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Histopathological Spectrum and Clinicopathological Correlates of *Helicobacter pylori*–Associated Gastritis in Western Libya: A Retrospective Cross-Sectional Study

Mayadah A. Rahoumah[1] | Ibrahim O. Eswaey[1] | Abdurraouf Zaet[2]*

[1]Department of Medical Laboratories, Faculty of Medical Technology, Sabratha University, Libya.
[2]Department of Dental Technology, Faculty of Medical Technology, University of Zawia, Libya.

ABSTRACT

Purpose: To delineate the histopathological spectrum of *Helicobacter pylori* associated gastritis, describe its clinicodemographic correlates, and determine the prevalence and severity patterns of infection among patients undergoing endoscopic gastric biopsy in western Libya.

Materials and Methods: A retrospective cross-sectional analysis was conducted on 136 histopathological specimens received between January 2025 and June 2026. Demographic, clinical, endoscopic, and histological data were extracted from laboratory records. *H. pylori* positivity was established histologically on hematoxylin and eosin staining. Descriptive statistics, chi-square tests, and the Mann Whitney U test were applied, with significance set at $p < 0.05$.

Results: Gastric biopsies accounted for 47.8% (n = 65) of all specimens. *H. pylori* was identified in 81.5% (n = 53; 95% CI: 70.4%–89.1%) of these biopsies. Infected patients were predominantly female (60.4%) with a mean age of 38.0 ± 15.1 years (range 17–76). Moderate chronic active gastritis predominated (64.2%), followed by severe (20.8%) and mild (13.2%) grades. Pangastritis was the commonest endoscopic pattern (39.6%). No significant association was found between infection and gender ($\chi^2 = 0.000$, $p = 1.000$) or age ($U = 235.000$, $p = 0.331$), nor did severity differ by sex ($\chi^2 = 1.300$, $p = 0.522$).

Conclusion: *H. pylori* infection is highly prevalent among biopsy-referred patients and most often presents as moderate chronic active gastritis. The absence of gender or age predisposition reflects the indiscriminate nature of the pathogen. Routine histopathological assessment of gastric biopsies remains essential for diagnosis, severity stratification, and gastric cancer risk assessment. (*Open J Biomed Res 2026;5:102-113*)

Keywords: Helicobacter pylori, chronic active gastritis, histopathology, gastric biopsy, endoscopy, Sydney system.

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*Corresponding author:
Abdurraouf Zaet
Email: a.zaet@zu.edu.ly

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AUDIOABSTRACT

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INTRODUCTION

The isolation of *Helicobacter pylori* by Marshall and Warren in 1984 fundamentally transformed our understanding of gastroduodenal disease, overturning the prevailing belief that the gastric mucosa was sterile and that peptic ulceration resulted solely from stress or acid hypersecretion.[1] This spiral, gram-negative, microaerophilic

organism colonizes the gastric mucus layer, persistently evading host defenses while provoking a chronic inflammatory reaction that may endure for decades in untreated carriers.[2] The International Agency for Research on Cancer has categorized *H. pylori* as a Group 1 carcinogen because of its established contribution to gastric adenocarcinoma and mucosa-associated lymphoid tissue (MALT) lymphoma.[3] The natural history of infection generally follows Correa's multistep cascade, in which chronic superficial gastritis advances through atrophy, intestinal metaplasia, and dysplasia toward invasive carcinoma a trajectory that can be interrupted by eradication of the organism.[4]

Although infection rates have fallen in industrialized countries, *H. pylori* remains among the most common chronic bacterial infections worldwide. Global estimates suggested that roughly 4.4 billion people were infected in 2015,[5] and pooled data from 2011–2022 indicate a worldwide prevalence of approximately 43%, with annual declines of 0.39–0.83% among recent birth cohorts reflecting gradual improvements in sanitation and living standards.[6] The distribution is strikingly uneven: prevalence surpasses 70% in parts of Africa and South Asia, whereas some high-income countries report rates below 19%.[5] Forecast models nevertheless project approximately 148 million diagnosed prevalent cases in the United States and EU4/UK alone by 2025, underscoring the persistent scale of the problem.[7]

Clinically, infected individuals span a broad symptomatic spectrum, from silent carriage to chronic epigastric pain, nausea, and complications such as bleeding or perforation.[8] Endoscopy may reveal erythema, erosions, nodularity, or ulceration, but definitive diagnosis and grading of gastritis depend on histopathological examination of mucosal biopsies, as codified by the updated Sydney system.[9] Management has been further complicated by rising antibiotic resistance, prompting the 2024 American College of Gastroenterology guideline to recommend 14-day bismuth quadruple therapy as first-line empiric treatment, with clarithromycin- or levofloxacin-based regimens reserved for confirmed susceptible isolates.[10]

North Africa carries a disproportionate share of this burden. In Libya, reported infection rates range from 76% to 90% across symptomatic and asymptomatic cohorts; however, most of these figures derive from serological surveys or stool antigen testing rather than systematic histopathological evaluation.[11,12] Such methods cannot distinguish active from past infection nor capture the morphological spectrum of mucosal injury from superficial inflammation to atrophy and intestinal metaplasia that ultimately determines malignancy risk. As international consensus increasingly endorses population-level screen-and-treat programs,[13] local histopathological benchmarks are needed to guide regional interventions. The present study was therefore designed to provide a contemporary histopathological audit of *H. pylori*–associated gastritis in western Libya, bridging the gap between international diagnostic standards and local clinical practice.

