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Research Article

Association of Helicobacter pylori Status with Fecal Calprotectin and Fecal Occult Blood among Primary Healthcare Attendees in Baghdad, Iraq: A Cross-Sectional Study

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ABSTRACT

Background: Fecal calprotectin is a marker of gastrointestinal inflammation due to neutrophils; however, there is a lack of evidence about its correlation with *H. pylori* infection, especially among symptomatic individuals seeking care from primary health centers. Aim: The aim of the study was to compare the levels of fecal calprotectin and the fecal occult blood test among patients with positive and negative *H. pylori* infection and to assess the association of increased calprotectin level with *H. pylori* infection.

Materials and Methods: In a cross-sectional study that took place in Baghdad from May 2025 to May 2026, patients with gastrointestinal symptoms were recruited. Active *H. pylori* infection was diagnosed using stool antigen test and ^{13}C-urea breath test. Fecal calprotectin was determined as a continuous variable and also at the cut-offs of 50 and 250 µg/g.

Results: Results: Out of 268 patients, 133 were *H. pylori* positive, and 135 were *H. pylori* negative. The median concentration of fecal calprotectin was significantly higher in *H. pylori*-positive subjects than in the negative ones (63.0 vs 17.0 µg/g; P<0.001). There was a rank biserial correlation between these variables equal to 0.409. The concentration exceeded 50 µg/g in 53.4% and 14.1% of subjects, respectively. After correction for age, sex, alcohol abuse, smoking, obesity, medication, diabetes mellitus, inflammatory bowel disease, diverticulitis, colon cancer, irritable bowel syndrome, peptic ulcer disease, celiac disease, lactose malabsorption, and Helicobacter species, elevated calprotectin concentration was significantly associated with *H. pylori* positivity (odds ratio 6.89, 95% confidence interval 3.77-12.60; P<0.001). Occult fecal positivity was also more common among H. pylori-positive patients, but adjusted odds ratio did not reach statistical significance. The area under the curve was 0.705.

Conclusion: Positive *H. pylori* status correlated with increased levels of fecal calprotectin. Nevertheless, the relatively low discrimination capacity and sensitivity demonstrated by this marker make it unsuitable to be used alone for diagnosis. Further longitudinal research is necessary to assess biomarker alterations following treatment while considering the presence of comorbid conditions.

1. INTRODUCTION

Helicobacter pylori chronically colonizes the gastric mucosa and remains common worldwide despite a gradual decline in prevalence. A global analysis covering 1980-2022 estimated a decrease from approximately 58% in 1980-1990 to 43% in 2011-2022, with marked variation by region, age, socioeconomic conditions, and diagnostic method [1]. Persistent infection produces active chronic gastritis and is associated with peptic ulcer disease, gastric mucosa-associated lymphoid tissue lymphoma, and gastric adenocarcinoma. International guidance therefore considers H. pylori an infectious disease that requires accurate diagnosis and eradication when detected [2,3].

The selection and interpretation of diagnostic tests require particular attention in Iraqi clinical practice. An Iraqi validation study comparing invasive and non-invasive methods reported test-dependent positivity rates and found that the urea breath test and stool antigen test performed well against quantitative polymerase chain reaction [4]. The diagnostic approach used to classify participants therefore directly affects the validity of the principal grouping variable and should be described precisely.

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Fecal calprotectin is a calcium and zinc binding S100A8/S100A9 protein complex secreted mainly by neutrophils. Its stability in feces allows non-invasive assessment of neutrophil activity in the intestine [5]. Clinically, the application is mainly to differentiate inflammatory bowel disease from functional diseases of the gastrointestinal tract and assess intestinal inflammation. The biomarker is non-specific and concentrations may increase in case of infection, medications, age, and upper gastrointestinal tract inflammation.

