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Original Article

Intestinal Metaplasia and *Helicobacter pylori*: An Endoscopic Biopsy Study of Detection Rates and Associated Factors in a Libyan Population

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ABSTRACT

Background: Gastric intestinal metaplasia (GIM) is a recognized premalignant lesion in the Correa cascade, yet its prevalence and associated factors remain poorly characterized in Libya. This study aimed to estimate the endoscopic biopsy detection rate of GIM and examine its associations with *Helicobacter pylori* infection, age, and sex in a Libyan endoscopic cohort.

Methods: This retrospective cross-sectional study included 442 patients who underwent esophagogastroduodenoscopy with gastric biopsy of macroscopically abnormal mucosa at Tripoli Central Hospital between January 2022 and August 2023. Data on age, sex, GIM, *H. pylori* status, and chronic gastritis were collected. Associations were assessed using chi-square tests, odds ratios, and logistic regression.

Results: Of the 442 patients included, 272 (61.5%) were female, and 170 (38.5%) were male; the median age was 40 years (interquartile range, 24–53 years; range, 2–94 years). GIM was present in 28 patients (6.3%), *H. pylori* infection in 300 (67.9%), and chronic gastritis in 439 (99.3%). Age group was significantly associated with GIM (χ^2 = 14.7, *P* = 0.022), with the highest detection rate in the 51 to 60 years (12.7%) and 61 to 70 years (14.7%) age groups. In multivariable analysis, patients aged 51 to 60 years (adjusted odds ratio [aOR], 4.03 [95% CI, 1.04–15.60]; *P* = 0.044) and 61 to 70 years (aOR, 4.86 [95% CI, 1.09–21.80]; *P* = 0.039) had significantly higher odds of GIM compared with those aged ≤20 years. Neither sex nor *H. pylori* infection was independently associated with GIM. *H. pylori* positivity declined with increasing age.

Conclusions: In this biopsy-selected Libyan cohort—in which only patients with macroscopically abnormal mucosa underwent sampling—the GIM detection rate was 6.3%—a figure that likely underestimates the true disease burden given the non-systematic biopsy protocol. Increasing age was the strongest independent factor associated with GIM, with significantly elevated odds in adults aged 51 to 70 years (aOR, 4.03–4.86), while neither sex nor current *H. pylori* infection status emerged as an independent predictor. Larger population-based studies using standardized biopsy protocols are needed to establish accurate prevalence estimates and evaluate whether targeted surveillance of adults aged ≥50 years is warranted in Libya.

Key words: Gastric intestinal metaplasia, GIM, *Helicobacter pylori*, gastric cancer, Libya, identified rate

INTRODUCTION

Gastric intestinal metaplasia (GIM) is a premalignant condition in which the normal gastric mucosa is replaced by metaplastic epithelium of intestinal type. [1] Correa's cascade

describes the well-characterized sequential progression from normal mucosa through chronic active gastritis, atrophic gastritis, intestinal metaplasia, and dysplasia to gastric adenocarcinoma. [2] Global estimates of GIM prevalence range from approximately 16% to 18%, and its incidence rises progressively with age. [3,4]

Established risk factors for GIM include advancing age, male sex, *Helicobacter pylori* infection, bile reflux, and cigarette smoking. [5–10] Dietary factors also play a role: high consumption of salt, red and processed meats, and smoked or canned foods has been associated with increased risk, whereas higher intakes of vitamins C and E appear protective. [11,12] Among these factors, *H. pylori* infection is the most significant and modifiable contributor to GIM development. [13]

Gastric cancer (GC) ranks as the fifth most common malignant disease and is the third most prevalent underlying etiology of cancer-related mortality globally. [14] Mitigating GC mortality demands primary and secondary prevention by *H. pylori* eradication, endoscopic early detection, and precancerous abnormalities monitoring, such as GIM. [4] Current guidelines advocate for comprehensive mucosal visualization via high-definition white-light endoscopy with image enhancement and systematic biopsies adhering to the Updated Sydney System biopsy protocol (USSBP) for patients with supplementary GIM risk factors, significantly enhancing the identification of preneoplastic lesions. [1,15]

Despite this established global framework, the GIM epidemiology in Libya is not well defined. To our knowledge, no study has been published that assessed the GIM prevalence or its correlating factors in the Libyan community. This study aims to investigate the detection rate of GIM and explore its relationships with *H. pylori* infection, age, and gender. The study is a retrospective study of patients who presented to the endoscopy department at Tripoli Central Hospital (Medicine Department) for upper endoscopy. Patients who had macroscopic findings that were suggestive of any pathological changes were biopsied. This study aimed to estimate the endoscopic biopsy detection rate of GIM and examine its associations with *H. pylori* infection, age, and sex in a Libyan endoscopic cohort.

