

The OLGIMA system for gastric cancer risk assessment. A useful method based on the histological Sydney consensus

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The OLGIMA system for gastric cancer risk assessment. A useful method based on the histological Sydney consensus

Introduction: The risk stratification of gastric cancer (GC) is graded by assessing well-established precursor lesions, glandular atrophy (GA), and intestinal metaplasia (IM), resulting in both the operative link on gastritis assessment (OLGA), and intestinal metaplasia (OLGIM) systems. Although the OLGIM stage is reproducible among pathologists, the OLGA system is laborious to calculate and has poor reproducibility. In addition, it does not comprehensively address the severity of both GA and IM as recommended by the Sydney consensus. We aimed to propose the Operative Link on Gastric Intestinal Metaplasia and Glandular Atrophy Assessment (OLGIMA) system, which identifies OLGIM III–IV and upstages 0–II with advanced GA.

Methods: A cross-sectional study of consecutive diagnostic gastroscopies in adults was designed. Systematic gastric biopsies were taken. The updated Sydney guidelines were used for histological grading of GA

and IM. Higher GC risk was defined as OLGIM III–IV and advanced GA.

Results: The OLGIMA stage was assessed based on the most severe GA and/or IM findings in both antrum and corpus. We included 998 patients (median age 57; 64% women; 35% *Helicobacter pylori* infection). Thirty-nine (3.9%) patients had higher GC risk: 17 (1.7%) with OLGIM III–IV; 12 (1.2%) with advanced GA, and 10 (1%) meeting both criteria. Among OLGIM 0–II, 12 (1.2%) patients had advanced GA. The OLGIMA system upstaged 39 (3.9%) patients to III–IV, being more sensitive than OLGIM.

Conclusions: The new OLGIMA system identifies patients at higher GC risk (OLGIMA III–IV), encompassing all OLGIM III–IV patients, and upstaging those OLGIM 0–II with advanced GA. This approach addresses the OLGA and OLGIM limitations by integrating GA and IM severity as recommended by the Updated Sydney consensus.

Keywords: atrophy, chronic gastritis, gastric carcinoma, intestinal metaplasia, OLGA, OLGIM, OLGIMA

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Abbreviations: CAG, chronic atrophic gastritis; GA, glandular atrophy; GC, gastric cancer; Hp, *Helicobacter pylori*; IM, intestinal metaplasia; NPV, negative predictive value; OLGA, operative link on gastritis assessment; OLGIM, Operative Link on Gastric intestinal metaplasia; OLGIMA, operative link on gastric intestinal metaplasia and glandular atrophy assessment; PPV, positive predictive value; UGI, upper gastrointestinal.

Introduction

Gastric cancer (GC) remains a major global health challenge. Despite advances in diagnosis and therapy, the prognosis remains poor in advanced stages, underscoring the importance of identifying and monitoring patients at increased risk of GC. Currently, there is no controversy regarding the identification and clinical relevance of gastric dysplasia.¹ However, this is not the case for intestinal metaplasia (IM) or glandular atrophy (GA). There is substantial variability among clinical guidelines regarding the histological criteria used to define high-risk patients.^{2,3}

The Sydney consensus, first published in 1990 and updated in 1996, represented a significant milestone in the histological standardization of IM and GA.^{4,5} These two parameters require individual reporting and scoring using a visual analogue scale (0 = absent; 1 = mild; 2 = moderate; 3 = severe). Nevertheless, this standardization applies to the assessment of individual biopsies and does not directly provide clinically actionable information for patients' risk stratification.

The first attempt to stratify patients was proposed by the OLGA (Operative Link on Gastritis Assessment) system in 2007, which classifies patients into five stages (0, I, II, III, and IV). Thus, patients classified as OLGA stage III or IV are considered to be at higher risk for GC.^{6,7} The OLGA system requires a combined assessment in both distal and proximal compartments of GA, with or without IM, the latter classified as complete or incomplete, including pseudopyloric metaplasia. Unlike the Sydney consensus, the OLGA system ultimately reports an average severity of chronic atrophic gastritis (CAG). Although it is accurate at stratifying patients at risk of GC, it is laborious to use on a daily basis, and the stage must be provided by the pathologist.^{8,9} Despite being the first attempt to stratify GC risk, the OLGA system is not commonly used in routine practice and is rather mostly focused on research settings.² This is due to the known impact of low histologic reproducibility of GA within a model that requires the integrated evaluation of both GA and IM, which ultimately results in a complex process with limited reliability. In fact, there is only moderate concordance in the assessment of GA among pathologists, which also depends on biopsy orientation, being more reproducible in the body than in the antrum/incisura.