The results obtained in the literature for the H. pylori infection and fecal calprotectin are inconsistent. Some authors noted higher levels in H. pylori-positive gastritis with correlation with neutrophil activity in stomach [6]. Other studies found moderate [7] or poor [8] diagnostic value, being rather related to gastritis activity rather than to infection presence [8] or absence of elevation in chronic gastritis [9]. In a recent pediatric study, there have been high levels of fecal calprotectin and systemic inflammatory factors in the infected patients [10], whereas an adult prospective study showed high levels that reduced post-eradication [11].

Differences among various studies might be due to different ages, sampling methods, assay characteristics, use of medications, severity of histology, presence of concurrent intestinal diseases, and methods used for detection of H. pylori infection. This study assessed the H. pylori status, fecal calprotectin, age, gender, and fecal occult blood test (FOB) status. The first research question of this study is to assess the level of fecal calprotectin in H. pylori positive and negative cases. The second research questions include the assessment of FOB status, relationship between high fecal calprotectin and H. pylori positivity, and discrimination within the study using receiver operating characteristic analysis. The study design does not estimate population prevalence, causation, and new diagnostic threshold [12].

2. MATERIALS AND METHODS

2.1 Study design and setting

The cross-sectional study was carried out in Baghdad, Iraq, between May 2025 and May 2026. The subjects were sourced from primary healthcare centers and referred to Al-Nokhba Private Laboratory for screening of gastrointestinal symptoms and suspected active infection of H. pylori. Screening for H. pylori, fecal calprotectin, and fecal occult blood was carried out at the above-mentioned laboratory.

The subjects were enrolled during the predetermined study period on a consecutive basis. Each subject underwent two tests for detecting active H. pylori infection and fecal calprotectin test as described in the following sections.

2.2 Participants and sampling

Patients of all ages who attended primary healthcare centres with gastrointestinal symptoms were assessed consecutively for eligibility. Inclusion required concordant H. pylori test results and an available fecal calprotectin measurement. Patients were excluded if they had recently used antibiotics or had previously received H. pylori eradication therapy. Before the ^{13}C-urea breath test, proton-pump inhibitors were withheld for at least two weeks, and antibiotics and bismuth-containing medications for at least four weeks. Classification as H. pylori-positive or H. pylori-negative followed the concordant two-test definition specified below.

2.3 Laboratory measurements and operational definitions

2.3.1 Determination of H. pylori status

Active H. pylori infection was evaluated using two non-invasive methods: an H. pylori stool antigen test and the ^{13}C-urea breath test. Final infection status was assigned only when the results of both tests were concordant.

- H. pylori stool antigen test
  Fresh stool samples were examined using the OnSite® H. pylori Ag Rapid Test CE (Cat. No. R0192C; CTK Biotech, Poway, California, USA), a qualitative immunochromatographic assay for H. pylori antigen in human feces. Each specimen was transferred to the supplied extraction device containing buffer and mixed until homogeneous. The prepared suspension was added to the cassette sample well, and the result was read after 10 minutes according to the manufacturer's instructions. Visible control and test lines indicated a positive result, while a control line alone indicated a negative result. Tests without a visible control line were considered invalid and repeated with a new cassette.
- ^{13}C-urea breath test
  The ^{13}C-urea breath test was conducted using the Heliforce® Urea [^{13}C] Breath Test Kit containing 50 mg of ^{13}C-urea (Beijing Richen-Force Science & Technology Co., Ltd., Beijing, China). Breath samples were analyzed with the Richen IR-force 200 ^{13}C-urea breath test analyzer from the same manufacturer. Participants fasted for at least four hours before testing. Proton-pump inhibitors were discontinued for at least two weeks, and antibiotics and bismuth-containing medications for at least four weeks.