LITERATURE REVIEW

Microbiology and Virulence Determinants

H. pylori displays extraordinary genetic diversity, and disease outcome is strongly shaped by bacterial virulence factors. The cytotoxin-associated gene A (cagA) and the vacuolating cytotoxin A (vacA) are the most extensively characterized determinants. CagA-positive strains inject the CagA oncoprotein into gastric epithelial cells through a type IV secretion system, activating pro-inflammatory signaling, disrupting tight junctions, and disturbing the balance between cellular proliferation and apoptosis.[2] VacA promotes epithelial vacuolation, mitochondrial dysfunction, and immune evasion. Together, these attributes govern the intensity of the host inflammatory response and the probability of progression to severe gastric pathology.[14] The resulting chronic gastritis arises from complex interactions between bacterial pathogen-associated molecular patterns and host pattern recognition receptors, and may persist for the lifetime of the host if the organism is not eliminated.[15]

Epidemiology and Transmission Dynamics

The prevalence of H. pylori infection correlates inversely with socioeconomic

development. Regions with prevalence above 80% are concentrated in developing countries, where overcrowding, inadequate sanitation, and limited access to clean water favor early childhood transmission via fecal–oral or oral–oral routes.[16] Industrialized nations have witnessed steady seroprevalence declines attributable to improved living conditions and widespread antimicrobial use, yet infection remains significant among immigrant communities and older cohorts who acquired the bacterium in childhood.[5] This heterogeneous distribution argues for region-specific surveillance to inform targeted screening and eradication policy.

Antibiotic Resistance and Evolving Treatment Paradigms

Antibiotic resistance has become the principal obstacle to successful eradication, fundamentally reshaping management.[10] Although clarithromycin was removed from the World Health Organization's priority pathogen list in 2024, resistance continues to climb in most countries, exceeding 15% in 24 of 31 surveyed nations.[7] A meta-analysis of more than 47,000 isolates from 36 countries confirmed rising primary resistance between 2013 and 2023, with consistently higher rates in adults than in children, reflecting cumulative lifetime antibiotic exposure.[6] Compounding the problem, over one billion people lack access to bismuth-based regimens the most effective first-line option while susceptibility testing remains unavailable or unreimbursed in most surveyed countries.[7] Histopathological diagnosis should therefore be viewed as an integral component of antimicrobial stewardship, informing the choice between empiric and tailored therapy.[10]

Histopathological Classification and Grading

Histological assessment of gastritis encompasses chronic inflammation, neutrophilic activity, atrophy, intestinal metaplasia, and bacterial density, each graded as mild, moderate, or severe alongside a topographic designation (antrum, corpus, or pangastritis) under the Sydney system.[9,17] On routine H&E staining, the organisms appear as curved or spiral bacilli within the superficial mucus; ancillary stains such as Giemsa or immunohistochemistry improve detection when bacterial load is low or metaplasia is extensive.[18] Characteristic accompanying features include foveolar hyperplasia, dense lamina propria inflammation, lymphoid follicle formation, and surface epithelial degeneration. A recent systematic grading study confirmed that all H. pylori-positive biopsies exhibited chronic inflammation and neutrophilic activity, with moderate severity in 44.2% and marked severity in 23.2%; intestinal metaplasia was present in 44.2% of positive cases,[19] reaffirming the Sydney system as an indispensable framework for standardized reporting.

Clinical and Endoscopic Correlates

Infected patients typically present with dyspepsia chronic or recurrent epigastric pain, postprandial fullness, and early satiety while endoscopic findings range from mucosal erythema, edema, and friability to erosions, nodularity, ulceration, and atrophy. Crucially, the correlation between endoscopic appearance and histological severity is imperfect: subtle macroscopic findings may conceal severe active inflammation, and vice versa.[20] A cross-sectional study of 133 infected patients found significant endoscopy histopathology correlations for antral atrophic gastritis, antral intestinal metaplasia, and gastric polyps, yet concordance between Giemsa staining and rapid urease testing was only 21%.[21] The American Gastroenterological Association likewise emphasizes that intestinal metaplasia on histology almost invariably implies atrophic gastritis, and that close collaboration between gastroenterologists and pathologists is essential for consistent documentation.[22] Histopathology thus remains the gold standard for diagnosing infection, grading inflammation, and detecting premalignant lesions.

Association with Gastric Neoplasia

Long-standing infection confers a substantially elevated risk of gastric adenocarcinoma; a landmark endoscopic cohort study calculated a relative risk of approximately 5.9 among infected individuals, rising further with cagA positivity, smoking, and high dietary salt intake.[23] *H. pylori* is also implicated in more than 90% of gastric MALT lymphomas, and eradication frequently produces complete remission in early-stage

disease.[3] Large-scale cohort evidence reinforces this relationship: in a matched analysis of 25,317 infected and 25,317 uninfected individuals followed for ten years, cancer developed in 12.8% versus 11.8% (p = 0.002; hazard ratio 1.11), with non-eradicated infection significantly associated with subsequent cancer (HR 1.18) while eradicated status was not (HR 1.06).[7] Post-eradication mucosal changes, including map-like redness, may serve as endoscopic markers of advanced atrophy, incomplete intestinal metaplasia, and dysplasia.[24] Routine histopathological assessment of every gastric biopsy is therefore imperative not merely for organism detection but for comprehensive risk stratification and surveillance.