The results of this study can offer preliminary local data that might guide the design of future population-based research and facilitate the formulation of focused initiatives for the early detection and prevention of stomach cancer in Libya.

MATERIALS AND METHODS

Study Design and Setting

This retrospective cross-sectional study was conducted at Tripoli Central Hospital, Tripoli, Libya, using data from patients who underwent esophagogastroduodenoscopy (EGD) between January 2022 and August 2023.

Participants

During the 20-month study period, approximately 2900 patients underwent upper gastrointestinal endoscopy in our unit. Among these, 495 patients had macroscopic gastric abnormalities—including erythema, erosion, ulceration, mucosal atrophy, focal irregular lesions, or other suspicious mucosal changes—and were considered eligible for inclusion. After excluding 49 patients due to incomplete data (missing histological diagnosis, *H. pylori* result, or sex) and a further four patients with missing age data, a total of 442 patients were included in the final analysis. These exclusions were driven by missing administrative or histological fields rather than any clinical characteristic. While the age range was 2 to 94 years, reflecting the broad tertiary referral pattern, GIM is virtually nonexistent in early childhood, and the inclusion of the youngest patients does not skew the overall GIM estimates. Patients were referred to endoscopy for various upper gastrointestinal symptoms; a gastric mucosal biopsy was performed only in patients with macroscopic abnormalities identified at the time of the EGD procedure.

Biopsy Technique and Histopathological Assessment

Biopsy specimens were obtained from the abnormal-appearing gastric corpus and antrum mucosa and from additional sites when endoscopic abnormalities warranted further sampling. The macroscopic findings of a single endoscopist determined the number and anatomical distribution of biopsies. Specimens were not labeled or mapped according to the Updated Sydney System; this approach reflects routine clinical practice, which, while limiting site-specific risk stratification, remains informative for estimating the overall GIM detecting rate. [1]

All biopsy specimens were fixed immediately in 10% neutral-buffered formalin and processed without delay to minimize autolysis and preserve histological architecture. Specimens were embedded in paraffin and stained routinely with hematoxylin and eosin (H&E) for histopathological diagnosis and *H. pylori* detection. In cases where *H. pylori* status was uncertain on H&E, additional Giemsa staining was performed. *H. pylori* was considered absent only when both staining methods were negative. All slides were assessed in the pathology department according to the Sydney classification. The departmental pathology team performed histopathological review in routine clinical practice; formal interobserver reliability assessment was not conducted as part of this retrospective study, which represents a limitation.

Chronic gastritis was diagnosed histologically based on the presence of chronic mononuclear inflammatory infiltrates (lymphocytes and plasma cells) within the gastric lamina propria, with or without associated activity, atrophy, intestinal metaplasia, or *H. pylori* colonization. Image-enhanced endoscopy was not routinely used during the study period.

Data Collection

Data collected for each patient included age, sex, and the histopathological presence or absence of GIM, *H. pylori* infection, and chronic gastritis. Data were entered and managed in Microsoft Excel.

Statistical Analysis

Statistical analysis was performed using Jamovi software (version 2.6.26). Categorical variables were summarized as frequencies and percentages. Associations between GIM and categorical predictors (age group, sex, *H. pylori* status) were assessed using chi-square tests and reported as unadjusted

odds ratios (OR) with 95% confidence intervals (CI). Binary logistic regression was used to calculate adjusted odds ratios (aORs), with age group, sex, and *H. pylori* status entered simultaneously into the model; the age group of ≤20 years served as the reference category. Age was also modeled as a continuous variable to estimate the per-year change in odds. Chronic gastritis could not be included in the regression model due to its near-universal prevalence, which produced unstable estimates. A *P*-value <0.05 was considered statistically significant.

Ethical Approval

The study protocol was reviewed and approved by the Libyan National Committee for Biosafety and Bioethics (LNCBB; reference number: NCB.019.H.25.18). The requirement for informed consent was waived by the ethics committee, given the retrospective nature of the study, which involved the analysis of pre-existing, anonymized clinical data. All patient data were handled in strict accordance with the principles of the Declaration of Helsinki.

