https://doi.org/10.54361/ajmas.269318

Clinicopathological Characteristics of Gastritis Associated with *Helicobacter pylori*: A Two-Year Retrospective Analysis

Abdurraouf Zaet[1,2,3*], Mohamed Alghoj[4], Ibrahim Eswaey[3,5], Nuri Ahwij[3,6], Hanan Alwuheeshi[7], Altayeb Elazomi[2,3,8]

[1]*Department of Dental Technology, Faculty of Medical Technology, University of Zawia, Libya.*
[2]*Biomedical Research Team, University of Zawia, Libya*
[3] *Libyan Society for Biomedical Research, Libya*
[4]*Department of Diabetes and Endocrinology, Faculty of Medicine, University of Zawia, Libya*
[5]*Department of Medical Laboratories, Faculty of Medical Technology, Sabratha University, Libya*
[6]*Department of Clinical Hematology, University Hospital, F-21000 Dijon, France*
[7]*Department of Botany, Faculty of Science, Sabratha University, Libya*
[8]*Department of Medical Laboratories, Faculty of Medical Technology, University of Zawia, Libya.*
Corresponding Email: *a.zaet@zu.edu.ly*

Abstract
Helicobacter pylori infection continues to represent one of the most prevalent chronic bacterial infections on a global scale and constitutes a principal etiological factor for gastritis, peptic ulcer disease, and gastric malignancies. The present investigation sought to delineate the clinicopathological attributes of *H. pylori*-related gastritis among individuals subjected to endoscopic assessment. A retrospective review was performed on 82 histologically verified cases of *H. pylori* infection identified from gastric mucosal biopsies collected over a 24-month interval (2023-2024) at private healthcare facilities situated in the western region of Libya. Variables including demographic parameters, clinical manifestations, endoscopic observations, and histopathological severity were evaluated employing standardized histopathological criteria. The investigation encompassed 82 participants with a mean chronological age of 39.3 ± 18.0 years (spanning 13-77 years). Female subjects constituted the majority (62.2% versus 37.8%). The preponderance of cases (78.0%) exhibited chronic active gastritis, with moderate severity representing the most frequent finding (46.3%), followed by severe (29.3%) and mild (13.4%) presentations, while 11.0% demonstrated unspecified or chronic non-active gastritis. Epigastric discomfort emerged as the principal presenting manifestation (63.4%). Concomitant pathological discoveries encompassed duodenitis (12.2%), ulcerative or erosive lesions (6.1%), and intestinal metaplasia (3.7%). These findings indicate a substantial prevalence of active *H. pylori* infection across diverse age demographics, with a marked female preponderance. The considerable proportion of moderate to severe active gastritis emphasizes the necessity of timely diagnostic evaluation and eradication therapy to avert progression toward more severe sequelae, including gastric carcinoma.
Keywords: *Helicobacter pylori*, Gastritis, Histopathology, Endoscopy, Epidemiology.

Introduction

Helicobacter pylori constitutes a spiral-shaped, gram-negative microorganism that establishes colonization within the gastric mucosa of roughly half of the global human population [1]. Initially cultivated by Marshall and Warren in 1982 [2], this pathogen has been conclusively associated with chronic active gastritis, peptic ulcer disease, gastric adenocarcinoma, and mucosa-associated lymphoid tissue (MALT) lymphoma [3]. The distinctive capacity of this bacterium to endure within the acidic gastric milieu through urease synthesis, combined with its flagellar motility, facilitates persistent colonization and sustained inflammatory responses [4]. The infection encourages cellular and humoral immune responses in most patients. Specific serum antibody measurements are used as a non-invasive method to detect infection of *H. pylori* [5].

Notwithstanding progress in diagnostic methodologies and therapeutic eradication regimens, *H. pylori* infection continues to pose a considerable public health challenge, especially in developing nations where prevalence estimates may surpass 80% [6]. The clinical spectrum of *H. pylori* infection extends from asymptomatic carriage to grave complications, including gastric malignancy, following the extensively documented Correa cascade of intestinal metaplasia and dysplasia [7]. Comprehension of regional epidemiological distributions and clinicopathological features is imperative for the formulation of targeted screening and therapeutic protocols [8].

This retrospective investigation aims to examine the demographic characteristics, clinical presentations, endoscopic findings, and histopathological severity of *H. pylori*-associated gastritis in patients presenting to our healthcare facility throughout a two-year observation period.