Rationale for the Present Study

Despite extensive global literature, localized histopathological data reflecting contemporary practice remain sparse. Routine diagnostic material spanning mild chronic inactive gastritis to severe active disease with ulceration and metaplastic transformation offers invaluable insight into disease burden and the clinicopathological interface.[25] Meaningful challenges persist in reproducible histopathological reporting: interobserver agreement for intestinal metaplasia is generally good to excellent, whereas glandular atrophy assessment remains more variable, and adjunctive tools such as TFF2 immunohistochemistry to clarify pseudopyloric metaplasia remain inaccessible in many resource-limited settings where the infection burden is paradoxically highest.[26,27] Modern practice has accordingly shifted from simply documenting infection toward comprehensive risk stratification using OLGA and OLGIM staging, recognizing that cumulative morphological sequelae rather than mere organism presence drive neoplastic progression.[24] Western Libya characterized by high infection rates, limited access to susceptibility-guided therapy, and a predominantly young adult population represents a setting where systematic histopathological characterization could substantially influence empiric treatment and resource allocation. The present study evaluates the demographic, endoscopic, and histological correlates of H. pylori–associated gastritis in a contemporary biopsy cohort to establish an evidence base that aligns international standards with local clinical realities.

MATERIALS AND METHODS

Study Design and Setting

This investigation employed a retrospective, descriptive, cross-sectional design conducted in the histopathology department. All specimens received between January 2025 and June 2026 were eligible for inclusion; specimens originated from private laboratories located in the western region of Libya. The protocol adhered to the ethical principles of the Declaration of Helsinki, and informed consent was waived given the retrospective, anonymized nature of the data.

Inclusion and Exclusion Criteria

Inclusion criteria were: (i) all histopathological specimens submitted during the study period; (ii) complete demographic and clinical data (age, sex, specimen type, clinical diagnosis, and histological report); and (iii) gastric biopsy specimens containing adequate mucosal tissue for histological evaluation. Exclusion criteria were: (i) specimens with insufficient clinical information or illegible records; and (ii) gastric biopsies with extensive autolysis or crush artifact precluding reliable interpretation.

Data Collection

A structured extraction form was used to retrieve the following variables from laboratory information systems and histopathology reports: demographic variables (age in years, sex); temporal variable (year of specimen reception, 2025 or 2026); specimen characteristics (gastric, duodenal, or colorectal biopsy; gall bladder; appendix; skin lesion; fluid cytology; etc.); clinical data (presenting symptoms and endoscopic findings as documented by the referring clinician); and the histopathological diagnosis, including H. pylori status, inflammation grade (mild, moderate, severe), activity, and associated features (erosion, ulceration, intestinal metaplasia, atrophy).

Histopathological Evaluation

All gastric biopsies were formalin-fixed, paraffin-embedded, sectioned at 4–5 µm, and stained with hematoxylin and eosin (H&E). H. pylori organisms were identified morphologically as S-shaped or curved bacilli lying within the superficial mucus layer or along the apical surface of foveolar epithelial cells. Gastritis severity was categorized according to the density of the inflammatory infiltrate and neutrophilic activity as mild, moderate, or severe chronic active gastritis. Associated histological features including surface erosion, ulceration, atrophy, and intestinal metaplasia were documented when present.

Statistical Analysis

Data were compiled and analyzed using Python (version 3.12) with the pandas, NumPy, SciPy, and statsmodels libraries. Categorical variables were expressed as frequencies and percentages; continuous variables were summarized as means with standard deviations (SD) or medians with ranges, depending on distributional characteristics assessed by the Shapiro–Wilk test. Group comparisons for categorical variables employed the chi-square test or Fisher's exact test where appropriate; non-normally distributed continuous variables were compared using the Mann–Whitney U test. The prevalence of H. pylori in gastric biopsies was calculated with 95% confidence intervals (CI) using the Wilson score method. A two-tailed p-value < 0.05 was considered statistically significant.

RESULTS

General Characteristics of the Study Population

A total of 136 histopathological specimens were analyzed. Most (n = 104, 76.5%) were received in 2025, with the remainder (n = 32, 23.5%) in 2026. The cohort comprised 81 females (59.6%) and 55 males (40.4%), with a mean age of 42.0 ± 18.6 years (median 41.0; range 0–79). Gastric biopsies formed the largest single category at 47.8% (n = 65), followed by fluid cytology (14.0%), colorectal biopsies (8.8%), skin lesions (5.9%), and gall bladder specimens (4.4%). These characteristics are summarized in Tables 1 and 2 and illustrated in Figures 1A and 1B.

Table 1: Demographic and Clinical Characteristics of All Cases (N = 136).

Characteristic	N (%) or Value
Total cases	136 (100.0)
Year 2025	104 (76.5)
Year 2026	32 (23.5)
Female	81 (59.6)
Male	55 (40.4)
Mean age (years) ± SD	42.0 ± 18.6
Median age (years)	41.0
Age range (years)	0–79
Gastric biopsy	65 (47.8)
Non-gastric specimens	71 (52.2)

Table 2: Breakdown of Specimen Types (N = 136).