RESULTS

Patient Demographics

The final study cohort comprised 442 patients: 272 (61.5%) female and 170 (38.5%) males. The median age was 40 years (interquartile range [IQR], 24–53 years; range, 2–94 years). The age distribution was right-skewed, with the IQR spanning considerably below the maximum observed age, consistent with the concentration of patients in younger and middle-aged groups. The largest age group was those aged ≤20 years (20.8%), followed by the 31 to 40 years group (19.9%) and the 41 to 50 years group (17.4%). The female predominance in this cohort (61.5%) likely reflects local healthcare utilization and endoscopy referral patterns rather than underlying disease epidemiology, though this was not formally assessed in the present study. The demographic distribution by age group and sex is presented in **Table 1**.

Detection Rate of GIM, *H. pylori* Infection, and Chronic Gastritis

GIM was found in 28 patients (6.3%), *H. pylori* infection in 300 patients (67.9%), and chronic gastritis in 439 patients (99.3%). These figures reflect the biopsy-selected subgroup of patients with macroscopic gastric abnormalities and do not represent the overall endoscopic population, in which the true frequencies may be higher than these data suggest.

Table 1: Patient demographics by age group and sex.

Age group (years)	Male	Female	Total
≤20	34 (20.0%)	58 (21.3%)	92 (20.8%)
21–30	21 (12.4%)	33 (12.1%)	54 (12.2%)
31–40	37 (21.8%)	51 (18.8%)	88 (19.9%)
41–50	25 (14.7%)	52 (19.1%)	77 (17.4%)
51–60	28 (16.5%)	43 (15.8%)	71 (16.1%)
61–70	13 (7.6%)	21 (7.7%)	34 (7.7%)
>70	12 (7.1%)	14 (5.1%)	26 (5.9%)
Total	170 (100%)	272 (100%)	442 (100%)

Association between GIM and *H. pylori*, Stratified by Sex

Table 2 presents the distribution of GIM according to *H. pylori* status, stratified by sex. A notable pattern warrants direct comment: in both males and females, and in the overall cohort, GIM appeared numerically more common among *H. pylori*-negative individuals than among *H. pylori*-positive individuals. This counterintuitive direction (overall OR 0.61 in univariate analysis, suggesting an apparent inverse association) most likely reflects the "disappearing *H. pylori*" phenomenon: bacterial density characteristically declines in advanced atrophic and metaplastic mucosa, reducing histological detectability, such that patients with established GIM may test negative for active *H. pylori* infection despite a significant prior exposure history. Accordingly, these differences did not reach statistical significance in either sex subgroup (males: $P = 0.298$; females: $P = 0.425$) or in the overall cohort ($P = 0.209$) and should not be interpreted as evidence that *H. pylori* is protective against GIM. **Table 2** summarizes GIM distribution according to *H. pylori* status, stratified by sex.

Association Between Age and GIM

Age group was significantly associated with the presence of GIM ($\chi^2 = 14.7$, $P = 0.022$). GIM identified rate rose progressively with age, being lowest in the ≤20-year group (3.3%) and highest in the 61 to 70-year group (14.7%) and 51 to 60-year group (12.7%). The distribution of *H. pylori* infection and GIM across age groups is summarized in **Table 3**.

Multivariable Analysis

On univariate analysis, patients aged 51 to 60 years and 61 to 70 years had significantly higher odds of GIM compared with those aged ≤20 years (OR, 2.69 [95% CI, 1.16–6.22]; $P = 0.021$ and OR, 2.89 [95% CI, 1.02–8.15]; $P = 0.045$, respectively). After adjustment for sex and *H. pylori* status, these associations strengthened: patients aged 51 to 60 years had an aOR of 4.03 (95% CI, 1.04–15.60; $P = 0.044$) and those aged 61 to 70 years had an aOR of 4.86 (95%

Table 2: Distribution of GIM according to *H. pylori* status, stratified by sex.

Sex	Intestinal metaplasia	*H. pylori* positive	*H. pylori* negative	Total
Male	Positive	8	6	14
	Negative	110	46	156
	Total	118	52	170
Female	Positive	8	6	14
	Negative	174	84	258
	Total	182	90	272
Overall	Positive	16	12	28
	Negative	284	130	414
	Total	300	142	442

GIM, gastric intestinal metaplasia; *H. pylori*, *Helicobacter pylori*. The numerically higher GIM frequency among *H. pylori*-negative patients reflects the "disappearing *H. pylori*" phenomenon—reduced bacterial density and histological detectability in advanced atrophic/metaplastic mucosa—rather than a protective effect; none of the between-group differences reached statistical significance (males, $P = 0.298$; females, $P = 0.425$; overall, $P = 0.209$).