Methods

Study Design and Population

This retrospective observational investigation analyzed histopathological records from individuals who underwent upper gastrointestinal endoscopy with gastric mucosal sampling between January 2023 and December 2024 at private healthcare facilities in the western region. Cases with histopathological confirmation of *H. pylori* infection were incorporated into the analytical dataset.

Received*: 07-01-2026 -* ***Accepted****: 06-03-2026 -* ***Published****: 12-03-2026*

Data Collection
Information was extracted from pathology reports, comprising demographic variables (age and sex), clinical manifestations and symptomatology, endoscopic observations, specimen classification and anatomical location, as well as histopathological diagnosis and severity stratification.

Histopathological Evaluation
Gastric mucosal specimens were processed utilizing conventional histological methodologies and stained with Hematoxylin and Eosin (H&E). The presence of *H. Pylori* organisms was verified through characteristic morphological features and tissue reaction patterns. Inflammatory changes were graded according to the Updated Sydney System as mild, moderate, or severe based upon the density of inflammatory infiltrate and activity [9].

Statistical Analysis
Descriptive statistical measures were computed for all variables. Categorical variables were expressed as frequencies and percentages, whereas continuous variables were presented as mean ± standard deviation or median with range. Data analysis was conducted utilizing Python statistical packages.

Results
Demographic Characteristics
A total of 82 histologically confirmed cases of *H. pylori* infection were identified during the study interval. The demographic distribution is summarized in Table 1. The age distribution demonstrated a mean age of 39.3 years with modest rightward skewness. The majority of participants (36.6%) were categorized within the middle-aged group (30-49 years), followed by young adults (26.8%). Notably, 7.3% of cases occurred in pediatric subjects (<18 years), with the youngest participant being 13 years of age.

Table 1: Demographic and Clinical Characteristics of H. pylori Cases

Parameter	Value
Total Cases	82 (100%)
Study Period	2023-2024
Mean Age (years)	39.3 ± 18.0
Age Range (years)	13 - 77
Female Patients	51 (62.2%)
Male Patients	31 (37.8%)
Pediatric Cases (<18y)	6 (7.3%)
Adult Cases (≥18y)	76 (92.7%)
Cases in 2023	34 (41.5%)
Cases in 2024	48 (58.5%)

Gender Distribution
Female participants substantially outnumbered male subjects, comprising 62.2% (n=51) of all cases compared to 37.8% (n=31) males. This female predominance was consistent across both study years and the majority of severity grades.

Histopathological Severity
The distribution of gastritis severity revealed that moderate chronic active gastritis represented the most prevalent presentation, accounting for 46.3% (n=38) of cases, followed by severe chronic active gastritis at 29.3% (n=24), mild chronic gastritis at 13.4% (n=11), and unspecified or chronic non-active gastritis at 11.0% (n=9).

A notable 78.0% of cases demonstrated active inflammatory changes, characterized by neutrophilic infiltration of the gastric mucosa, indicating ongoing mucosal injury and bacterial activity [9].

Clinical Presentations
Epigastric or abdominal discomfort constituted the predominant presenting complaint, documented in 63.4% (n=52) of cases. Additional clinical manifestations are included in Table 2.

Table 2: Clinical Presentations of H. pylori Cases

Symptom	Cases (%)
Epigastric/Abdominal Pain	52 (63.4%)
Anemia	5 (6.1%)
Vomiting	5 (6.1%)
GI Bleeding/Melena	3 (3.7%)
Weight Loss	1 (1.2%)
Dysphagia	1 (1.2%)

Received*: 07-01-2026 -* ***Accepted****: 06-03-2026 -* ***Published****: 12-03-2026*

Endoscopic Findings
Endoscopic examination revealed diverse mucosal abnormalities, with generalized gastritis representing the most prevalent observation at 54.9% (n=45), followed by mucosal congestion or erythema in 22.0% (n=18) of cases, ulcerative lesions in 6.1% (n=5), erosive gastritis in 4.9% (n=4), and gastric polyps in 2.4% (n=2).

Associated Pathological Findings
Concurrent pathological discoveries in *H. pylori*-positive individuals encompassed duodenitis in 12.2% (n=10), frequently with preserved villous architecture, ulceration or erosion in 6.1% (n=5), intestinal metaplasia (a premalignant alteration [6]) in 3.7% (n=3), gastric polyps in 3.7% (n=3), and reactive lymphoid hyperplasia in 1.2% (n=1).

