5. Variables Collected

The following variables were collected:

- Sociodemographic characteristics: age, sex;
- Presence or absence of *Helicobacter pylori* and its density;
- Histological characteristics (chronic inflammation, activity, atrophy, intestinal metaplasia, lymphoid follicles);
- OLGA and OLGIM stages.

2.6. Statistical Analysis

Data were entered in Microsoft Excel 2013 and analyzed with R software, version 4.5.0 (R Foundation for Statistical Computing, Vienna, Austria). Descriptive statistics used means $\pm$ standard deviations for quantitative variables and frequencies and percentages for qualitative variables. Associations between stage and each of the four Sydney-parameter grades (activity, antral chronic inflammation, lymphoid follicles and *H. pylori* density) were tested separately for OLGA and OLGIM, giv-

ing eight secondary comparisons. Pearson’s chi-square test was used when expected counts met conventional assumptions; sparse tables were analyzed with the Fisher-Freeman-Halton exact test using Monte Carlo estimation (200,000 replicates). Holm’s method was applied to the eight secondary P values. A separate exploratory comparison of absent versus present activity across OLGIM stages used the same exact test and was reported outside the eight-test Holm family. These omnibus tests assess distributional association, not a monotonic trend. Agreement between the five-level OLGA and OLGIM classifications was assessed with quadratic-weighted Cohen’s kappa; its 95% confidence interval was estimated by a percentile bootstrap with 200,000 resamples. For the paired stages 0 - 2 versus 3 - 4 comparison, discordant pairs were reported and an exact McNemar test was used. The significance level was 5%.

2.7. Ethical Considerations

The study was approved by the Ethics Committee of the Faculty of Health Sciences of the University of Abomey-Calavi and by the Director of the CNHU-HKM. Owing to the retrospective design and the use of anonymized data, informed consent was waived for this secondary analysis; consent for endoscopy and biopsy had been obtained in the parent study. The study complied with the principles of the Declaration of Helsinki.

3. Results

3.1. Patient Flow and Epidemiological Data

Patient flow is summarized in **Figure 1**. During the study period, gastric biopsy specimens from 750 patients were examined by pathology. Histology showed chronic gastritis in 700; 50 patients without histological chronic gastritis were not included. Of the 700, 100 met one or more exclusion criteria, and 600 were analyzed. The epidemiological data and the OLGA and OLGIM stages of the 600 cases are presented in **Table 1**. Patients ranged in age from 10 to 95 years (mean 47.70 years $\pm$ 17.21 years), with a male-to-female ratio of 0.91 (286 men, 314 women). Sex distribution did not differ significantly across OLGA stages (chi-square test, $P = 0.57$).

Table 1. Distribution of epidemiological data by OLGA and OLGIM stage (n = 600).

OLGA Stages	Stage 0	Stage 1	Stage 2	Stage 3	Stage 4	Total
Number of Cases (%)	83 (13.8)	43 (7.2)	142 (23.7)	275 (45.8)	57 (9.5)	600 (100)
Mean Age (years)	41.75 $\pm$ 16.63	50.37 $\pm$ 16.53	47.75 $\pm$ 18.31	49.01 $\pm$ 16.90	47.80 $\pm$ 15.83	47.70 $\pm$ 17.21
Sex (M/F)	38/45	25/18	70/72	129/146	24/33	286/314
OLGIM Stages	Stage 0	Stage 1	Stage 2	Stage 3	Stage 4	Total
Number of Cases (%)	565 (94.2)	21 (3.5)	7 (1.2)	7 (1.2)	0 (0)	600 (100)
Mean Age (years)	47.30 $\pm$ 17.11	53.19 $\pm$ 15.16	50.57 $\pm$ 20.23	62.50 $\pm$ 24.00	-	47.70 $\pm$ 17.21
Sex (M/F)	275/290	6/15	3/4	2/5	-	286/314

M: Male; F: Female.