CI, 1.09–21.80; P = 0.039). No significant associations were observed for sex (aOR, 1.69 [95% CI, 0.77–3.72]; P = 0.190) or *H. pylori* infection (aOR, 0.66 [95% CI, 0.30–1.49]; P = 0.323). Although patients aged >70 years did not reach statistical significance (aOR, 3.16 [95% CI, 0.58–17.35]; P = 0.185), the elevated point estimate and GIM identified rate of 11.5% in this subgroup suggest a plausible age-related trend that is likely attenuated by limited statistical power (n = 26); caution is warranted in interpreting this apparent plateau as a true biological phenomenon rather than a small-sample artifact. When modeled as a continuous variable, each additional year of age was independently associated with higher odds of GIM (unadjusted OR 1.03 per year [95% CI, 1.01–1.05]; P = 0.002; adjusted OR, 1.03 per year [95% CI, 1.01–1.05]; P = 0.003). Multivariable results are presented in full in **Table 4**.

Association Between Age and *H. pylori*

A significant association was observed between age group and *H. pylori* infection status (χ^2 = 14.5, P = 0.025). *H. pylori* positivity was highest in the ≤20-year group (79.3%) and declined progressively with advancing age, reaching its lowest level in the >70-year group (42.3%). **Figure 1** illustrates the distribution of *H. pylori*–positive cases across age groups, stratified by sex. No significant association was found between sex and *H. pylori* infection (95% CI, 0.74–1.70; P = 0.584 [95% CI, 0.76–1.76]; P = 0.509). **Figure 2** presents the GIM distribution across age groups based on sex.

Table 3: Distribution of *H. pylori* infection and GIM across age groups.

Age group (years)	N	*H. pylori* positive	*H. pylori* negative	GIM positive
≤20	92	73 (79.3%)	19 (20.7%)	3 (3.3%)
21–30	54	38 (70.4%)	16 (29.6%)	1 (1.9%)
31–40	88	60 (68.2%)	28 (31.8%)	3 (3.4%)
41–50	77	49 (63.6%)	28 (36.4%)	4 (5.2%)
51–60	71	46 (64.8%)	25 (35.2%)	9 (12.7%)
61–70	34	23 (67.6%)	11 (32.4%)	5 (14.7%)
>70	26	11 (42.3%)	15 (57.7%)	3 (11.5%)
Total	442	300 (67.9%)	142 (32.1%)	28 (6.3%)

GIM, gastric intestinal metaplasia; *H. pylori*, *Helicobacter pylori*.

DISCUSSION

In this retrospective cross-sectional study of 442 patients who underwent EGD evaluation with biopsy for macroscopic gastric abnormalities at Tripoli Central Hospital, GIM was identified in 6.3% of participants, *H. pylori* infection in 67.9%, and chronic gastritis in 99.3%. Increasing age was the strongest independent predictor of GIM, with patients aged 51–60 and 61–70 years showing significantly higher odds of GIM than those aged ≤20 years (aOR, 4.03 and 4.86, respectively). Neither sex nor current *H. pylori* infection status was independently associated with GIM in multivariable analysis.

The detection rate of GIM observed in this cohort (6.3%) is notably lower than that reported in many other settings. Global estimates suggest an overall prevalence of approximately 17.5%, [3] and higher rates have been reported across comparable regional and international cohorts: 11% in Tunisia, [16] 13.8% in Turkey, [5] 20.5% in Iraq, [6] and approximately 15% among patients undergoing endoscopic evaluation in the United States. [7] However, this figure must be interpreted with the understanding that it is likely a substantial underestimate of the true GIM burden in this population. Two key methodological factors conspire to reduce detection: only patients with macroscopic gastric abnormalities underwent biopsy (thereby excluding cases where GIM develops without endoscopically visible changes), and biopsies were not obtained according to the USSBP. Because GIM is frequently patchy and site-dependent, non-targeted or limited sampling may fail to detect focal lesions, [1] as a minimum estimate rather than a true prevalence.