The second major attempt to stratify patients was the introduction of the OLGIM system (Operative Link on Gastric Intestinal Metaplasia assessment) in 2010.

In this system, patients classified as stage III or IV are considered to be at higher risk for GC.¹⁰ Unlike the OLGA system, the OLGIM proposal focuses solely on the severity of IM, following the visual analogue scale recommended by the Updated Sydney consensus. By excluding GA – which has lower histological reproducibility – and eliminating the need to average different conditions, this system improves interobserver agreement by concentrating exclusively on IM.¹¹ Although the OLGIM system is simpler than the OLGA system, some patients classified as OLGIM stage 0–II may be missed as high risk for GC due to the exclusive focus on IM, leaving aside the severity of GA and pseudopyloric metaplasia in the oxyntic mucosa.¹²

Both the OLGA and OLGIM systems overlook the severity of GA as recommended by the Sydney consensus. Nevertheless, some guidelines, including the American, Italian, and MAPS I–II–III recommendations, advocate for considering patients with advanced GA – defined as moderate to marked severity involving both the antrum and corpus – as high-risk individuals.^{1,13–15}

This study proposes OLGIMA (Operative Link on Gastric Intestinal Metaplasia and Glandular Atrophy assessment), as a novel and feasible histological GC risk stratification system, based not only on IM severity, but also on that of GA. Therefore, we aimed at demonstrating the no-inferiority of the OLGIMA system compared with OLGIM in identifying patients at high risk for GC.

Materials and Methods

STUDY DESIGN AND PATIENTS

This observational, cross-sectional, and multicentre research was conducted across 28 Spanish hospitals from April 2021 to July 2023. Data for upper gastrointestinal (UGI) endoscopies were collected from the EpiGASTRIC/EDGAR registry (NCT05551416; first posted: September 22, 2022). This registry prospectively recorded consecutive diagnostic UGI endoscopies performed on patients aged 18 and older for any indication. Patients under surveillance for other conditions or those with a history of previous gastric surgery were excluded. At each UGI endoscopy, gastric biopsies were taken according to the Updated Sydney consensus. Patients diagnosed with dysplasia, adenocarcinoma, or any other malignancy were excluded from the study, as were those with suspected autoimmune chronic atrophic gastritis, or lacking histological data. Informed consent for the

procedure and study participation was obtained from all patients, and the study protocol received approval from the Ethics Committee of each participating hospital (Ref. HCB/2021/0482, in April 2021).

PROCEDURES

Gastric biopsies were collected in accordance with the Updated Sydney consensus and submitted in two separate vials for histological assessment. One vial contained antral lesser and greater curvature biopsies, and an additional biopsy from the middle part of the incisura. Another vial included biopsies from the lesser and greater curvature of the middle part of the corpus. The histological severity for both GA and IM was recorded based on the Sydney recommendation and graded into four categories: 0 = absent; 1 = mild; 2 = moderate; 3 = marked separately for both the antrum (including incisura) and corpus (Figure 1). The rapid urease test, and immunohistochemistry for *Helicobacter pylori* (Hp) infection were performed in accordance with the routine practices of the respective hospitals.

PATIENTS AT HIGHER RISK OF GC

The high-risk criteria were established according to the severity and distribution of both IM and GA based on the Sydney consensus. Regarding IM, high-risk patients were those with OLGIM Stages III and IV. Regarding GA, high-risk patients were those with advanced GA, defined as the presence of extensive GA involving both the antrum and corpus, with a severity ranging from moderate to marked in both topographical areas. The total number of patients at higher risk for GC (patients with OLGIM stage III–IV and patients with advanced GA) was used as the *gold standard*. As this study was based on the Sydney recommendations of histological report, we did not include information about the compartmental average scores, and the OLGA stage was not assessed.