Specimen Type	Frequency	Percentage
Gastric biopsy	65	47.8
Fluid cytology	19	14.0
Colorectal biopsy	12	8.8
Skin lesion	8	5.9
Gall bladder	6	4.4
Other	5	3.7
Gynecological	5	3.7
Appendix	5	3.7
Soft tissue	4	2.9
Prostate	2	1.5
Lung	2	1.5
Bone	1	0.7
Breast	1	0.7
Duodenal biopsy	1	0.7
Total	136	100.0

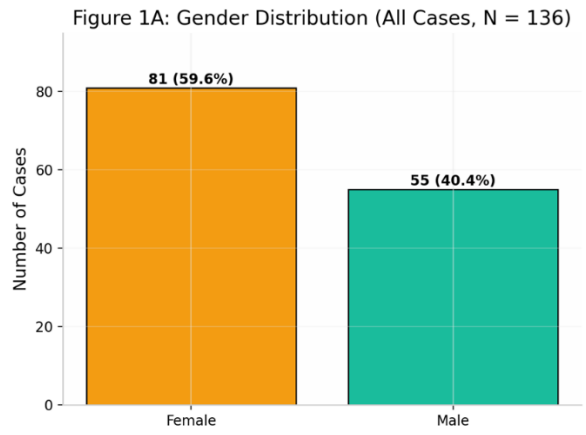


Fig. 1A: Gender distribution of all cases (N = 136).

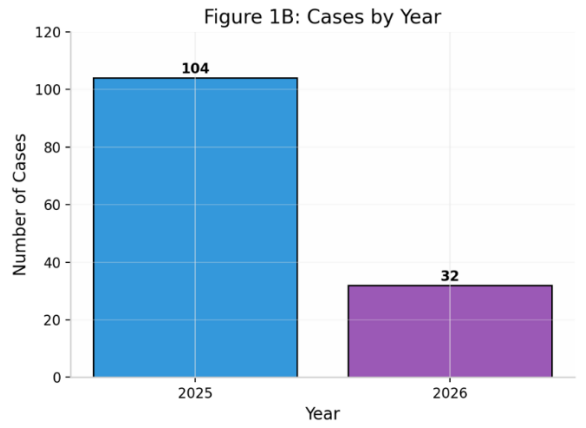


Fig. 2B: Gender distribution of all cases (N = 136).

Prevalence of H. pylori Infection

Among the 65 gastric biopsies, H. pylori was histologically identified in 53 cases, corresponding to an overall prevalence of 81.5% (95% CI: 70.4%–89.1%). The 12 organism-negative biopsies (18.5%) yielded alternative diagnoses including reactive (chemical) gastropathy, chronic atrophic gastritis with intestinal metaplasia, and mild chronic inactive gastritis. Across the entire cohort of 136 specimens, H. pylori positivity was observed in 39.0% of cases. Figure 2 depicts the comparative age distributions and the detection proportion within gastric biopsies.

Demographic Profile of H. pylori–Positive Patients

The 53 H. pylori–positive patients comprised 32 females (60.4%) and 21 males (39.6%). The mean age was 38.0 ± 15.1 years, with a median of 35.5 years (range 17–76). Age stratification showed that young adults (18–39 years) formed the largest subgroup (n = 16, 30.2%), closely followed by middle-aged adults (40–44 years, n = 15, 28.3%). Older adults (45–59 years) and elderly patients (≥60 years) accounted for 17.0% and 13.2%, respectively; a single pediatric case (<18 years) was identified, and five cases lacked documented age. Figure 3 illustrates gender distribution, severity grading, and age patterns.

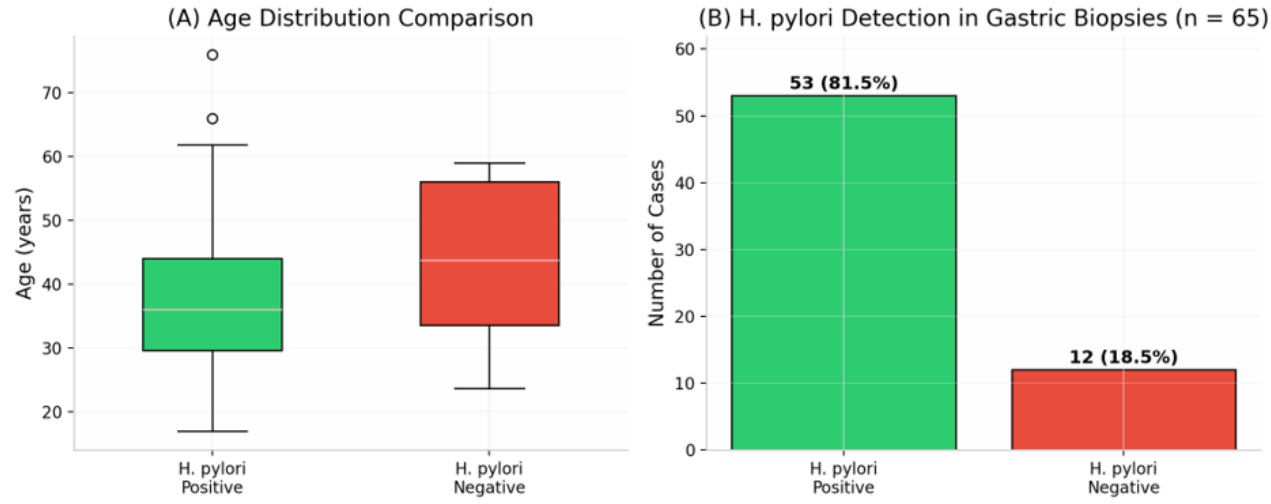


Fig. 2: Comparative analysis of gastric biopsy cases (n = 65). (A) Box plot comparing age distributions between H. pylori–positive and H. pylori–negative patients. (B) Absolute and relative frequencies of H. pylori detection within gastric biopsies.