3.2. OLGA and OLGIM Staging

Under the OLGA classification, lower histological stages (0 - 2) accounted for 268 cases (44.7%) and advanced stages (3 - 4) for 332 cases (55.3%). Under OLGIM, lower stages accounted for 593 cases (98.8%) and advanced stages for only 7 cases (1.2%). The most frequent OLGA stage was stage 3 (275 cases; 45.8%), whereas 565 cases (94.2%) were OLGIM stage 0 and no case was OLGIM stage 4 (**Table 1**).

3.3. Activity

Most cases (522 patients; 87.0%) had mild activity, and severe activity was rare (2 cases; 0.3%). Absence of activity was most frequent at OLGA stage 0 (42/83; 50.6%). Under OLGIM, the highest proportion with absent activity was at stage 2 (2/7; 28.6%), compared with 58/565 (10.3%) at stage 0. The four-category activity distribution differed across OLGA and OLGIM stages (nominal Fisher-Freeman-Halton Monte Carlo $P < 0.001$ for both; Holm-adjusted $P < 0.001$ for both). The OLGIM comparison is based on small stage 1 - 3 groups and should not be interpreted as a monotonic trend (**Table 2**).

Table 2. Distribution of histological grades by OLGA and OLGIM stage (n = 600). (a) OLGA, (b) OLGIM.

(a)							
Parameter	Grade	Stage 0	Stage 1	Stage 2	Stage 3	Stage 4	Total
Activity	Absent	42 (50.6)	8 (18.6)	8 (5.6)	2 (0.7)	2 (3.5)	62 (10.3)
Activity	Mild	36 (43.4)	35 (81.4)	128 (90.1)	269 (97.8)	54 (94.7)	522 (87.0)
Activity	Moderate	5 (6.0)	0 (0.0)	5 (3.5)	3 (1.1)	1 (1.8)	14 (2.3)
Activity	Severe	0 (0.0)	0 (0.0)	1 (0.7)	1 (0.4)	0 (0.0)	2 (0.3)
Chronic inflammation	Absent	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)
Chronic inflammation	Mild	45 (54.2)	13 (30.2)	5 (3.5)	5 (1.8)	0 (0.0)	68 (11.3)
Chronic inflammation	Moderate	37 (44.6)	27 (62.8)	70 (49.3)	116 (42.2)	8 (14.0)	258 (43.0)
Chronic inflammation	Severe	1 (1.2)	3 (7.0)	67 (47.2)	154 (56.0)	49 (86.0)	274 (45.7)
Lymphoid follicles	Absent	76 (91.6)	42 (97.7)	117 (82.4)	233 (84.7)	44 (77.2)	512 (85.3)
Lymphoid follicles	Mild	7 (8.4)	1 (2.3)	23 (16.2)	42 (15.3)	12 (21.1)	85 (14.2)
Lymphoid follicles	Moderate	0 (0.0)	0 (0.0)	1 (0.7)	0 (0.0)	1 (1.8)	2 (0.3)
Lymphoid follicles	Severe	0 (0.0)	0 (0.0)	1 (0.7)	0 (0.0)	0 (0.0)	1 (0.2)
<i>H. pylori</i> density	Absent	42 (50.6)	9 (20.9)	9 (6.3)	8 (2.9)	1 (1.8)	69 (11.5)
<i>H. pylori</i> density	Mild	17 (20.5)	20 (46.5)	74 (52.1)	132 (48.0)	20 (35.1)	263 (43.8)
<i>H. pylori</i> density	Moderate	21 (25.3)	6 (14.0)	32 (22.5)	69 (25.1)	19 (33.3)	147 (24.5)
<i>H. pylori</i> density	Severe	3 (3.6)	8 (18.6)	27 (19.0)	66 (24.0)	17 (29.8)	121 (20.2)
(b)							
Parameter	Grade	Stage 0	Stage 1	Stage 2	Stage 3	Stage 4	Total
Activity	Absent	58 (10.3)	2 (9.5)	2 (28.6)	0 (0.0)	-	62 (10.3)
Activity	Mild	494 (87.4)	19 (90.5)	3 (42.9)	6 (85.7)	-	522 (87.0)