OLGIMA STAGING

The OLGIMA staging or severity is based on the maximum severity score (0–3) of either GA or IM, across all biopsy sites (antrum including incisura and

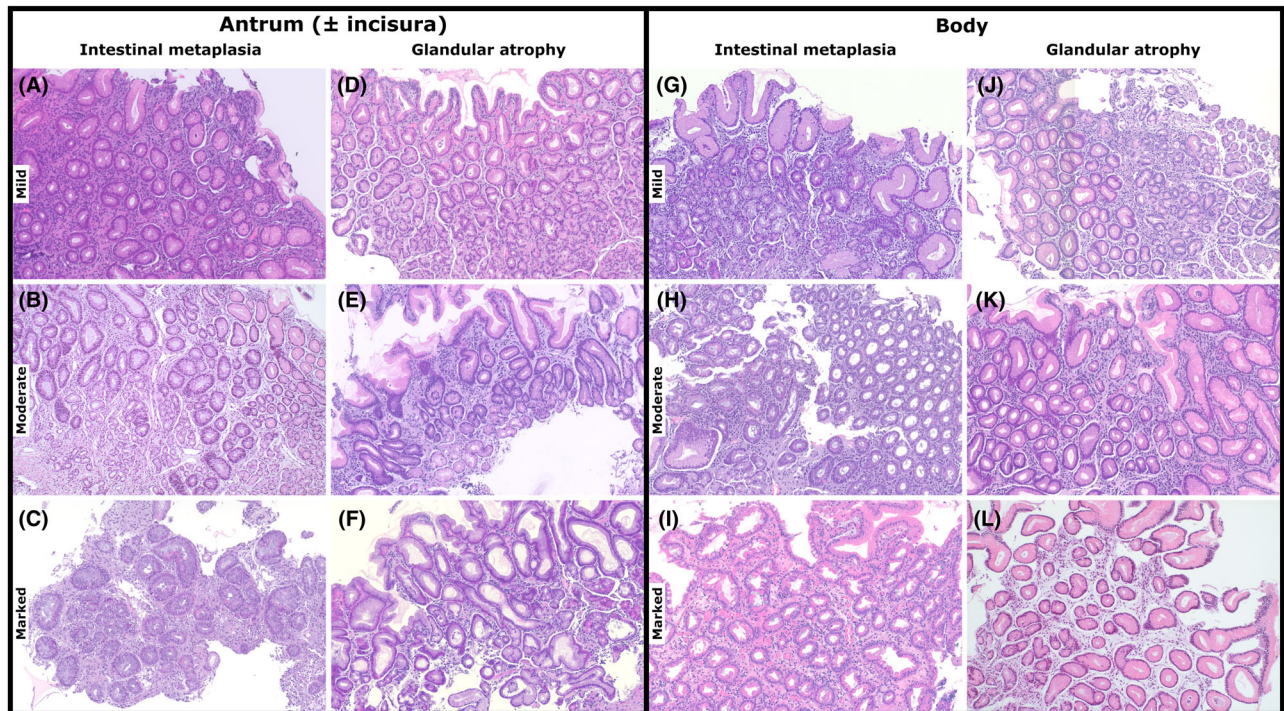


Figure 1. Histological severity assessment of GA and IM to obtain the OLGIMA score, based on the Sydney recommendations. Left panel: gastric mucosa from the antrum ± incisura is scored for both intestinal metaplasia as mild (A), moderated (B), marked (C), and for glandular atrophy as mild (D), moderated (E), marked (F). Right panel: gastric mucosa from the corpus is scored for both intestinal metaplasia as mild (G), moderated (H), marked (I), and for glandular atrophy as mild (J), moderated (K), marked (L). The OLGIMA stage results from the highest score of either intestinal metaplasia and/or atrophy from both locations, antrum/incisura and corpus.

corpus), in line with the Updated Sydney System. The severity of GA and IM was documented separately for both the antrum (including incisura) and corpus and was categorized into four levels: absence (score 0), mild (score 1), moderate (score 2), and marked (score 3). The OLGIMA staging or severity was defined as the highest score for either of them in both topographical areas (Figures 1 and 2A). After that, staging was done according to the same approach used by the OLGA and OLGIM systems (Figure 2B).^{8,11}

STATISTICAL ANALYSIS

A descriptive analysis was conducted using SPSS version 27 (IBM Corp, Armonk, NY, USA). Absolute and relative frequencies were calculated for categorical variables. For quantitative variables, mean and standard deviation were computed if they had a normal distribution, while median and interquartile ranges were used otherwise. The diagnostic yield of OLGIM and OLGIMA, in terms of sensitivity, specificity, positive predictive value (PPV), negative predictive value (NPV), and accuracy, was calculated using the total number of patients at higher risk as the *gold standard*. The concordance for detecting patients at higher risk of the new OLGIMA proposal and the previous OLGIM

was estimated using Cohen's Kappa coefficient. The one-sample proportion test was employed to evaluate the ability of each strategy to detect patients at high risk. Moreover, the ROC curve for OLGIM and OLGIMA was plotted.