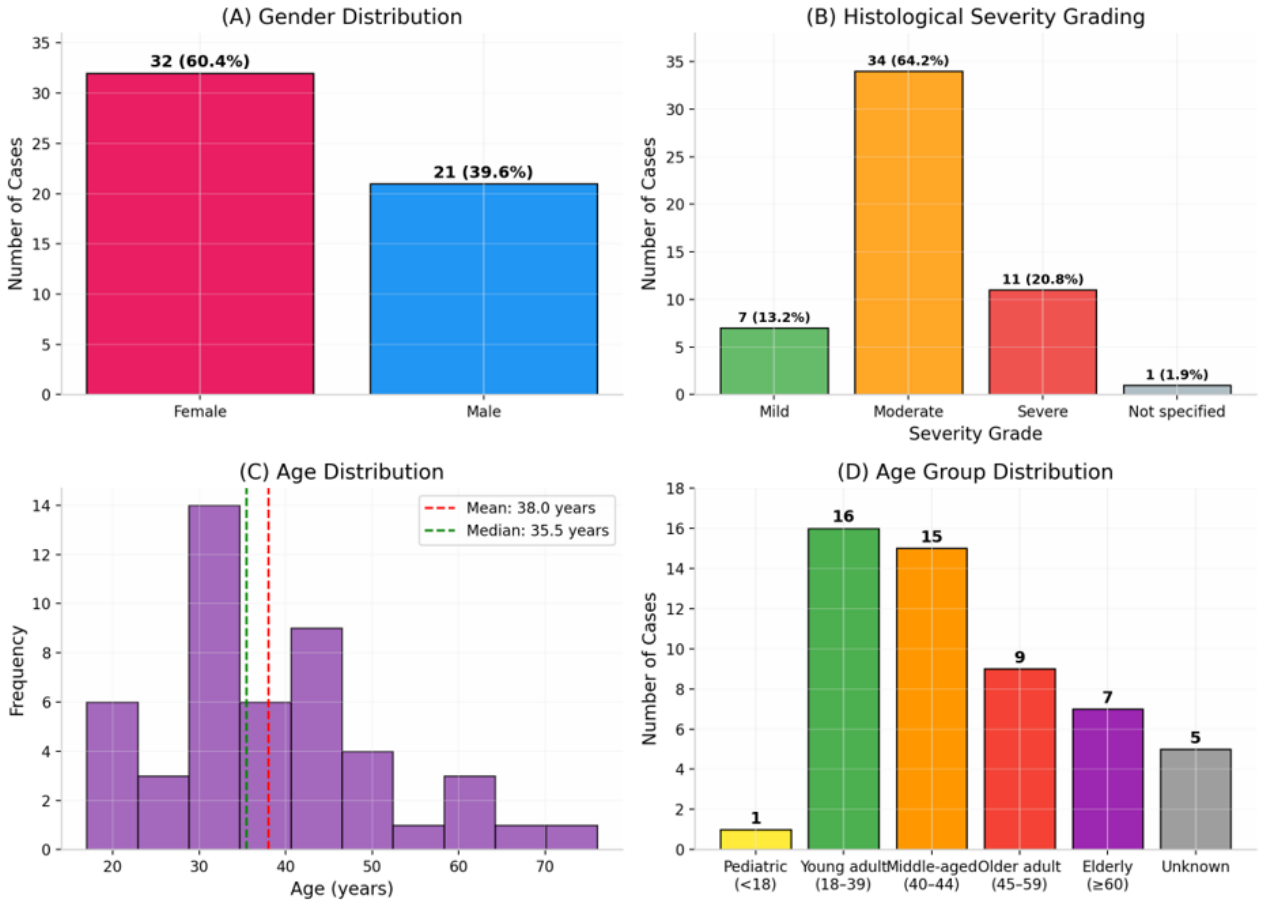


Fig. 3: Detailed analysis of H. pylori–associated gastritis cases (n = 53). (A) Gender distribution. (B) Histological severity grading. (C) Histogram of age distribution with superimposed mean and median values. (D) Age group distribution.

Histological Severity Grading

Histological severity was graded as moderate chronic active gastritis in 34 cases (64.2%), severe in 11 (20.8%), and mild in 7 (13.2%), as illustrated in Figure 3B; one case was reported without explicit severity grading (Figure 4). Notably, severe disease occurred across the adult age spectrum, including patients as young as 18 years, indicating that chronological age does not reliably predict histological severity. Associated histological features comprised superficial ulceration or surface erosion in six cases, reactive/chemical gastropathy in two, and focal intestinal metaplasia with reactive atypia in one.

Endoscopic Findings

Clinical and endoscopic data were available for the majority of H. pylori–positive patients. Pangastritis was the most frequently documented pattern (n = 21, 39.6%), followed by non-specific or unremarkable descriptions (n = 16, 30.2%). Erythematous gastritis and erosive gastritis each accounted for five cases (9.4%), while ulcerative lesions and atrophic gastritis were less common (3.8% each); a cobblestone mucosal appearance was observed in a single case. Table 4 summarizes these endoscopic correlates.