The post-adjustment strengthening of the association between older age groups and GIM—with ORs increasing after adjustment for *H. pylori* and sex—likely reflects negative confounding. *H. pylori* positivity was highest in younger participants, while GIM detection rate increased with age; adjustment for *H. pylori*, therefore, unmasked a stronger independent association between older age and GIM. The overall finding of a progressive age-related increase in GIM is consistent with data from diverse populations globally, including the United States, [7,8] Thailand, [15] Germany, [9] and Australia, [11] all of which describe higher GIM prevalence in persons aged over 50 years. [12] This pattern is biologically credible and mirrors the progressive nature of

Table 4: Predictors of gastric intestinal metaplasia: univariate and multivariable analysis (reference: age ≤ 20 years).

Variable	Univariate OR (95% CI)	P-value	Adjusted OR (95% CI)	P-value	Notes
Age 21–30 years	0.25 (0.03–1.90)	0.181	0.52 (0.05–5.19)	0.579	
Age 31–40 years	0.46 (0.14–1.58)	0.218	0.97 (0.19–4.96)	0.967	
Age 41–50 years	0.78 (0.26–2.31)	0.652	1.56 (0.33–7.25)	0.573	
Age 51–60 years	2.69 (1.16–6.22)	0.021	4.03 (1.04–15.60)	0.044	
Age 61–70 years	2.89 (1.02–8.15)	0.045	4.86 (1.09–21.80)	0.039	
Age >70 years	2.04 (0.57–7.26)	0.271	3.16 (0.58–17.35)	0.185	N = 26; trend likely attenuated by limited power
Male sex	1.65 (0.77–3.56)	0.199	1.69 (0.77–3.72)	0.190	
H. pylori positive	0.61 (0.28–1.33)	0.213	0.66 (0.30–1.49)	0.323	See *Discussion* for interpretation of OR direction.

aOR, adjusted odds ratio; CI, confidence interval. *P*-values indicate statistical significance (P < 0.05). Multivariable model adjusted for age group, sex, and *H. pylori* status.

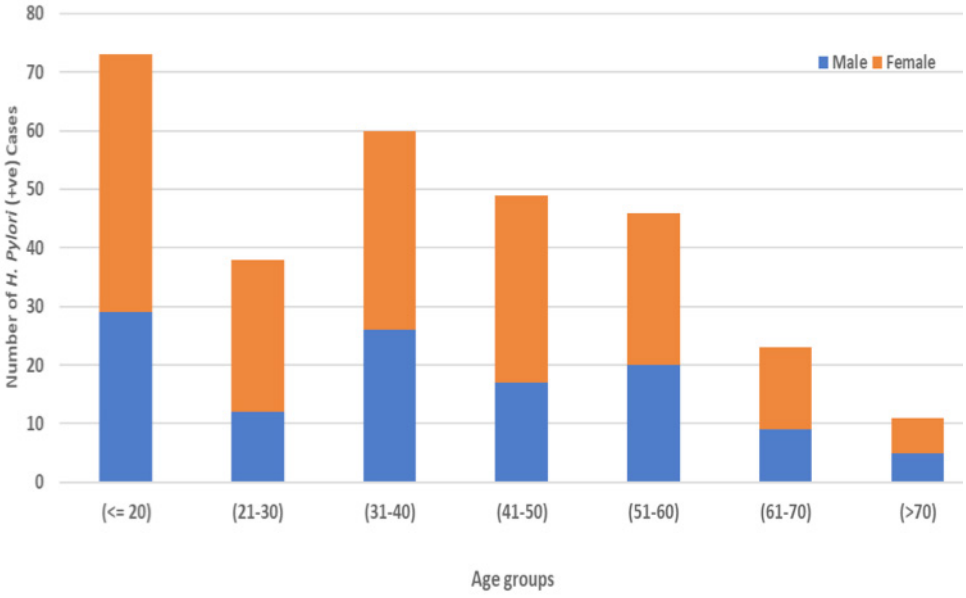

Figure 1: Distribution of *H. pylori*–positive cases across age groups by gender.