Continued

Activity	Moderate	12 (2.1)	0 (0.0)	2 (28.6)	0 (0.0)	-	14 (2.3)
Activity	Severe	1 (0.2)	0 (0.0)	0 (0.0)	1 (14.3)	-	2 (0.3)
Chronic inflammation	Absent	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	-	0 (0.0)
Chronic inflammation	Mild	66 (11.7)	2 (9.5)	0 (0.0)	0 (0.0)	-	68 (11.3)
Chronic inflammation	Moderate	244 (43.2)	8 (38.1)	4 (57.1)	2 (28.6)	-	258 (43.0)
Chronic inflammation	Severe	255 (45.1)	11 (52.4)	3 (42.9)	5 (71.4)	-	274 (45.7)
Lymphoid follicles	Absent	483 (85.5)	18 (85.7)	6 (85.7)	5 (71.4)	-	512 (85.3)
Lymphoid follicles	Mild	79 (14.0)	3 (14.3)	1 (14.3)	2 (28.6)	-	85 (14.2)
Lymphoid follicles	Moderate	2 (0.4)	0 (0.0)	0 (0.0)	0 (0.0)	-	2 (0.3)
Lymphoid follicles	Severe	1 (0.2)	0 (0.0)	0 (0.0)	0 (0.0)	-	1 (0.2)
<i>H. pylori</i> density	Absent	67 (11.9)	2 (9.5)	0 (0.0)	0 (0.0)	-	69 (11.5)
<i>H. pylori</i> density	Mild	246 (43.5)	10 (47.6)	3 (42.9)	4 (57.1)	-	263 (43.8)
<i>H. pylori</i> density	Moderate	137 (24.2)	5 (23.8)	3 (42.9)	2 (28.6)	-	147 (24.5)
<i>H. pylori</i> density	Severe	115 (20.4)	4 (19.0)	1 (14.3)	1 (14.3)	-	121 (20.2)

Values are counts (column percentages within each stage); percentages in the Total column use n = 600. Panels A and B show parallel distributions for the same patients.

3.4. Chronic Inflammation

Antral chronic inflammation was present in all 600 cases, as in the previously reported cohort [12]: it was mild in 68 cases (11.3%), moderate in 258 (43.0%) and severe in 274 (45.7%). The distribution differed across OLGA stages; severe inflammation was observed in 1/83 cases (1.2%) at stage 0 and 49/57 (86.0%) at stage 4 (Pearson chi-square test, $P < 0.001$; Holm-adjusted $P < 0.001$). No association was detected across OLGIM stages (Fisher-Freeman-Halton Monte Carlo $P = 0.879$; Holm-adjusted $P = 1.000$; **Table 2**).

3.5. Lymphoid Follicles

Of the 600 patients, 85.3% (512 cases) showed no lymphoid follicles. The four-category follicle-grade distribution had a nominal association with OLGA stage (Fisher-Freeman-Halton Monte Carlo $P = 0.016$), but this did not remain statistically significant after Holm correction across the eight secondary tests (adjusted $P = 0.062$). No association was detected with OLGIM stage ($P = 0.683$; adjusted $P = 1.000$; **Table 2**). Given the sparse counts, these results are exploratory.

3.6. *H. pylori* Density and Classification Stage

H. pylori density varied widely: 69 cases (11.5%) were negative, 263 (43.8%) had mild density, 147 (24.5%) had moderate density, and 121 (20.2%) had severe density. Across OLGA stages, the observed proportion of *H. pylori*-negative cases declined from 50.6% (42/83) at stage 0 to 1.8% (1/57) at stage 4; severe density ranged

from 3.6% (3/83) at stage 0 to 29.8% (17/57) at stage 4. The density distribution differed across OLGA stages (Pearson chi-square test, $P < 0.001$; Holm-adjusted $P < 0.001$). No association was detected across OLGIM stages (Fisher-Freeman-Halton Monte Carlo $P = 0.994$; Holm-adjusted $P = 1.000$). These are cross-sectional associations and do not establish temporal change or causality (Table 2).