Results

A total of 1149 first diagnostic UGI endoscopies with gastric protocolized biopsies were recorded during this period. Thirty cases with dysplasia, 12 cases with cancer, and 67 cases with histological suspicion of chronic atrophic autoimmune gastritis were excluded. In addition, 42 cases were also excluded due to lack of information about the severity of GA or IM. A total of 998 patients were finally included in this study (Figure S1).

PATIENT'S CHARACTERISTICS

Patient's median age was 57 years old (IQR 46–67), and 64% were women. *Hp* infection was identified in 34.4% (344) patients, being active in 18.5%, and with a medical record of *Hp* eradication in 15.9% (159) cases. 19.2% (192) patients had GA, being limited to the antrum/incisura in 113 (11.3%), and

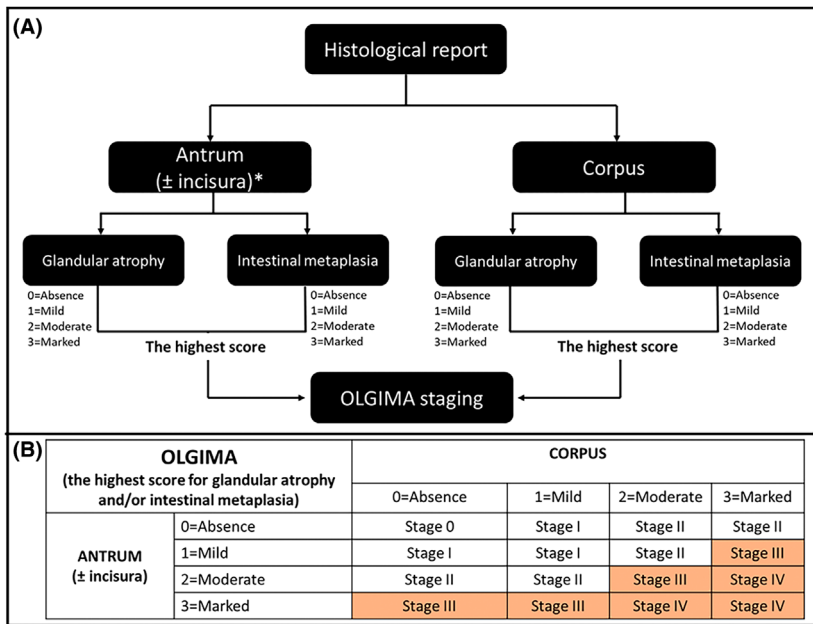


Figure 2. Flow diagram of the OLGIMA (operative link on gastric intestinal metaplasia and glandular atrophy assessment) staging system. (A) For both antrum including incisura and orpus, assessment of glandular atrophy and intestinal metaplasia is performed, according to the Upstaged Sydney consensus. The highest score will be used from both locations to obtain the OLGIMA stage. (B) The gastric cancer risk assessment according to the OLGIMA system is staged from 0 to IV. *In our study, a biopsy from the incisura was taken in all patients and included in the antrum vial.

Table 1. Demographics and patient characteristics

Characteristics	N (total 998)	%
Age, median (IQR)	57	46–67
<i>Sex</i>		
Women	634	63.5
<i>Hp status</i>		
Negative	654	65.5
Eradicated	159	15.9
Positive	185	18.5
<i>Glandular atrophy severity</i>		
Antrum (including incisura)		
Absence	806	80.8
Presence	192	19.2
Mild	137	13.7
Moderate	50	5.0
Marked	5	0.5
Corpus		
Absence	919	92.1
Presence	79	7.9
Mild	49	4.9
Moderate	23	2.3
Marked	7	0.7
<i>Glandular atrophy distribution</i>		
Absence		
Presence		
Limited to the antrum/incisura	113	11.3
Extensive	79	7.9
Non-advanced	57	5.7
Advanced	22	2.2
<i>IM severity</i>		
Antrum (including incisura)		
Absence	839	84.1
Presence	159	15.9
Mild	101	10.1
Moderate	43	4.3

Continued

Table 1. (Continued)