Temporal Trends
The quantity of diagnosed cases increased from 34 cases in 2023 to 48 cases in 2024, representing a 41.2% increment. This elevation may reflect heightened awareness, enhanced diagnostic rates, or a genuine increase in prevalence.

Discussion
This retrospective evaluation of 82 histologically confirmed cases of *H. pylori* infection yields valuable insights into the regional epidemiology and clinicopathological spectrum of *H. pylori* infection. The mean age of 39.3 years corresponds with global patterns demonstrating peak *H. pylori* prevalence among middle-aged adults [10]. However, the substantial proportion of cases (36.6%) within the 30-49 age category suggests active transmission or acquisition during early adulthood. The presence of *H. pylori* in 7.3% of pediatric patients is concerning, as childhood infection is associated with an elevated risk of complications and suggests intrafamilial transmission [11]. Weyermann et al. [11] demonstrated that infected mothers, fathers, and siblings independently contribute to childhood acquisition, supporting family-based screening approaches. The female predominance (62.2%) observed in this investigation diverges from certain reports demonstrating equivalent or male-predominant distributions [10]. This may reflect disparities in healthcare-seeking behavior, as women tend to pursue medical evaluation for dyspeptic symptoms more readily than men, or could represent genuine biological differences in susceptibility or exposure patterns within our population.

The predominance of moderate to severe active gastritis (75.6% combined) carries clinical significance. Active inflammation, characterized by neutrophilic infiltration, signifies ongoing mucosal damage and is associated with elevated risks of ulcer formation and malignant transformation [12]. Rugge and Genta [12] demonstrated that the Sydney System grading of activity correlates with bacterial burden and risk of complications. Our finding that nearly half of patients exhibited moderate inflammation suggests substantial bacterial load and host immune response necessitating prompt intervention.

The elevated prevalence of epigastric discomfort (63.4%) as the presenting manifestation underscores the symptomatic burden of *H. pylori* infection. However, the diversity of presentations, including anemia (6.1%), which may reflect chronic occult blood loss, and vomiting (6.1%) highlights the protean manifestations of this infection described in the literature [3]. The identification of intestinal metaplasia in 3.7% of cases is particularly noteworthy. Intestinal metaplasia represents a critical step in the Correa cascade of gastric carcinogenesis [7], and its presence mandates eradication therapy and surveillance according to current guidelines [13]. The finding of reactive lymphoid hyperplasia in one case suggests MALT formation, which can progress to lymphoma if left untreated [3].

The co-occurrence of duodenitis in 12.2% of cases substantiates the established pathophysiology of *H. pylori* in duodenal ulcer disease described by Marshall and Warren [2]. Interestingly, the majority of duodenal biopsies demonstrated normal villous architecture, suggesting that the inflammation was secondary to *H. pylori* rather than celiac disease or other malabsorptive disorders.

The elevated proportion of active, moderate-to-severe gastritis identified in this cohort carries several important clinical implications. First, the predominance of active inflammation underscores the urgency of implementing aggressive eradication strategies to prevent progression along the Correa cascade toward atrophy, intestinal metaplasia, and dysplasia [7]. Second, the detection of a 7.3% prevalence among pediatric patients highlights the need for family-based screening and treatment programs to reduce intrafamilial transmission, in line with recommendations by Weyermann et al. [11]. Third, the presence of intestinal metaplasia and reactive lymphoid hyperplasia in a subset of patients emphasizes the necessity of surveillance endoscopy to confirm resolution following therapy [13]. Finally, the observed female predominance suggests that gender-specific approaches may be warranted in public health messaging and screening initiatives, although further research is required to determine whether this reflects true biological susceptibility or differences in healthcare-seeking behavior [10].

Limitations
This investigation possesses several limitations inherent to its retrospective design. The sample represents patients who underwent endoscopy and may not reflect the true community prevalence of *H. pylori* infection

Received: *07-01-2026* - ***Accepted***: *06-03-2026* - ***Published***: *12-03-2026*

[1]. Selection bias toward symptomatic patients likely overestimates the severity of disease in the general population. Additionally, the absence of data on socioeconomic status, hygiene practices, and family history limits the ability to identify risk factors described in the literature [11]. The two-year timeframe, while providing contemporary data, may not capture long-term trends or seasonal variations.