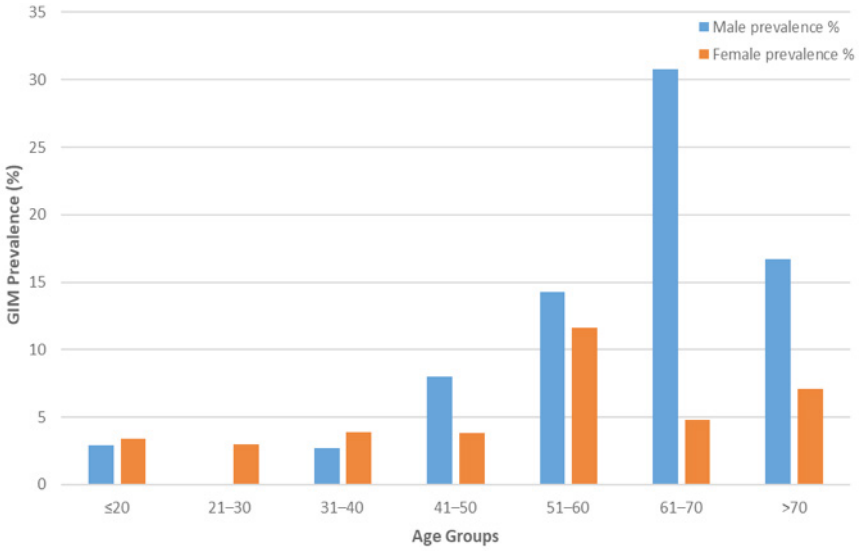

Figure 2: Distribution of gastric intestinal metaplasia (GIM) across age groups by gender.

Correa's cascade, in which sustained gastric inflammation and collective exposure to environmental risk factors over decades can cause atrophic changes and eventually GIM. [2]

The absence of a significant association between current *H. pylori* infection status and GIM in this cohort contrasts with the findings of a recent systematic review and meta-analysis, which reported a significant positive association between *H. pylori* and GIM risk. [4] The direction of the *H. pylori* OR in this study—below unity in both univariate (OR, 0.61) and multivariable (aOR, 0.66) analyses—is counterintuitive and warrants explicit explanation rather than dismissal as a non-significant finding. This inverse direction most likely reflects the well-described "disappearing *H. pylori*" phenomenon, whereby bacterial density falls in advanced atrophic and metaplastic mucosa, reducing histological detectability in precisely those patients who have been most durably infected. [17] Compounding this, although prior eradication status was not recorded in this dataset, some older patients with established GIM may have undergone prior eradication therapy and tested negative at the time of biopsy despite substantial earlier exposure. [13] Together, these mechanisms mean that cross-sectional histological testing at a single point in time is an imperfect surrogate for the cumulative *H. pylori* exposure that drives GIM development. A Turkish endoscopic study similarly found no significant association between *H. pylori* and intestinal metaplasia, [18] suggesting that this dissociation is a recognized phenomenon in cross-sectional biopsy studies rather than a Libya-specific finding.

The observed decline in *H. pylori* positivity with advancing age in this cohort is consistent with prior reports.

[17] Two non-mutually exclusive mechanisms may contribute: progressive atrophic and metaplastic mucosal changes reduce bacterial colonization density and, therefore, histological detectability in older individuals; and cohort effects may mean that younger generations have been exposed to different *H. pylori* acquisition patterns reflecting contemporary epidemiological conditions. Notably, the GIM detection rate did not continue to rise monotonically in the oldest stratum, declining slightly from 14.7% in the 61 to 70-year group to 11.5% in the >70-year group. This apparent plateau most plausibly reflects the small size of the >70 subgroup (n = 26) and possible survival or selection effects, rather than a genuine reversal of the age–GIM relationship; the continuous-age model, which showed a consistent 3% increase in odds per year (OR, 1.03 [95% CI, 1.01–1.05]), supports an overall monotonic trend.

Male sex showed a modest but non-significant association with GIM (OR, 1.65; aOR, 1.69; P = 0.190). This finding contrasts with reports from Iraq, where 61% of GIM cases occurred in men, [6] and from Iran, where a higher prevalence of GIM was observed in men than women (20.2% vs 16.4%). [19] Available evidence does not consistently demonstrate a significant sex-based difference in GIM prevalence; observed variation more likely reflects differences in age distribution, referral patterns, and endoscopy population selection than a true sex-specific biological effect. The women in this cohort (61.5%) were remarkable and not readily explicable. The higher proportion of women may indicate gender-specific disparities in healthcare-seeking conduct or gastrointestinal symptomatology within the Libyan environment. Nonetheless, these assumptions are not explicitly verifiable or addressed with the existing data, necessitating additional multicenter research.