Characteristics	N (total 998)	%
Marked	15	1.5
Corpus		
Absence	953	95.5
Presence	45	4.5
Mild	29	2.9
Moderate	13	1.3
Marked	3	0.3
<i>IM distribution</i>		
Absence	839	84.1
Presence		
Limited to the antrum/incisura	114	11.4
Extensive	45	4.5
Non-advanced	32	3.2
Advanced	13	1.3
<i>IM severity and distribution (OLGIM)</i>		
OLGIM 0	839	84.1
OLGIM I	99	9.9
OLGIM II	33	3.3
OLGIM III	24	2.4
OLGIM IV	3	0.3

Hp, *Helicobacter pylori*; IM, intestinal metaplasia; IQR, interquartile range; OLGIM, operative link on gastric intestinal metaplasia assessment.

extensive in 7.9% (79) cases. IM was identified in 15.9% (159) patients; in 11.4% (114), it was limited to the antrum/incisura, and extensive in 4.5% (45) cases (Table 1).

PATIENTS AT HIGH-RISK FOR GC

The OLGIM system detected 27 (2.7%) patients at stages III–IV. Advanced GA was detected in 22 (2.2%) patients (Table 1). By considering both IM and GA at stage III–IV, 39 (3.9%) high-risk patients were detected: 17 (1.7%) with OLGIM stage III–IV, 12 (1.2%) with advanced GA, and 10 (1%) meeting both criteria of OLGIM stage III–IV and advanced GA (Table 2).

Table 2. Glandular atrophy and intestinal metaplasia at high risk for GC

GC high-risk patients, <i>n</i> (%)	Glandular atrophy				Total
	Absence	Presence		Advanced	
		Limited	Extensive Non-advanced		
Intestinal metaplasia	Absence	Limited	Non-advanced	Advanced	Total
OLGIM 0	743 (74.3)	58 (5.8)	34 (3.4)	4 (0.4)	839 (83.9)
OLGIM I	48 (4.8)	32 (3.2)	15 (1.5)	4 (0.4)	99 (9.9)
OLGIM II	8 (0.8)	14 (1.4)	7 (0.7)	4 (0.4)	33 (3.3)
OLGIM III	7 (0.7)	7 (0.7)	1 (0.1)	9 (0.9)	24 (2.4)
OLGIM IV	0	2 (0.2)	0	1 (0.1)	3 (0.3)
Total	806 (80.6)	113 (11.3)	57 (5.7)	22 (2.2)	998 (100)

Thirty-nine patients were identified as being at high-risk for GC: 17 with OLGIM III–IV, 12 with extensive advanced GA, and 10 meeting both criteria of OLGIM III–IV and extensive advanced GA.

GA, glandular atrophy; GC, gastric cancer; OLGIM, operative link on gastric intestinal metaplasia assessment.

Table 3. OLGIMA staging versus OLGIM system and glandular atrophy severity

Intestinal metaplasia	OLGIMA, <i>n</i> (%)					Total
	0	I	II	III	IV	
OLGIM 0	743 (74.3)	75 (7.5)	17 (1.7)	3 (0.3)	1 (0.1)	839 (84)
OLGIM I	0	86 (8.6)	9 (0.9)	3 (0.3)	1 (0.1)	99 (10)
OLGIM II	0	0	29 (2.9)	4 (0.4)	0	33 (3.3)
OLGIM III	0	0	0	17 (1.7)	7 (0.7)	24 (2.4)
OLGIM IV	0	0	0	0	3 (0.3)	3 (0.3)
Total	743 (74.3)	161 (16.2)	55 (5.5)	27 (2.7)	12 (1.2)	998 (100)
Glandular atrophy						
Absence	743 (74.3)	48 (4.8)	8 (0.8)	7 (0.7)	0	806 (80.6)
Limited	0	77 (7.7)	27 (2.7)	7 (0.7)	2 (0.2)	113 (11.3)
Extensive						
Non-advanced	0	36 (3.6)	20 (2)	0	1 (0.1)	57 (5.7)
Advanced	0	0	0	13 (1.3)	9 (0.9)	22 (2.2)
Total	743 (74.3)	161 (16.1)	55 (5.5)	27 (2.7)	12 (1.2)	998 (100)

Applying the OLGIMA approach, all 39 (3.9%) patients at high risk for GC were categorized at stages III and IV, including 27 patients at OLGIM III–IV and 22 patients with extensive advanced GA.

GA, glandular atrophy; GC, gastric cancer; OLGIM, operative link on gastric intestinal metaplasia assessment; OLGIMA, operative link on gastric intestinal metaplasia and glandular atrophy assessment.

