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Age–symptom dissociation and histological burden in *Helicobacter pylori* gastritis: a retrospective cross-sectional study at a Colombian referral centre, 2020–2024

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Abstract

Background: *Helicobacter pylori* gastritis may progress to gastric cancer through premalignant histological changes. Whether symptom burden reliably reflects histological stage across age groups remains unclear, particularly in high-risk Latin American populations. This study aimed to describe OLGA/OLGIM histological stages and symptom profiles by age group in a Colombian referral cohort with biopsy-confirmed *H. pylori* infection and to evaluate age as a predictor of histological stage independent of symptom burden.

Methods: Retrospective cross-sectional study of 801 patients with biopsy-confirmed *H. pylori* infection treated at a Colombian tertiary referral centre (2020–2024). Histological stages were assigned through structured reclassification of pathology reports using OLGA/OLGIM staging grids. Associations were assessed using logistic regression, ordinal logistic regression, and exploratory receiver operating characteristic analysis.

Results: Symptom data were available for 432 patients; 367 (84.9%) were symptomatic. Children had higher odds of abdominal pain (OR 3.25, 95% CI 2.10–5.04) and nausea (OR 3.98, 95% CI 1.91–8.30), whereas older adults had lower odds of pain (OR 0.24, 95% CI 0.14–0.39) and dyspepsia (OR 0.45, 95% CI 0.23–0.89). Among 521 stageable patients, OLGA/OLGIM stage $\geq$ II increased from 1.0% in children to 11.0% in older adults. Age was the sole independent predictor of higher histological stage (OR per year 1.043, 95% CI 1.028–1.059). Three-site topographic sampling was achieved in only 25.8% of patients; complete sampling was associated with a 3.3-fold higher prevalence of stage $\geq$ II.

Conclusions: Symptoms declined with age while histological burden increased, demonstrating an age–symptom dissociation. These findings support age-informed rather than symptom-driven histological risk assessment and highlight a modifiable quality gap in biopsy sampling practice.

Keywords: *Helicobacter pylori*; OLGA; OLGIM; gastric atrophy; intestinal metaplasia; gastric cancer

Background

Helicobacter pylori infection affects approximately 50% of the global population and remains the principal cause of chronic active gastritis, peptic ulcer disease, and gastric adenocarcinoma [1]. The pathological trajectory from normal mucosa to carcinoma follows the Correa cascade, passing through atrophic gastritis, intestinal metaplasia, and dysplasia [2]. Risk of progression is determined by complex interactions among bacterial virulence factors, host genetics, and environmental exposures [1].

The Operative Link on Gastritis Assessment (OLGA) and Operative Link on Gastric Intestinal Metaplasia Assessment (OLGIM) staging systems provide structured frameworks for quantifying premalignant histological burden in gastric biopsy specimens [3,4]. Prospective data and meta-analyses confirm that patients with OLGA/OLGIM stages III–IV carry significantly higher gastric cancer and high-grade dysplasia risk, supporting intensified endoscopic surveillance [5,6]. Adequate staging requires topographic biopsy sampling, classically from the antrum, incisura angularis, and corpus, a requirement that is not uniformly achieved in everyday practice [1,3,6,7].

A clinically important question is whether symptom burden reliably reflects histological severity. In children, *H. pylori* infection is frequently asymptomatic; when symptoms occur, they are non-specific and do not reliably distinguish infected from uninfected peers [8,9]. In adults, *H. pylori* may be associated with dyspeptic symptoms, but symptom burden alone is insufficient to identify patients with premalignant histological changes, particularly in chronic atrophic gastritis [6,7]. If symptom burden and histological stage are systematically misaligned across age groups—

older adults bearing greater premalignant burden but fewer symptoms—then symptom-driven surveillance would under-identify the highest-risk patients.

Despite the relevance of *H. pylori* infection and gastric cancer in Colombia, no age-stratified characterisation of OLGA/OLGIM stages and their relationship to symptoms has been reported. This study aimed to: (i) describe the distribution of OLGA/OLGIM stages and symptom profiles by age group in patients with biopsy-confirmed *H. pylori* at Fundación Santa Fe de Bogotá (FSFB); (ii) explore the association between age, symptoms, and advanced histological stage; and (iii) evaluate age as a potential marker for identifying patients at higher histological risk independent of symptom burden.