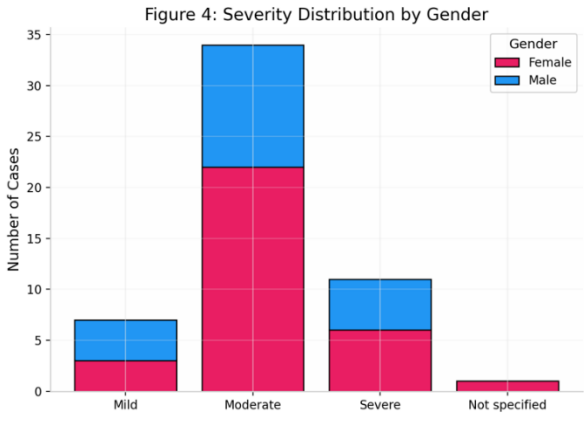


Fig. 4: Distribution of histological severity by gender (n = 53).

Table 4: Endoscopic Findings in H. pylori–Positive Cases (N = 53).

Endoscopic Finding	Frequency	Percentage
Pangastritis	21	39.6
Non-specific/Not described	16	30.2
Erythematous gastritis	5	9.4
Erosive gastritis	5	9.4
Ulcerative lesions	2	3.8
Atrophic gastritis	2	3.8
Cobblestone appearance	1	1.9
Not provided	1	1.9

Non–H. pylori Gastric Pathology

Among the 12 H. pylori–negative gastric biopsies, histological diagnoses included reactive (chemical) gastropathy negative for dysplasia or malignancy (n = 2), chronic atrophic gastritis with moderate intestinal metaplasia (n = 1), mild chronic inactive gastritis with vascular ectasia (n = 1), and benign peptic ulcer disease (n = 1). The remaining cases showed non-specific inflammatory changes or had inadequate clinical documentation.

Statistical Associations

No statistically significant association was found between sex and H. pylori positivity across the entire cohort ($\chi^2 = 0.000$, $p = 1.000$). The approximately 3:2 female-to-male ratio among infected patients mirrors the overall institutional patient demographics rather than a biological predisposition. Likewise, ages did not differ significantly between H. pylori–positive (mean 37.96 ± 15.12 years) and H. pylori–negative gastric biopsy patients (mean 42.67 ± 15.89 years; Mann–Whitney U = 235.000, $p = 0.331$). Finally, the distribution of histological severity did not differ significantly between sexes ($\chi^2 = 1.300$, $p = 0.522$), both showing a predominance of moderate-grade inflammation. Table 5 summarizes all statistical tests.

Table 5: Summary of Statistical Tests .

Comparison	Test	Statistic	p-value	Interpretation
Gender vs. H. pylori status (all cases)	Chi-square	$\chi^2 = 0.000$	1.000	Not significant
Age: H. pylori–positive vs. negative (gastric biopsies)	Mann–Whitney U	U = 235.000	0.331	Not significant
Severity by gender (H. pylori–positive)	Chi-square	$\chi^2 = 1.300$	0.522	Not significant
H. pylori prevalence in gastric biopsies (95% CI)	Wilson method	81.5%	70.4%–89.1%	High prevalence

DISCUSSION

This retrospective review of 136 histopathological specimens demonstrates that H. pylori infection is extraordinarily prevalent among patients referred for gastric biopsy, with more than four-fifths of such specimens showing histologically confirmed organisms. This finding is consistent with the established position of H. pylori as the leading cause of chronic gastritis and confirms the pathogen's continued dominance in contemporary gastroenterological practice.[8,28]

The demographic profile of infected individuals a mean age of 38 years with slight female predominance resembles patterns reported in several regional studies, although it differs from endemic settings where acquisition typically occurs in early childhood and a male predominance is frequently described.[5,16] The absence of significant associations between

infection and either gender or age suggests that, within this biopsy-selected population, the bacterium acts indiscriminately. This observation likely reflects selection bias, since patients undergoing endoscopy are inherently symptomatic, thereby masking demographic risk differentials that would be evident in community-based seroprevalence surveys.[29]

The striking predominance of moderate chronic active gastritis (64.2%) is particularly noteworthy. This pattern denotes a vigorous ongoing inflammatory process combining chronic lymphoplasmacytic infiltrates with active neutrophilic epithelial injury. The sizeable proportion of severe disease (20.8%) further indicates that many patients present with advanced mucosal damage, plausibly attributable to high-virulence strains, prolonged infection, or host factors such as impaired immune regulation.[2,14] The identification of surface erosion, ulceration, and intestinal metaplasia in a minority of cases serves as a histological reminder of the progressive potential of untreated infection, consistent with the early stages of Correa's cascade.[4]

Endoscopically, pangastritis emerged as the commonest macroscopic pattern, documented in nearly 40% of cases. This diffuse involvement of antral and corpus mucosa carries particular clinical weight, as pangastritis has been linked to higher risks of ulceration, atrophic transformation, and neoplastic progression compared with antrum-predominant disease.[17,23] Equally important is the substantial fraction of cases (30.2%) with non-specific endoscopic descriptions: H. pylori can inflict significant histological damage without dramatic macroscopic abnormalities. This discordance reinforces the guideline recommendation that all patients undergoing upper gastrointestinal endoscopy should have biopsies submitted for histopathological examination regardless of endoscopic appearance.[18]

The 81.5% prevalence observed here far exceeds the estimated global prevalence of approximately 43% but is consistent with the enrichment expected in a symptomatic, biopsy-referred cohort.[5,6] Patients referred for endoscopy typically have refractory dyspepsia, alarm symptoms, or documented ulcer disease—clinical scenarios in which infection prevalence predictably exceeds that of the general population. Nevertheless, the near-ubiquity of the organism in this biopsy population emphasizes the need for a high index of suspicion and adequate sampling, including both antral and corpus biopsies to optimize detection.[9]

Several limitations must be acknowledged. The retrospective design precluded standardized data collection, leaving clinical documentation incomplete for a subset of cases particularly precise endoscopic descriptors and risk factors such as prior antibiotic exposure or proton pump inhibitor use. The exclusive reliance on H&E staining, without special stains or immunohistochemistry, may have underestimated true prevalence in low-density or extensively metaplastic cases. Moreover, the 18-month study window limited assessment of temporal trends and seasonal variation. Future prospective studies incorporating molecular virulence profiling, comprehensive endoscopic–histological correlation, and long-term follow-up would substantially deepen understanding of disease trajectories in this setting.