OLGIMA VERSUS OLGIM STRATEGY

According to the OLGIMA approach, 39 (3.9%) patients were classified at stages III and IV, thus at

high risk for GC. It included all OLGIM III–IV patients plus those with OLGIM 0–II and advanced GA (Table 3, Figure 3). The sensitivity, specificity, PPV, NPV, and accuracy for OLGIM were 69.2%, 100%,

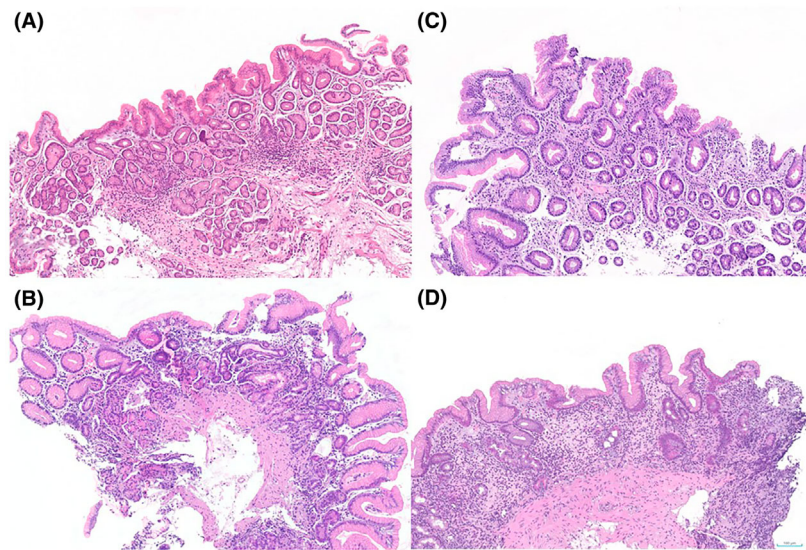


Figure 3. Patients upstaged with the OLGIMA system. One case without intestinal metaplasia in antral (A) or corporal biopsies (B), was initially classified as OLGIM 0. Notice the mild atrophy in the antrum (A) and severe atrophy in the corpus (B), thus, finally upstaged to OLGIMA III. Another case with mild intestinal metaplasia in both antrum (C) and corpus (D), classified as OLGIM II, had marked atrophy in both sites, and was upstaged to OLGIMA IV.

Table 4. Diagnostic yield and concordance of OLGIM and OLGIMA

	Sensibility	Specificity	PPV	NPV	Accuracy	<i>K</i>
OLGIM	69.2 (66.4–72.1)	100 (100)	100 (100)	98.8 (98.1–99.5)	98.8 (98.1–99.5)	0.81
OLGIMA	100 (100)	100 (100)	100 (100)	100 (100)	100 (100)	1

The *gold standard* is the total number of patients identified as being at higher risk, including those with OLGIM III–IV and patients with advanced glandular atrophy.

K, Cohen's kappa coefficient; NPV, negative predictive value; OLGIM, operative link on gastric intestinal metaplasia assessment; OLGIMA, operative link on gastric intestinal metaplasia and glandular atrophy assessment; PPV, positive predictive value.

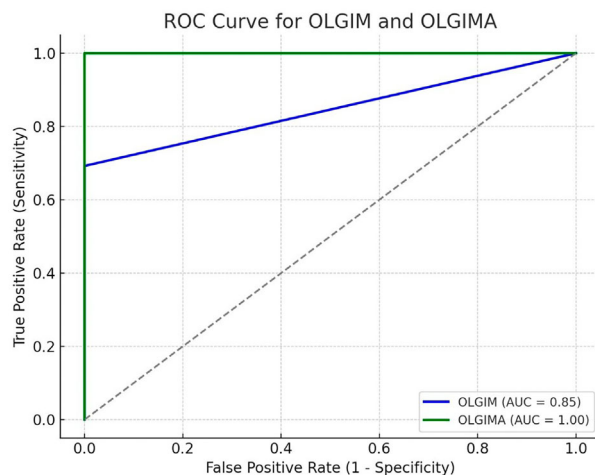


Figure 4. ROC curve for OLGIM and OLGIMA in detecting high-risk patients.

100%, 98.8% and 98.8%, while for OLGIMA, they were 100%, 100%, 100%, 100% and 100%, respectively. The concordance of OLGIMA in detecting patients at higher risk was perfect ($\kappa = 1$), whereas it was 0.81 for OLGIM (Table 4, Figure 4). The proportion of patients detected by OLGIM was significantly lower, being 27/39 (69%) compared with OLGIMA, which detected all high-risk patients (100%) ($P < 0.01$).