3.7. Agreement between the OLGA and OLGIM Systems

Under the OLGA classification, 268 patients (44.7%) were classified in stages 0 - 2, compared with 593 (98.8%) under OLGIM. As shown in Table 3, many patients at OLGIM stage 0 were classified at more advanced histological stages by OLGA: among the 565 patients at OLGIM stage 0, 132 were OLGA stage 2, 260 OLGA stage 3 and 52 OLGA stage 4. Patients at OLGIM stages 1 and 2 were often found at more advanced OLGA stages (3 and 4), while those at OLGIM stage 3 showed more variable OLGA stages. Agreement between the two classifications was negligible (quadratic-weighted Cohen’s kappa = 0.0043; 95% percentile-bootstrap CI, -0.007 to 0.015). For stages 0 - 2 versus 3 - 4, 329 patients were classified as advanced by OLGA but lower-stage by OLGIM, whereas 4 were classified as advanced by OLGIM but lower-stage by OLGA. The binary Cohen’s kappa was -0.0053 (observed agreement 267/600; 44.5%); the exact McNemar test showed a difference in marginal classifications ($P < 0.001$). Figure 2 illustrates one reported histological example of discordance.

Table 3. Cross-tabulation of OLGA and OLGIM stages (n = 600).

OLGA Stages	OLGIM Stages					Total
	OLGIM 0	OLGIM 1	OLGIM 2	OLGIM 3	OLGIM 4	
OLGA 0	79	3	1	0	0	83
OLGA 1	42	0	0	1	0	43
OLGA 2	132	5	2	3	0	142
OLGA 3	260	10	3	2	0	275
OLGA 4	52	3	1	1	0	57
Total	565	21	7	7	0	600

Lower stage: 0 - 2; advanced stage: 3 - 4. Quadratic-weighted Cohen’s kappa = 0.0043 (95% percentile-bootstrap CI, -0.007 to 0.015). For the dichotomy 0 - 2 versus 3 - 4, binary Cohen’s kappa = -0.0053; discordant pairs were 329 (OLGA advanced/OLGIM lower) versus 4 (OLGA lower/OLGIM advanced), exact McNemar $P < 0.001$.

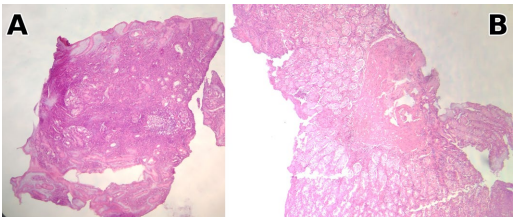


Figure 2. Illustration of a discrepancy between the OLGA and OLGIM classifications (hematoxylin-eosin).

In the example shown in **Figure 2**, antral atrophy (A) was scored 3 and corpus (oxyntic/fundic) atrophy (B) was scored 1, corresponding to OLGA stage 3. Intestinal metaplasia was not identified in either compartment, yielding OLGIM stage 0.

3.8. Atrophy and Intestinal Metaplasia by Compartment

Compartment-level atrophy and intestinal-metaplasia grades are summarized in **Table 4**. Atrophy was moderate or severe in 468 of 600 scores from the antral compartment (78.0%) and 313 of 600 scores from the corpus (fundic) compartment (52.2%). Intestinal metaplasia was absent in 571 of 600 scores from the antral compartment (95.2%) and 591 of 600 scores from the corpus (fundic) compartment (98.5%). These are compartment distributions; counts across compartments must not be added to estimate unique patients.

Table 4. Distribution of atrophy and intestinal metaplasia grades by compartment (n = 600 per compartment).