Methods

Study design and setting

This was a retrospective cross-sectional study conducted at FSFB, a tertiary-care university hospital in Bogotá, Colombia. Patients were identified through the hospital information system (HIS-ISIS) with records from January 2020 to December 2024.

Study population

Eligibility required biopsy-confirmed *H. pylori* infection. Patients with incomplete medical records or unavailable pathology reports were excluded. Stratified random sampling ensured proportional representation by sex and by four predefined age strata: children (< 18 years), young adults (18–29 years), middle-aged adults (30–59 years), and older adults ($\geq$ 60 years). The final cohort comprised 801 patients.

Data extraction

Extracted variables included age, sex, area of residence, occupation, and gastrointestinal symptoms (abdominal pain, dyspepsia, heartburn, nausea, hyporexia, abdominal distension, acute diarrhoea, chronic diarrhoea, and other/non-specific symptoms). Symptom data were available for 432 patients; the remaining 369 had no symptom information recorded and were excluded from symptom analyses. Endoscopic findings and biopsy site information were also recorded.

OLGA/OLGIM reclassification workflow

Most pathology reports in HIS-ISIS predated routine OLGA/OLGIM staging. A structured reclassification workflow was therefore implemented. Trained research assistants abstracted each report using a pre-specified form capturing Sydney System descriptors for each available anatomic site (antrum, corpus, incisura angularis): degree of glandular atrophy, presence and extent of intestinal metaplasia, *H. pylori* colonisation grade, and acute and chronic inflammation grades. OLGA stages were assigned by mapping atrophy scores to the 2007 OLGA staging grid [3]; OLGIM stages used reported intestinal metaplasia scores [4]. When both could be assigned, the higher stage was recorded as the composite OLGA/OLGIM stage.

Quality-control procedures included: (a) a standardised training session with review of the data dictionary and mapping rules; (b) independent double extraction of 20 pilot reports by two abstractors, with discrepancies resolved by consensus; and (c) random verification of 10% of entered records. Patients were considered stageable only when pathology reports provided sufficient site-specific histological detail from the minimum anatomical locations required for OLGA/OLGIM staging under the updated Sydney System: the antrum–incisura angularis compartment and the corpus. Reports lacking sufficient information from either compartment were excluded from staging analyses. Of 801 patients, 521 had adequate data for staging.

Statistical analysis

Categorical variables are reported as frequencies and percentages; continuous variables as medians with interquartile ranges or means with standard deviations, as appropriate. Normality was assessed using the Shapiro–Wilk test. Pearson’s chi-square test was used to examine associations between categorical variables; the Kruskal–Wallis test compared age across groups; and Spearman’s rank correlation assessed the association between age and OLGA/OLGIM stage. Ordinal logistic regression was used to identify predictors of higher OLGA/OLGIM stage. Candidate predictors were selected using forward stepwise selection with an entry threshold of $p < 0.05$. The proportional odds assumption was assessed for the final ordinal model using a generalised ordered logit model and was not violated. Separate logistic regression models estimated odds ratios and 95% confidence intervals for each symptom by age group, with middle-aged adults as the reference category. All tests were two-sided, with statistical significance set at $\alpha = 0.05$. Analyses were performed in Stata 17 (StataCorp LLC, College Station, TX, USA).

An exploratory ROC analysis examined the discriminative capacity of age for OLGA/OLGIM stage $\geq$ II. Given only 30 events (5.8% of staged patients), 95% confidence intervals (CIs) for the AUC were estimated by bootstrap (2000 iterations). A prespecified sensitivity analysis was restricted to patients with three-site topographic sampling, including antrum, corpus, and incisura (antrum + corpus + incisura, $n = 194$).

Ethical approval

The study protocol (version 2.0, 24 November 2024) was approved by the Comité Corporativo de Ética en Investigación of Fundación Santa Fe de Bogotá (CCEI-17310-2025, Acta 02, 10 February 2025). The study was classified as minimal risk and used routinely collected clinical and pathology data analysed in anonymised form. The study was conducted in accordance

with the Declaration of Helsinki, Colombian regulations for research involving human participants, and institutional policies on confidentiality and personal data protection.

Use of large language model tools

Large language model tools (Claude, Anthropic, and ChatGPT, OpenAI) were used solely to support English translation, language editing, and adaptation of the manuscript to journal formatting requirements. No AI tool was used to generate or analyse study data. All scientific content, data interpretation, and conclusions were generated and verified by the authors, who take full responsibility for the accuracy and integrity of the final manuscript.