Discussion

The results of this multicentre study support the clinical utility of the OLGIMA classification system for histological GC risk assessment. OLGIMA demonstrated a greater sensitivity than OLGIM by identifying all OLGIM stage III–IV patients and additional high-risk

patients staged as OLGIM 0–II but with advanced GA. In all cases, the final OLGIMA stage was equal to or higher than the corresponding OLGIM stage. Thus, it reinforces its association with GC risk and highlights its complementary value in minimizing missed diagnoses.

Although the OLGA system is a reliable indicator of the GC risk of an individual patient, it is not an easy system to implement in routine, as it combines both the atrophy score and its topography. The atrophy score is calculated at each mucosal compartment as an average score of any histological subtype of atrophy, including IM. The changes observed in antral versus oxyntic mucosa result in five gastritis stages (0–IV) with different GC risk. Unlike the OLGA system, the OLGIMA system offers a simplified approach considering the most severe finding between GA and IM. This is a conceptual difference from the OLGA system, which averages atrophy scores between compartments. In contrast, OLGIMA uses the most severe histological finding — either GA or IM — as the basis for staging, regardless of its location. This approach enhances sensitivity, allowing the system to rescue patients who might otherwise be misclassified as low-risk under OLGA criteria due to the dilutional effect of averaging. The OLGIMA retains the OLGIM simplicity, enhancing the clinical relevance and reproducibility of pathology reports without modifying established diagnostic protocols, and clinicians can easily determine the OLGIMA stage using routinely reported histological data based on the Sydney consensus. The OLGIMA system does not require additional diagnostic efforts or novel staining techniques. It simply reframes existing histological information, offering a standard staging language that can guide surveillance recommendations. This streamlining of risk assessment enhances sensitivity without sacrificing reproducibility or clinical practicality.

IM is a marker of GA and is by far the most reliable histological marker for CAG, being considered a more advanced step in the gastric carcinogenesis process. Previous studies have demonstrated that the OLGIM system, focusing exclusively on IM, exhibits superior agreement among pathologists than OLGA. In contrast, GA has historically been less reproducible due to difficulties in grading, particularly for low-grade GA, or when biopsy orientation is suboptimal and only superficial foveolar epithelium is visible. Despite the clinical value of IM, histological evaluation in routine practice can be limited by factors such as biopsy quality (targeted vs. random), orientation, and depth. In our study, the known challenges in GA assessment did not limit the classification process, underscoring the potential shortcoming of relying

solely on OLGIM in clinical practice. The presence of IM, even if mild, should alert the pathologist to the possible presence of moderate to marked GA. Indeed, our results show that OLGIMA successfully identified all high-risk patients classified as OLGIM stage III–IV, as well as additional patients staged as OLGIM 0–II who had advanced GA.

In addition to validating the OLGIMA staging system, this study provides relevant data on the prevalence of the primary trigger for CAG and GC, *Hp* infection in a real-world Spanish population. This infection is involved in nearly 90% of GC cases, encompassing both intestinal and diffuse types.¹⁶ Consequently, *Hp* eradication is recommended as a crucial element in strategies for both primary and secondary GC prevention.^{17,18} In the Spanish population, the overall *Hp* infection prevalence is estimated to be around 55%.¹⁹ However, many patients undergo primary care testing and treatment, experiencing symptom improvement and avoiding UGI endoscopy.²⁰ In our study, among 998 patients undergoing a diagnostic UGI endoscopy for any indication, the *Hp* prevalence was 34.4%. It is important to note that even after successful *Hp* eradication, certain individuals with CAG remain at a heightened risk for GC.^{21,22} Therefore, implementing reliable histological risk stratification systems becomes essential in guiding long-term surveillance strategies and ensuring appropriate follow-up for patients who remain at risk even after *H. pylori* eradication.

Gastric dysplasia is widely recognized as a direct precursor to GC,¹ thus, patients with dysplasia were excluded from our study. In contrast, the stratification of cancer risk among patients with CAG remains a challenge, as it requires identifying those with a higher probability of progression. Focusing on the histological landscape of CAG, our study also provides insights into the prevalence of these histological conditions that mediate this risk. Among the 998 patients included, the prevalence of GA and IM was 19.2% and 15.9%, respectively. These results are consistent with prior meta-analyses, which estimated pooled prevalence rates of 22.8% for GA and 21.7% for IM in 2014,²³ and 21.7% and 11.3% in 2024.²⁴ Our numbers fall within the expected range for a low-risk European population and add valuable epidemiological context.