Histological Feature	Grade	Antral Compartment, n (%)	Corpus (Fundic) Compartment, n (%)
Atrophy	Absent	83 (13.8)	134 (22.3)
Atrophy	Mild	49 (8.2)	153 (25.5)
Atrophy	Moderate	389 (64.8)	307 (51.2)
Atrophy	Severe	79 (13.2)	6 (1.0)
Intestinal metaplasia	Absent	571 (95.2)	591 (98.5)
Intestinal metaplasia	Mild	17 (2.8)	7 (1.2)
Intestinal metaplasia	Moderate	5 (0.8)	2 (0.3)
Intestinal metaplasia	Severe	7 (1.2)	0 (0.0)

Percentages are within each compartment. The same patient may contribute a grade to both compartments; therefore, the columns are not mutually exclusive patient counts.

4. Discussion

In this study of 600 patients whose gastric biopsies were examined at the CNHU-HKM in Cotonou, the OLGA and OLGIM systems produced markedly different histological classifications. OLGA classified 332 patients (55.3%) in advanced stages, compared with 7 (1.2%) under OLGIM; 329 patients were advanced by OLGA only and 4 by OLGIM only. The quadratic-weighted kappa was 0.0043 (95% bootstrap CI, -0.007 to 0.015), indicating negligible agreement. These findings describe classification differences, not observed differences in cancer progression.

Advanced OLGIM stages occurred in 7 patients (1.2%) in our cohort, compared with none in Mbengue *et al.* and 7% in Ben Slama *et al.* [13] [14]. The advanced-OLGA proportion (55.3%) was higher than the 3% and 6% reported in those studies, respectively; these are descriptive comparisons across differently selected pop-

ulations and cannot establish why the estimates differ. The systems assess different lesions: OLGA stages glandular atrophy, with or without metaplastic change, whereas OLGIM stages intestinal metaplasia only [6] [7]. Ben Slama *et al.* reported 6% advanced OLGA and 7% advanced OLGIM; Mansour-Ghanaei *et al.* reported 4.3% and 4.0%, respectively [9] [14]. Thus, these studies do not support a general claim that OLGIM always classifies fewer patients as advanced. Atrophy without intestinal metaplasia can account for an OLGA-OLGIM discrepancy. In our cohort, moderate or severe atrophy was recorded in 78.0% of antral and 52.2% of corpus (fundic) compartment scores (Table 4); these compartment summaries provide context but do not independently validate each patient-level stage assignment or establish clinical risk. The high proportion of advanced OLGA stages warrants cautious interpretation. Sampling variability and the challenge of distinguishing true gland loss from inflammation-rich mucosa could affect grading; the available data cannot determine whether either contributed. Independent validation of atrophy scoring would be valuable.

The antral chronic-inflammation grade distribution differed across OLGA stages, with severe inflammation observed in 1.2% at stage 0 and 86.0% at stage 4. This cross-sectional association does not establish a role in progression to gastric cancer. The four-category activity distribution was associated with both OLGA and OLGIM stage (nominal $P < 0.001$ for both; Holm-adjusted $P < 0.001$ for both). For OLGIM, however, the category proportions were non-monotonic, and the stage 1 - 3 groups were small. A separate exploratory comparison of absent versus present activity across OLGIM stages was not significant (Fisher-Freeman-Halton Monte Carlo $P = 0.394$); it was reported separately and was not included in the eight-test Holm correction. The remaining OLGIM comparisons (antral inflammation, follicle grade and *H. pylori* density) were not significant after Holm adjustment. These secondary results should not be described as evidence of an ordered trend.

Lymphoid follicles were infrequent in our samples. Their grade distribution showed a nominal association with OLGA stage ($P = 0.016$) that did not remain significant after Holm adjustment ($P = 0.062$); no association was detected with OLGIM stage. This exploratory cross-sectional analysis does not establish whether follicles predict disease progression or reflect a particular mechanism.

With regard to *H. pylori* density, negative cases were more frequent at OLGA stage 0 than stage 4 (50.6% versus 1.8%), while severe density was observed in 3.6% and 29.8%, respectively. The omnibus distribution differed across OLGA stages ($P < 0.001$), but this cross-sectional pattern cannot be interpreted as a temporal increase in bacterial burden or as evidence of progression. It may be affected by biopsy sampling, the patient-level aggregation rule for density, selection and scoring; these possibilities cannot be separated with the supplied summaries. The IARC citation supports the etiological role of *H. pylori*, not a specific density trajectory across atrophy stages.