Results

Cohort characteristics and patient flow

Of 801 patients with biopsy-confirmed *H. pylori* infection, 432 (54.0%) had symptom data available and 521 (65.0%) had sufficient histological information for OLGA/OLGIM staging. The 280 patients excluded from staging did not differ meaningfully from staged patients in age (median 47 vs 49 years). The cohort was predominantly female (58.8%), urban (98.0%), and comprised of middle-aged or older adults (72.3%). Sociodemographic and clinical characteristics are presented in Table 1.

Table 1. Sociodemographic and clinical characteristics of the study cohort (N = 801).

Characteristic	n (%)
Sex (N = 801)	
Male	330 (41.2)
Female	471 (58.8)
Age group (N = 801)	
Children (< 18 years)	118 (14.7)
Young adults (18–29 years)	104 (13.0)
Middle-aged adults (30–59 years)	323 (40.3)
Older adults (≥ 60 years)	256 (32.0)
Residence (N = 788)	
Urban	772 (98.0)
Rural	16 (2.0)
Occupation (N = 774)	
Professional	442 (57.1)
Non-professional	332 (42.9)
Symptoms (N = 432) †	
Symptomatic	367 (84.9)
Abdominal pain	202 (46.8)
Dyspepsia	69 (16.0)
Abdominal distension	56 (13.0)
Heartburn	47 (10.9)
Nausea	46 (10.7)
Acute diarrhoea	20 (4.6)
Chronic diarrhoea	19 (4.4)
Hyporexia	14 (3.2)
Other symptoms	143 (33.1)
Asymptomatic	65 (15.1)

†Symptom data were available for 432 patients; the remaining 369 had no symptom information in their clinical records. Percentages for individual symptoms are calculated over N = 432. Percentages may not sum to 100 due to rounding.

Symptom distribution by age group

Among 432 patients with symptom data, 367 (84.9%) reported at least one symptom. Abdominal pain (46.8%), dyspepsia (16.0%), and abdominal distension (13.0%) were the most frequent complaints. The high symptomatic rate likely reflects referral bias at a tertiary endoscopy centre.

Logistic regression models showed that several reported symptoms were less frequent with advancing age (Table 2). Children had higher odds of abdominal pain (OR 3.25, 95% CI 2.10–5.04), nausea (OR 3.98, 95% CI 1.91–8.30), hyporexia (OR 5.86, 95% CI 1.79–19.03), and non-specific symptoms (OR 4.13, 95% CI 2.48–6.91) compared with middle-aged adults, but lower odds of heartburn (OR 0.21, 95% CI 0.05–0.88). Young adults had higher odds of chronic diarrhoea (OR 3.83, 95% CI 1.26–11.66), heartburn (OR 2.02, 95% CI 1.02–4.01), and non-specific symptoms (OR 2.20, 95% CI 1.24–3.91). Older adults had significantly lower odds of abdominal pain (OR 0.24, 95% CI 0.14–0.39), dyspepsia (OR 0.45, 95% CI 0.23–0.89), and heartburn (OR 0.24, 95% CI 0.09–0.63).

Table 2. Symptom odds ratios by age group (N = 432).

Symptom	Children (< 18 yr)	Young adults (18–29 yr)	Middle-aged (30–59 yr) [Reference]	Older adults (≥ 60 yr)
Abdominal pain	3.25 (2.10–5.04) ***	0.73 (0.44–1.23)	1.00 (ref)	0.24 (0.14–0.39) ***
Dyspepsia	1.33 (0.69–2.55)	0.97 (0.46–2.05)	1.00 (ref)	0.45 (0.23–0.89) *
Heartburn	0.21 (0.05–0.88) *	2.02 (1.02–4.01) *	1.00 (ref)	0.24 (0.09–0.63) **
Nausea	3.98 (1.91–8.30) ***	1.85 (0.75–4.59)	1.00 (ref)	0.53 (0.20–1.40)
Hyporexia	5.86 (1.79–19.03) **	NA‡	1.00 (ref)	NA‡
Chronic diarrhoea	1.38 (0.34–5.62)	3.83 (1.26–11.66) *	1.00 (ref)	0.63 (0.17–2.54)
Abdominal distension	1.21 (0.56–2.63)	1.39 (0.64–3.03)	1.00 (ref)	0.70 (0.35–1.41)
Other symptoms	4.13 (2.48–6.91) ***	2.20 (1.24–3.91) **	1.00 (ref)	1.52 (0.94–2.45)

ORs are from separate simple logistic regression models among patients with *H. pylori* infection and available symptom data; middle-aged adults (30–59 years) were the reference group. Values are OR (95% CI). * $p < .05$; ** $p < .01$; *** $p < .001$. ‡Model not estimable because of zero events in the corresponding cell. OR, odds ratio; CI, confidence interval; NA, not applicable.