Focusing on the more advanced forms of mucosal damage, the prevalence of extensive GA and IM, for low-intermediate risk countries has been reported at 7.3% and 7.7%, respectively, in the 2014 meta-analysis.²³ In our cohort, we had similar prevalences for GA (7.9%), but slightly lower for IM (4.5%),

confirming that a relevant proportion of patients present with extensive histological involvement. When applying the OLGIMA and OLGIM systems, which consider not only the extent but also the severity of conditions, we identified 3.9% and 2.7% of patients, respectively, as stage III–IV, the high-risk categories that are candidates for endoscopic surveillance. These findings confirm the value of histological staging for selecting individuals who may benefit most from structured follow-up strategies. This reinforces the need for accurate histological evaluation and reporting in routine clinical practice to support timely identification and surveillance of premalignant gastric conditions.

The main strength of this study lies in its multicentre design, including UGI endoscopies with protocolized gastric biopsies, involving several community and referral Spanish hospitals. This approach allows for the collection of real-life data, reflecting daily clinical practices and offering insights into the prevalence of *Hp* infection and CAG. Importantly, OLGIMA is built on the Updated Sydney classification, which is already widely used by pathologists around the world. It does not require additional stains or special training and may therefore serve as a practical second-layer strategy, particularly valuable in settings where OLGIM is already used or where subtyping IM is not systematically reported. OLGIMA offers an opportunity to rescue additional patients, using a more systematic and standardized histological approach.

The primary limitation of our study stems from its cross-sectional design, which precludes the acquisition of long-term data to assess the predictive ability for GC risk. However, the ability of the OLGIMA system for detecting high-risk patients was already validated by previous studies with the OLGIM system. The new OLGIMA system complements the OLGIM risk stratification system by upstaging those with stage 0-II and advanced GA. Another limitation is that we did not include pseudopyloric metaplasia (SPeM) in our risk model, although SPeM is a recognized risk factor for GC. Nevertheless, the recognition and quantification of the replacement of normal oxyntic glands by pyloric or pseudopyloric-type glands expressing spasmodic polypeptide are better recognized using TFF2 immunostains and dedicated training. Nevertheless, SPeM deserves further attention as it may represent an important histological marker of risk. Future refinements of OLGIMA may explore the integration of SPeM once its implementation in routine diagnosis becomes more feasible. A third important limitation is that the exact assessment of the severity of GA may not be possible for biopsies that are not well oriented, which, in some

cases, would limit an accurate application of the OLGIMA system. Despite this limitation regarding GA, the final OLGIMA stage will never be inferior to the OLGIM stage. To elucidate the potential utility of this simple new scoring system, future studies should undertake direct comparison with the OLGA system. Moreover, the significance of upgrading patients OLGIM 0–II with advanced GA and the different types of IM should be considered in future studies, which should also include intra and inter-observer analysis of the different scoring systems.

In conclusion, with this system, pathologists are encouraged to report the severity of both IM and GA, as routinely done following the Updated Sydney consensus. The final staging, however, should be determined by the more advanced condition — either IM or GA. Based on this rule, the OLGIMA staging system encompasses all OLGIM III–IV cases, and has demonstrated its usefulness in identifying patients at higher risk, by upgrading OLGIM 0-II patients with advanced GA into a higher risk group (OLGIMA stage III–IV). This approach addresses the limitations of the OLGA and OLGIM systems by integrating GA and IM severity as recommended by the Updated Sydney consensus, enhancing risk stratification in clinical practice.

Author contributions

Conception and design: PGDG, MC, SM, ACu, GFE, LM, and EA. Data acquisition: NFV, JLL, IA, PFD, EBA, JTT, RV, MTS, AH, SPO, HN, OP, CM, DZ, LH, LEY, GHo, GHl, MJDA, SZ, AHDT, and ACo. Analysis and interpretation of data: PGDG, MC, GFE, LM, and EA. Article drafting and critical revision for important intellectual content: PGDM, MC, EA, MGF, LM. Final approval of the version to be published: All authors.

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Conflict of interests

The authors declare no conflicts of interest.

Data availability statement

Data sharing is not applicable to this article as no new data were created or analysed.

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Supporting Information

Additional Supporting Information may be found in the online version of this article:

Figure S1. Flowchart of patients included in the study.