From a public health standpoint, these findings support strengthened screening and eradication programs, particularly among young and middle-aged adults who constitute the bulk of infected individuals. Early intervention in this demographic may interrupt the Correa cascade, reducing the incidence of atrophic gastritis, intestinal metaplasia, and ultimately gastric cancer.[3,13,23] Histopathology laboratories are central to this effort, providing accurate diagnosis, severity grading, and identification of preneoplastic changes that warrant surveillance.

CONCLUSION

This retrospective histopathological study demonstrates that Helicobacter pylori infection remains extraordinarily prevalent among patients undergoing gastric biopsy in western Libya, with histologically confirmed organisms

identified in over 80% of such specimens. Infection most commonly manifests as moderate chronic active gastritis and is frequently accompanied by pangastritis on endoscopy. Neither gender nor age emerged as a significant determinant of infection status or histological severity, underscoring the pervasive, indiscriminate nature of this pathogen. These findings highlight the critical role of routine histopathological evaluation in upper gastrointestinal endoscopy not only for confirming H. pylori infection but also for stratifying disease severity and identifying preneoplastic mucosal change. Integrating these insights into clinical practice will facilitate targeted eradication therapy, reduce the risk of peptic ulcer complications, and contribute to gastric cancer prevention.

AUTHOR CONTRIBUTIONS

Mayadah A. Rahoumah: Writing – original draft, literature review, investigation, and manuscript preparation. **Ibrahim O. Eswaey:** Data curation, investigation, and manuscript preparation. **Abdurraouf Zaet:** Conceptualization, methodology, supervision, validation, critical review, writing – review & editing, and final approval of the manuscript.

CONFLICT OF INTEREST

The authors declare no conflicts of interest concerning the research, authorship, and publication of this article.

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REFERENCES

1. Marshall B., Warren JR. Unidentified curved bacilli in the stomach of patients with gastritis and peptic ulceration. Lancet (London, England), 1984;1:1311–1315. https://doi.org/10.1016/s0140-6736(84)91816-6
2. Kusters JG, van Vliet AH, Kuipers EJ. Pathogenesis of Helicobacter pylori infection. Clinical microbiology reviews, 2006; 19:449–490. https://doi.org/10.1128/CMR.00054-05
3. IARC Working Group on the Evaluation of Carcinogenic Risks to Humans. Schistosomes, liver flukes and Helicobacter pylori. Lyon: International Agency for Research on Cancer; 1994. (IARC Monographs, Vol. 61).
4. Correa P. Human gastric carcinogenesis: a multistep and multifactorial process-First American Cancer Society Award Lecture on Cancer Epidemiology and Prevention. Cancer Res. 1992;52(24):6735-6740.
5. Hooi JKY, Lai WY, Ng WK, Suen MMY, Underwood FE, et al. Global Prevalence of Helicobacter pylori Infection: Systematic Review and Meta-Analysis. Gastroenterology, 2017;*153*:420-429. https://doi.org/10.1053/j.gastro.2017.04.022
6. Yu Y, Xue J, Lin F, Liu D, Zhang W, Ru S, et al. Global Primary Antibiotic Resistance Rate of Helicobacter pylori in Recent 10 years: A Systematic Review and Meta-Analysis. Helicobacter, 2024;29:e13103. https://doi.org/10.1111/hel.13103
7. Schulz C, Liou JM, Alboraie M, Bornschein J, Campos Nunez C, Coelho LG, et al. Helicobacter pylori antibiotic resistance: a global challenge in search of solutions. Gut, 2025;74:1561–1570. https://doi.org/10.1136/gutjnl-2025-335523
8. Suerbaum S, Michetti P. Helicobacter pylori infection. N Engl J Med. 2002;347:1175-86. https://doi.org/10.1056/NEJMra020542.
9. Dixon MF, Genta RM, Yardley JH, Correa P. Classification and grading of gastritis. The updated Sydney System. International Workshop on the Histopathology of Gastritis, Houston 1994. Am J Surg Pathol, 1996; *20*:1161–1181. https://doi.org/10.1097/00000478-199610000-00001
10. Chey WD, Howden CW, Moss SF, Morgan DR, Greer KB, Grover S, et al. ACG Clinical Guideline: Treatment of Helicobacter pylori Infection. Am J Gastroenterol., 2024;119: 1730–1753. https://doi.org/10.14309/ajg.0000000000002968
11. Bakka AS, Salih BA. Prevalence of Helicobacter pylori infection in asymptomatic subjects in Libya. Diagn Microbiol Infect Dis. 2002; 43:265–268. doi: 10.1016/S0732-8893(02)00411-X.