Biopsy sampling adequacy

The median number of biopsy fragments per endoscopy was 4 (IQR 3–5). Three-site topographic sampling, including antrum, corpus, and incisura was achieved in 201 of 780 patients with available site data (25.8%). Single-site sampling occurred in 28.3% of cases; antrum–corpus two-site sampling was the most frequent pattern (40.8%; see Additional file 1). Staged patients with complete three-site sampling had a markedly higher prevalence of OLGA/OLGIM stage $\geq$ II (10.3%) than those with incomplete sampling (3.1%), indicating probable underestimation of advanced disease in this cohort.

OLGA/OLGIM stage distribution

Of 521 staged patients, the great majority had low stages: stage 0 in 444 (85.2%), stage I in 45 (8.6%), stage II in 21 (4.0%), and stage III in 9 (1.7%). OLGA/OLGIM stage $\geq$ II was present in 30 patients (5.8%). Stage distribution by age group is shown in Table 3. OLGA/OLGIM stage $\geq$ II was rare in children (1.0%) and young adults (1.9%) and increased to 4.6% in middle-aged adults and 11.0% in older adults.

Table 3. OLGA/OLGIM stages by age group (N = 521).

Age group	Staged n	Stage 0 n (%)	Stage I n (%)	Stage II n (%)	Stage III n (%)	Stage IV n (%)
Children (< 18 yr)	99	98 (99.0)	0 (0.0)	1 (1.0)	0 (0.0)	0 (0.0)
Young adults (18–29 yr)	53	50 (94.3)	2 (3.8)	1 (1.9)	0 (0.0)	0 (0.0)
Middle-aged (30–59 yr)	194	165 (85.1)	20 (10.3)	6 (3.1)	3 (1.5)	0 (0.0)
Older adults ($\geq$ 60 yr)	173	131 (75.7)	23 (13.3)	13 (7.5)	6 (3.5)	0 (0.0)
Total†	521	444 (85.2)	45 (8.6)	21 (4.0)	9 (1.7)	0 (0.0)

Percentages are calculated within each age group. OLGA, Operative Link on Gastritis Assessment; OLGIM, Operative Link on Gastric Intestinal Metaplasia. No patients were classified as stage IV. †Of 801 patients, 280 lacked sufficient biopsy report detail for staging and were excluded from this analysis.

Age as a predictor of histological stage

Ordinal logistic regression (n = 519 with complete age and staging data) identified age as the sole independent predictor of higher OLGA/OLGIM stage (OR per year 1.043, 95% CI 1.028–1.059, $p < .001$). In the stepwise multivariable model, older adults had higher odds (OR 2.17, 95% CI 1.30–3.62) and children had lower odds (OR 0.07, 95% CI 0.009–0.515) of higher OLGA/OLGIM stage. Sex, occupation, residence, and symptom variables were not independently associated.

An exploratory ROC analysis yielded an AUC of 0.72 (95% CI 0.63–0.80) for age discriminating OLGA/OLGIM stage $\geq$ II from low stages (0–I) in 519 staged patients (30 events, 5.8%). This estimate should be interpreted cautiously given the limited number of events, which constrains precision and precludes robust clinical utility claims. In the sensitivity analysis restricted to three-site topographic sampling (n = 194, 20 events), the AUC was 0.66 (95% CI 0.54–0.78), consistent with a modest and exploratory discriminative signal.

Discussion

In this single-centre cohort of 801 patients with biopsy-confirmed *H. pylori* infection, we observed an age–symptom dissociation. The frequency of reported symptoms declined with age, whereas higher OLGA/OLGIM stages became more frequent in older patients. Younger patients, particularly children, more often reported abdominal pain, nausea, and non-specific symptoms, yet had predominantly low-stage histology. Older adults, conversely, reported fewer symptoms but showed the greatest histological burden, including the highest prevalence of OLGA/OLGIM stage $\geq$ II. These findings suggest that, in this cohort, symptom-driven clinical assessment may under-identify patients with premalignant histological changes, especially among older adults.