Conclusion

This investigation demonstrates a substantial burden of Helicobacter pylori-associated gastritis, with significant active inflammation observed across diverse age groups, consistent with global epidemiological patterns. The female predominance, the elevated rate of moderate-to-severe active disease, and the presence of premalignant changes in a subset of patients highlight the importance of systematic screening among dyspeptic patients, particularly women and middle-aged adults. Prompt eradication therapy is essential to halt the progression of mucosal damage and reduce the risk of gastric cancer. Surveillance protocols should be implemented for patients with intestinal metaplasia or severe active inflammation, while family-based approaches are warranted to address pediatric cases and potential transmission dynamics. Future prospective investigations should focus on evaluating eradication success rates, recurrence patterns, and the long-term impact of treatment on histological regression in this population, guided by the framework established by the Updated Sydney System.

Conflict of interest. Nil

References

1. Hooi JKY, Lai WY, Ng WK, Suen MMY, Underwood FE, Tanyingoh D, et al. Global Prevalence of Helicobacter pylori Infection: Systematic Review and Meta-Analysis. Gastroenterology. 2017 Aug;153(2):420-429. doi: 10.1053/j.gastro.2017.04.022.
2. Marshall BJ, Warren JR. Unidentified curved bacilli in the stomach of patients with gastritis and peptic ulceration. Lancet. 1984 Jun 16;1(8390):1311-1315. doi: 10.1016/s0140-6736(84)91816-6.
3. Peek RM Jr, Blaser MJ. Helicobacter pylori and gastrointestinal tract adenocarcinomas. Nat Rev Cancer. 2002 Jan;2(1):28-37. doi: 10.1038/nrc703.
4. Eaton KA, Brooks CL, Morgan DR, Krakowka S. Essential role of urease in pathogenesis of gastritis induced by Helicobacter pylori in gnotobiotic piglets. Infect Immun. 1991 Jul;59(7):2470-2475. doi: 10.1128/iai.59.7.2470-2475.1991.
5. Bakka A, Mohammad MA, Altayar M, Toboloi AB, Gariena A, Boushala M. Helicobacter pylori Infection among Libyan chronic dyspeptic patients in Benghazi. Libya J Inf Dis., 2009; 3: 30-36.
6. Eusebi LH, Zagari RM, Bazzoli F. Epidemiology of Helicobacter pylori infection. Helicobacter. 2014 Sep;19 Suppl 1:1-5. doi: 10.1111/hel.12165.
7. Correa P. Human gastric carcinogenesis: a multistep and multifactorial process--First American Cancer Society Award Lecture on Cancer Epidemiology and Prevention. Cancer Res. 1992 Dec 15;52(24):6735-6740.
8. Fock KM, Katelaris P, Sugano K, Ang TL, Hunt R, Talley NJ, et al; Second Asia-Pacific Conference. Second Asia-Pacific Consensus Guidelines for Helicobacter pylori infection. J Gastroenterol Hepatol. 2009 Oct;24(10):1587-1600. doi: 10.1111/j.1440-1746.2009.05982.x.
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 Oct;20(10):1161-1181. doi: 10.1097/00000478-199610000-00001.
10. Malaty HM, El-Kasabany A, Graham DY, Miller CC, Reddy SG, Srinivasan SR, et al. Age at acquisition of Helicobacter pylori infection: a follow-up study from infancy to adulthood. Lancet. 2002 Mar 16;359(9310):931-935. doi: 10.1016/S0140-6736(02)08025-X.
11. Weyermann M, Rothenbacher D, Brenner H. Acquisition of Helicobacter pylori infection in early childhood: independent contributions of infected mothers, fathers, and siblings. Am J Gastroenterol. 2009 Jan;104(1):182-189. doi: 10.1038/ajg.2008.61.
12. Rugge M, Genta RM. Staging and grading of chronic gastritis. Hum Pathol. 2005 Mar;36(3):228-233. doi: 10.1016/j.humpath.2004.12.008.
13. Malfertheiner P, Megraud F, Rokkas T, Gisbert JP, Liou JM, Schulz C, et al. European Helicobacter and Microbiota Study group. Management of Helicobacter pylori infection: the Maastricht VI/Florence consensus report. Gut. 2022 Aug 8:gutjnl-2022-327745. doi: 10.1136/gutjnl-2022-327745.

Received: *07-01-2026* - ***Accepted***: *06-03-2026* - ***Published***: *12-03-2026*