12. Alsulaimany FAS, Awan ZA, Almohamady AM, Koumu MI, Yaghmoor BE, Elhady SS, et al. Prevalence of *Helicobacter pylori* Infection and Diagnostic Methods in the Middle East and North Africa Region. Medicina (Kaunas, Lithuania), 2020;56:169. https://doi.org/10.3390/medicina56040169

13. Park JY, Lee YC, Moayyedi P, Lansdorp-Vogelaar I, Camargo MC, Tepeš B, et al. *Helicobacter pylori* Screen-and-Treat Programs for Gastric Cancer Prevention - IARC Working Group Report. N Engl J Med. 2026;394:1131–1137. https://doi.org/10.1056/NEJMsb2515372

14. Peek RM Jr, Blaser MJ. Helicobacter pylori and gastrointestinal tract adenocarcinomas. Nat Rev Cancer. 2002;2:28-37. https://doi.org/10.1038/nrc703

15. Robinson K, Argent RH, Atherton JC. The inflammatory and immune response to Helicobacter pylori infection. Best Pract Res Clin Gastroenterol. 2007;21:237–259. https://doi.org/10.1016/j.bpg.2007.01.001

16. Fock KM, Ang TL. Epidemiology of Helicobacter pylori infection and gastric cancer in Asia. J Gastroenterol Hepatol. 2010;25:479–486. https://doi.org/10.1111/j.1440-1746.2009.06188.x

17. Rugge M, Correa P, Dixon MF, Fiocca R, Hattori T, Lechago J, et al. Gastric mucosal atrophy: interobserver consistency using new criteria for classification and grading‡. Aliment Pharmacol Ther.2002;16:12491259. https://doi.org/10.1046/j.1365-2036.2002.01301.x

18. McColl KE. Clinical practice. Helicobacter pylori infection. N Engl J Med. 2010;362:1597–1604. https://doi.org/10.1056/NEJMcp1001110

19. Muraleedharan A, Asokan CA, Muraleedharan D, Sadasivan S. *Helicobacter pylori* footprint: A systematic grading of histopathological changes in gastric mucosa. Ann Med Med Sci. 2026;5:389–392.

20. Malfertheiner P, Megraud F, O'Morain CA, Gisbert JP, Kuipers EJ, Axon AT, et al. European Helicobacter and Microbiota Study Group and Consensus panel. Management of Helicobacter pylori infection-the Maastricht V/Florence Consensus Report. Gut, 2017;66:6–30. https://doi.org/10.1136/gutjnl-2016-312288

21. Dănilă C, Cardos IA, Pop-Crisan A, Marc F, Hoza A, Chirla R, et al. Correlations between Endoscopic and Histopathological Assessment of Helicobacter pylori-Induced Gastric Pathology-A Cross-Sectional Retrospective Study. Life (Basel, Switzerland), 2022;12:2096. https://doi.org/10.3390/life12122096

22. Shah SC, Piazuelo MB, Kuipers EJ, Li D. AGA Clinical Practice Update on the Diagnosis and Management of Atrophic Gastritis: Expert Review. Gastroenterology, 2021;161:1325–1332.e7. https://doi.org/10.1053/j.gastro.2021.06.078

23. Uemura N, Okamoto S, Yamamoto S, Matsumura N, Yamaguchi S, Yamakido, M, et al. Helicobacter pylori infection and the development of gastric cancer. N Engl J Med. 2001;*345*:784–789. https://doi.org/10.1056/NEJMoa001999

24. You Y, Zheng J, Lin W, Yu S, Gao C, Zheng, Y, et al. Histopathological features of gastric map-like redness under image-enhanced endoscopy. Front Med. 2026;13:1833606. https://doi.org/10.3389/fmed.2026.1833606

25. Chaudhari MA, Ajaybhai SC, Chahare WV, Lazarus NS. Histopathological spectrum of gastric biopsies in *Helicobacter pylori*-associated chronic gastritis: A tertiary care analysis. Healthcare Bulletin. 2025. https://healthcare-bulletin.co.uk/article/histopathological-spectrum-of-gastric-biopsies-in-helicobacter-pylori-associated-chronic-gastritis-a-tertiary-care-analysis-3721/

26. Rugge M, Sacchi D, Genta RM, Zanco F, Guzzinati S, Pizzi M, et al. Histological assessment of gastric pseudopyloric metaplasia: Intra- and inter-observer consistency. Digestive and liver disease : official journal of the Italian Society of Gastroenterology and the Italian Association for the Study of the Liver, 2021;53:61-65. https://doi.org/10.1016/j.dld.2020.09.003

27. Lerch JM, Pai RK., Brown I, Gill AJ, Jain D, Kovari B, et al. Interobserver agreement of estimating the extent of intestinal metaplasia in patients with chronic atrophic

gastritis. Virchows Arch. 2022;480:1277–1281. https://doi.org/10.1007/s00428-021-03245-9

28. Malfertheiner P, Megraud F, Rokkas T, Gisbert JP, Liou JM, Schulz C, et al. Management of Helicobacter pylori infection: the Maastricht VI/Florence consensus report. Gut, 2022; 71:1724–1762. https://doi.org/10.1136/gutjnl-2022-327745

29. Izeit H, Alghoj M, Al-Gadhi A, Zaet AA. Rediscovering Helicobacter pylori in Pediatric and Young Populations through Histopathological Evidence. Open J Biomed Res.2025;4:24-29. https://doi.org/10.26629/ojbr.2025.06

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