The paediatric pattern observed in this cohort is consistent with current evidence indicating that gastrointestinal symptoms in children with *H. pylori* infection are non-specific and should not be used alone to guide testing or treatment decisions [8,9]. Cross-sectional data from schoolchildren in a resource-limited setting also found frequent gastrointestinal symptoms, including nausea and gastric pain, among *H. pylori*-seropositive children, while emphasising that the role of infection in abdominal symptoms remains uncertain [10]. Our findings suggest that this limitation may not be restricted to children, as reported symptoms did not reliably parallel histological severity across age groups. This should not be interpreted as evidence that age alone can replace histological staging, but rather as evidence that symptoms alone are an unreliable marker of histological risk in this cohort.

Among individual symptoms, heartburn illustrated a clinically relevant age-dependent pattern. Young adults had higher odds of heartburn, whereas children and older adults had lower odds. This pattern is biologically plausible, as antral-predominant *H. pylori* gastritis has been associated with higher acid output, whereas corpus-predominant or atrophic gastritis is associated with reduced acid secretion [11]. In the population-based HUNT Study, gastric atrophy, defined by low serum pepsinogen I, was associated with a markedly lower risk of reflux symptoms, while *H. pylori* seropositivity alone was not protective [12]. However, our study did not measure acid secretion, pepsinogen levels, or topographic gastritis phenotypes directly; therefore, this finding should be interpreted as exploratory. In older adults, the attenuation of symptoms alongside histological advancement may plausibly reflect chronic *H. pylori*-related mucosal injury, progressive glandular atrophy, reduced acid secretion, and cellular senescence, mechanisms implicated in the progression of chronic atrophic gastritis toward gastric cancer [1,13].

Age was the only independent predictor of higher OLGA/OLGIM stage in the ordinal model (OR per year, 1.043; 95% CI, 1.028–1.059). This finding is compatible with cumulative *H. pylori*-related mucosal injury over time, particularly in patients with long-standing infection. Longitudinal data from a Japanese cohort of 4,655 asymptomatic subjects followed for a mean of 7.7 years showed that gastric cancer risk increased stepwise with progression from *H. pylori*-associated gastritis to chronic atrophic gastritis and severe atrophic gastritis with extensive intestinal metaplasia [14]. In our cross-sectional cohort, the age gradient in OLGA/OLGIM stage may reflect a similar time-integrated process. Nevertheless, longitudinal studies are needed to determine whether age-stratified histological patterns predict future progression in Colombian patients.

The staging results must also be interpreted in light of incomplete biopsy sampling. Only 25.8% of patients with available site data had three-site topographic sampling, including antrum, corpus, and incisura. This is not merely a methodological limitation, but a clinically meaningful finding. Patients with complete three-site sampling had a substantially higher prevalence of OLGA/OLGIM stage $\geq$ II than those with incomplete sampling, suggesting probable underestimation of premalignant burden in routine practice. This pattern is consistent with large-scale evidence showing that adherence to Sydney System biopsy recommendations increases the detection of *H. pylori* gastritis and intestinal metaplasia [15], and with real-world Latin American data showing that implementation of the updated Sydney protocol improves the diagnostic yield of chronic atrophic gastritis, gastric intestinal metaplasia, autoimmune gastritis, and advanced OLGA stages [16]. It also highlights a modifiable quality gap in endoscopic practice. In high-risk settings, improving adherence to topographic biopsy protocols should be considered a necessary complement to identifying which patients should undergo endoscopy.

The Colombian context amplifies the relevance of these findings. Colombia is part of a region with a substantial gastric cancer burden, with marked geographic variation and higher rates reported in Andean populations [17,18]. Despite this burden, local data on OLGA/OLGIM staging remain limited. To our knowledge, this represents one of the largest Colombian cohorts of biopsy-confirmed *H. pylori* infection with age-stratified OLGA/OLGIM reclassification and symptom analysis. The finding that older adults had both fewer symptoms and greater histological burden supports histological risk assessment strategies that do not rely exclusively on symptom presentation. Rather than proposing a new clinical threshold, our results support the development of prospective, age-informed studies using standardised biopsy protocols to better define premalignant histological risk in Colombian and Latin American populations.