Atrophy without intestinal metaplasia can yield an advanced OLGA stage while

the corresponding OLGIM stage remains 0, because OLGIM uses intestinal metaplasia alone whereas OLGA considers a broader spectrum of gland loss and atrophic change [6] [7] [15]. Intestinal metaplasia was reported in 35 patients (5.8%), consistent with the low number of advanced OLGIM stages. Meta-analyses have associated advanced OLGA and OLGIM stages with increased gastric cancer risk [2] [16]; however, this study included no dysplasia, gastric cancer, or surveillance outcomes and cannot assess that risk.

A direct comparison of OLGA and OLGIM shows that the systems are not interchangeable as histological staging methods in this cohort. OLGA identified patients classified as stage 0 by OLGIM, but this difference does not establish which system better predicts outcomes in this population. The clinical consequences cannot be determined from these data.

These results support reporting both stages as complementary histological descriptors, because OLGIM alone does not display non-metaplastic atrophy. They do not establish that combined reporting improves patient outcomes or should change endoscopic surveillance. Prospective outcome data are required.

Several limitations remain. The study is retrospective and cross-sectional, with no longitudinal follow-up or data on dysplasia, gastric cancer or surveillance outcomes; clinical progression cannot be inferred. The single-center design may limit generalizability. The cohort included patients aged 10 - 95 years, but age-specific outcomes in participants younger than 18 were not analyzed. Twenty cases (3.3%) lacked one recommended biopsy site. In a sensitivity analysis restricted to the 580 cases with all five sites, 318 (54.8%) were classified as advanced by OLGA, compared with 332/600 (55.3%) in the full cohort. Secondary associations involving activity, lymphoid follicles and *H. pylori* density were based on the patient-level scores recorded in the study dataset and should be interpreted cautiously. The present study is a secondary analysis of the same 600-case cohort previously reported in [12]. It focuses on OLGA/OLGIM staging and does not represent an independent cohort.

Nationwide multicenter studies with longitudinal follow-up are needed to validate these findings on a larger scale. Including additional histological or serological markers, such as pepsinogens, and recording dysplasia and cancer endpoints could improve risk stratification and refine surveillance programs.

5. Conclusion

In this cross-sectional study of 600 cases of chronic gastritis at the CNHU-HKM in Cotonou, OLGA and OLGIM showed negligible agreement in histological staging (quadratic-weighted $\kappa = 0.0043$; 95% bootstrap CI, -0.007 to 0.015). OLGA classified 55.3% of patients in advanced histological stages versus 1.2% for OLGIM. This disparity is compatible with the systems' different target lesions and the low reported prevalence of intestinal metaplasia, but it does not establish a difference in clinical progression or cancer risk. Reporting both stages may provide complementary histological information. Prospective studies with

clinical outcomes are needed before implications for surveillance or prevention are inferred.

Author Contributions

Conceptualization: F.S.; Methodology: F.S. and A.H.; Software: A.H.; Validation: F.S., L.V.C.B., and M.T.A.-A.; Formal analysis: A.H.; Investigation (Endoscopy and Gastric Biopsies): M.S.; Investigation (Histological Assessment): F.S., D.R.H.G., F.N.D.S., and M.A.; Resources: M.S. and M.T.A.-A.; Data Curation: A.G.H.-Y., D.-G.H., and J.A.; Writing—Original Draft Preparation: F.S. (Introduction and Methodology), A.H. (Results), and D.R.H.G. (Discussion); Writing—Review and Editing: F.S., D.R.H.G., A.H., L.V.C.B., and M.T.A.-A.; Visualization: A.H. and A.G.H.-Y.; Supervision: F.S. and M.T.A.-A.; Project Administration: F.S.

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Conflicts of Interest

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

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