Chronic gastritis was present in nearly all biopsied patients (99.3%). This near-universal frequency reflects the biopsy-selected nature of the study population, in which only patients with macroscopic gastric abnormalities underwent tissue sampling. While this frequency rendered chronic gastritis statistically non-informative as an independent covariate in regression modeling, it does not in itself indicate a diagnostic concern: the finding is expected in a cohort selected based on visible mucosal disease, and the histological criteria applied were standard. Because only macroscopically abnormal mucosa was sampled, the histological baseline of this cohort is inherently skewed toward pathology, and normal gastric mucosa was neither sampled nor histologically defined. Furthermore, the diagnostic threshold for chronic gastritis—the presence of any chronic mononuclear infiltrate within the lamina propria—is intrinsically sensitive and may overestimate disease in a population selected for visible mucosal abnormality. This 99.3% figure, therefore, represents the frequency within a biopsy-selected, endoscopically abnormal subgroup and cannot be extrapolated to the general Libyan population.

Limitations

This study has several important limitations. First, the retrospective cross-sectional design precludes causal inference. Second, and most consequentially, biopsies were obtained according to routine clinical practice rather than a standardized Sydney mapping protocol, and only from patients with macroscopically abnormal mucosa. Because GIM may be patchy and site-dependent, this approach likely led to underdetection of focal lesions and substantial underestimation of true GIM prevalence. Consequently, site-specific risk staging systems such as Operative Link on Gastritis (OLGA) and Operative Link on Gastric Intestinal Metaplasia (OLGIM) could not be applied. In addition, the exclusion of 49 patients with incomplete records may have introduced selection bias, although these exclusions reflected missing data fields rather than clinical characteristics. Third, important potential confounders—including smoking history, dietary habits, prior *H. pylori* treatment, proton pump inhibitor use, and medication history—were unavailable in this retrospective dataset; unrecorded proton pump inhibitor use in particular may have reduced the sensitivity of histological *H. pylori* detection and further confounded the *H. pylori*–GIM relationship. Fourth, data on prior eradication therapy were not available, limiting interpretation of the *H. pylori*–GIM relationship and precluding distinction between current and historical infection. Fifth, the relatively small number of GIM cases (n = 28) limited statistical power for subgroup analyses, particularly in smaller age categories such as the >70-year group (n = 26), where the wide confidence intervals reflect substantial uncertainty. Sixth, a histopathological review was performed by the departmental pathology team as part of routine clinical care without a formal inter-observer reliability assessment; this may introduce diagnostic variability, particularly for borderline lesions. Seventh, the single-center design and single endoscopist, while ensuring procedural consistency, limit the generalizability of findings to other Libyan institutions and may introduce individual operator variability in biopsy site selection. Eighth, our histopathological assessment did not distinguish complete (type I) from incomplete (type II/III) intestinal metaplasia. Incomplete subtypes carry a substantially higher risk of progression to gastric cancer, and this subtyping—increasingly recommended for risk stratification—could not be performed retrospectively from the available records. Finally, image-enhanced endoscopy was not routinely employed, which may have reduced the detection of subtle mucosal abnormalities.

CONCLUSIONS

In this biopsy-selected Libyan cohort, the observed GIM detection rate of 6.3% is likely an underestimate of true disease burden, attributable to the non-systematic biopsy protocol and restriction of sampling to patients with macroscopic gastric abnormalities. Increasing age was the strongest independent factor associated with GIM, with significantly elevated odds in adults aged 51 to 70 years (aOR, 4.03–4.86), while neither sex nor current *H. pylori* infection status emerged as an independent predictor. The apparent inverse association between current *H. pylori* status and GIM most likely reflect the disappearing *H. pylori* phenomenon in advanced mucosal disease rather than a true protective effect. These findings suggest that larger population-based studies in Libya using standardized biopsy protocols to establish accurate prevalence estimates are required. Future studies should also investigate whether targeted endoscopic surveillance strategies for adults aged ≥50 years, analogous to approaches recommended in other intermediate- and high-risk regions, would be appropriate and feasible in the Libyan healthcare setting.

AUTHORS' CONTRIBUTION

Each author has made a substantial contribution to the present work in one or more areas, including conception, study design, conduct, data collection, analysis, and interpretation. All authors have given final approval of the version to be published, agreed on the journal to which the article has been submitted, and agree to be accountable for all aspects of the work.

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CONFLICT OF INTEREST

None.

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