This study has several strengths. It included a large biopsy-confirmed cohort, used a structured OLGA/OLGIM reclassification workflow with quality-control procedures, and examined the relationship between age, symptoms, and histological stage in a Colombian referral setting. The main limitations are its retrospective cross-sectional design, which precludes inference about longitudinal progression; incomplete adherence to topographic biopsy sampling, which likely underestimated OLGA/OLGIM stage $\geq$ II; and symptom data available for only 54% of patients. In addition, we lacked information on bacterial virulence factors, prior eradication therapy, family history, environmental exposures, acid secretion, and pepsinogen biomarkers. Referral bias at a tertiary endoscopy centre may also have inflated the overall symptomatic rate and may limit generalisability to primary care or community settings.

Conclusions

Younger patients were more symptomatic despite predominantly low-stage histology, whereas older adults had fewer symptoms but greater histological burden. These findings support

age-informed histological risk assessment strategies rather than symptom-driven approaches alone, particularly in older patients. Prospective longitudinal studies with standardised biopsy protocols, complete symptom capture, and larger advanced-stage event numbers are required to validate age-informed approaches and assess histological trajectories after eradication therapy.

List of abbreviations

AI, artificial intelligence; AUC, area under the receiver operating characteristic curve; CI, confidence interval; FSFB, Fundación Santa Fe de Bogotá; HIS, hospital information system; IQR, interquartile range; OLGA, Operative Link on Gastritis Assessment; OLGIM, Operative Link on Gastric Intestinal Metaplasia Assessment; OR, odds ratio; ROC, receiver operating characteristic; SD, standard deviation.

Declarations

Ethics approval and consent to participate

The study protocol (version 2.0, 24 November 2024) was approved by the Comité Corporativo de Ética en Investigación of Fundación Santa Fe de Bogotá (CCEI-17310-2025, Acta 02, 10 February 2025). The study was classified as minimal risk and used routinely collected clinical and pathology data analysed in anonymised form. Individual informed consent was not required for this retrospective analysis of anonymised routinely collected data under the approval granted by the committee. The study was conducted in accordance with the Declaration of Helsinki, Colombian regulations for research involving human participants, and institutional policies on confidentiality and personal data protection.

Consent for publication

Not applicable.

Availability of data and materials

The datasets used and/or analysed during the current study are not publicly available because they contain clinical information subject to institutional confidentiality requirements, but they are available from the corresponding author on reasonable request and with permission from Fundación Santa Fe de Bogotá.

Competing interests

The authors declare that they have no competing interests.

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No external funding was received for this study.

Authors' contributions

EMO contributed to conceptualization, data curation, formal analysis, investigation, and manuscript review and editing. LAC contributed to data curation, formal analysis, investigation, methodology, and manuscript review and editing. JSPR contributed to conceptualization, methodology, supervision, validation, project administration, and manuscript review and editing. LJP contributed to investigation and data curation. PAN and ASS contributed to investigation, data curation, and manuscript review and editing. AMR contributed to methodology, validation, and manuscript review and editing. BEM contributed to conceptualization, methodology, supervision, validation, and manuscript review and editing. RDPL contributed to conceptualization, methodology, supervision, validation, project administration, and manuscript review and editing. All authors read and approved the final manuscript.

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Authors' information

Not applicable.

Additional files

Additional file 1 (.docx). Supplementary Table 1: Gastric biopsy sampling patterns in the study cohort (N = 801). Distribution of one-site, two-site, and three-site gastric biopsy sampling patterns, including missing site information.

References

- [1] Malfertheiner P, Camargo MC, El-Omar E, et al. Helicobacter pylori infection. Nat Rev Dis Primers 2023;9. <https://doi.org/10.1038/s41572-023-00431-8>.
- [2] Zhou J, Li J, Chen J, et al. Decoding inflammatory mediators in the Correa's cascade: From chronic gastritis to carcinogenesis and targeted therapies. Int Immunopharmacol 2025;162. <https://doi.org/10.1016/j.intimp.2025.115191>.
- [3] Rugge M, Meggio A, Pennelli G, et al. Gastritis staging in clinical practice: The OLGA staging system. Gut 2007;56:631–6. <https://doi.org/10.1136/gut.2006.106666>.
- [4] Capelle LG, de Vries AC, Haringsma J, et al. The staging of gastritis with the OLGA system by using intestinal metaplasia as an accurate alternative for atrophic gastritis. Gastrointest Endosc 2010;71:1150–8. <https://doi.org/10.1016/j.gie.2009.12.029>.
- [5] Benites-Goñi H, Cabrera-Hinojosa D, Latorre G, Hernandez A V., Uchima H, Riquelme A. OLGA and OLGIM staging systems on the risk assessment of gastric cancer: a

- systematic review and meta-analysis of prospective cohorts. *Therap Adv Gastroenterol* 2025;18. <https://doi.org/10.1177/17562848251325461>.
- [6] Lahner E, Zagari RM, Zullo A, et al. Chronic atrophic gastritis: Natural history, diagnosis and therapeutic management. A position paper by the Italian Society of Hospital Gastroenterologists and Digestive Endoscopists [AIGO], the Italian Society of Digestive Endoscopy [SIED], the Italian Society of Gastroenterology [SIGE], and the Italian Society of Internal Medicine [SIMI]. *Digestive and Liver Disease* 2019;51:1621–32. <https://doi.org/10.1016/j.dld.2019.09.016>.
- [7] Malfertheiner P, Megraud F, Rokkas T, et al. Management of *Helicobacter pylori* infection: the Maastricht VI/Florence consensus report. *Gut* 2022;71:1724–62. <https://doi.org/10.1136/gutjnl-2022-327745>.
- [8] Homan M, Jones NL, Bontems P, et al. Updated joint ESPGHAN/NASPGHAN guidelines for management of *Helicobacter pylori* infection in children and adolescents (2023). *J Pediatr Gastroenterol Nutr* 2024;79:758–85. <https://doi.org/10.1002/jpn3.12314>.
- [9] Nguyen J, Kotilea K, Bontems P, Miendje Deyi VY. *Helicobacter pylori* Infections in Children. *Antibiotics* 2023;12. <https://doi.org/10.3390/antibiotics12091440>.
- [10] Abbas M, Sharif FA, Osman SM, et al. Prevalence and Associated Symptoms of *Helicobacter pylori* Infection among Schoolchildren in Kassala State, East of Sudan. *Interdiscip Perspect Infect Dis* 2018;2018. <https://doi.org/10.1155/2018/4325752>.
- [11] Smolka AJ, Schubert ML. *Helicobacter pylori*-induced changes in gastric acid secretion and upper gastrointestinal disease. *Curr. Top. Microbiol. Immunol.*, vol. 400, Springer Verlag; 2017, p. 227–52. https://doi.org/10.1007/978-3-319-50520-6_10.

- [12] Nordenstedt H, Nilsson M, Johnsen R, Lagergren J, Hveem K. *Helicobacter pylori* Infection and Gastroesophageal Reflux in a Population-Based Study (The HUNT Study). *Helicobacter* 2007;12:16–22. <https://doi.org/10.1111/j.1523-5378.2007.00466.x>.
- [13] Zhang Z, Chen S, Li S, Zheng Y, Mai L, Zhang X. Association of *Helicobacter pylori* related chronic atrophic gastritis and gastric cancer risk: a literature review. *Front Med (Lausanne)* 2025;12. <https://doi.org/10.3389/fmed.2025.1504749>.
- [14] Ohata H, Kitauchi S, Yoshimura N, et al. Progression of chronic atrophic gastritis associated with *Helicobacter pylori* infection increases risk of gastric cancer. *Int J Cancer* 2004;109:138–43. <https://doi.org/10.1002/ijc.11680>.
- [15] Lash JG, Genta RM. Adherence to the Sydney System guidelines increases the detection of *Helicobacter* gastritis and intestinal metaplasia in 400 738 sets of gastric biopsies. *Aliment Pharmacol Ther* 2013;38:424–31. <https://doi.org/10.1111/apt.12383>.
- [16] Latorre G, Vargas JI, Shah SC, et al. Implementation of the updated Sydney system biopsy protocol improves the diagnostic yield of gastric preneoplastic conditions: Results from a real-world study. *Gastroenterol Hepatol* 2024;47:793–803. <https://doi.org/10.1016/j.gastrohep.2023.08.005>.
- [17] Torres J, Correa P, Ferreccio C, et al. Gastric cancer incidence and mortality is associated with altitude in the mountainous regions of Pacific Latin America. *Cancer Causes and Control* 2013;24:249–56. <https://doi.org/10.1007/s10552-012-0114-8>.
- [18] Torres-Roman JS, Alvarez CS, Guerra-Canchari P, et al. Sex and age differences in mortality trends of gastric cancer among Hispanic/Latino populations in the United States, Latin America, and the Caribbean. *The Lancet Regional Health - Americas* 2022;16:100376. <https://doi.org/10.1016/j.lana.2022.100376>.

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