A baseline breath sample was collected before administration of 50 mg of ^{13}C-urea, followed by a second sample 30 minutes later. Both samples were analyzed with the IR-force 200 instrument. Results were expressed as the difference over baseline (DOB), calculated from the change in the $^{13}CO_2/^{12}CO_2$ ratio. A DOB value ≥4.0‰ was considered positive, and a value <4.0‰ negative. Participants were classified as H. pylori-positive when both tests were positive and as H. pylori-negative when both were negative. When initial results were discordant, both tests were repeated, and classification was based on the concordant repeat results.

2.3.2 Fecal calprotectin quantification

Fecal calprotectin levels were quantitated using Alegria® automated ELISA system along with Alegria® Calprotectin test strips (Cat. No. ORG 280; ORGENTEC Diagnostika GmbH, Mainz, Germany). The fresh stool samples were prepared in the provided extraction tubes and buffer following the manufacturer's instructions. After homogenization, the supernatant was added to the analyzer. Incubation, wash steps, reaction with conjugate, substrate development, calibration and calculation were done automatically. Results were reported in micrograms per gram of feces (µg/g). The instrument calibration and internal quality control were carried out according to the manufacturer's guidelines and laboratory's protocol, and only successful tests were included in the analysis.

Fecal calprotectin was analyzed both as a continuous variable and categorized as follows: normal (≤50 µg/g), intermediate (>50 to ≤250 µg/g), or high (>250 µg/g). Four values reported as >1000 µg/g were top-coded at 1000 µg/g. This treatment did not alter the rank-based group comparison or analyses based on the predefined clinical thresholds.

2.3.3 Fecal occult blood testing

Fecal occult blood was assessed using the OnSite® FOB Rapid Test CE (Cat. No. R2010C; CTK Biotech, Poway, California, USA), a qualitative immunochromatographic assay for human hemoglobin in feces. A portion of each specimen was placed in the supplied collection device containing 2 mL of extraction buffer. The prepared extract was added to the sample well, and the result was read at 10 minutes; readings outside the specified interval were considered invalid. Visible control and test lines indicated a positive result, while a control line alone indicated a negative result. Tests without a control line were repeated using a new cassette. The stated analytical detection limit was 50 ng/mL of human hemoglobin. Age was recorded in completed years, sex as female or male, and FOB status as positive or negative.

2.4 Statistical analysis

Data were analyzed in R 4.6.1 using two-sided tests (P<0.05). Group comparisons used Welch's t test, Mann-Whitney U, chi-square, or Fisher's exact test. Rank-biserial correlation was reported for the Mann-Whitney comparison. Effect estimates were presented with 95% CIs. Logistic regression adjusted for age, sex, and FOB. Exploratory ROC analysis reported AUC, bootstrap 95% CI, sensitivity, and specificity.

3. RESULTS

3.1 Participant characteristics

The analysis included 268 participants: 133 H. pylori-positive and 135 H. pylori-negative. Mean age was 39.7 ± 15.5 years (range 4-88) and did not differ significantly between groups (P=0.212). The sample comprised 108 females (40.3%) and 160 males (59.7%), with no significant difference in sex distribution (P=0.273). FOB positivity occurred more often in the H. pylori-positive group than in the negative group (12.0% vs 5.2%), although the difference was not statistically significant (P=0.051) (As shown in the Table I).

TABLE I. PARTICIPANT CHARACTERISTICS AND FECAL FINDINGS ACCORDING TO H. PYLORI STATUS

Characteristic	Total sample (n=268)	H. pylori positive (n=133)	H. pylori negative (n=135)	P value
Age, years, mean ± SD	39.7 ± 15.5	40.9 ± 15.1	38.5 ± 15.8	0.212
Sex, n (%)				0.273
Female	108 (40.3)	58 (43.6)	50 (37.0)	
Male	160 (59.7)	75 (56.4)	85 (63.0)	
FOB positive, n (%)	23 (8.6)	16 (12.0)	7 (5.2)	0.051
Fecal calprotectin, µg/g, median (IQR)	27.5 (9.0-84.5)	63.0 (11.8-224.0)	17.0 (6.4-34.5)	<0.001
Calprotectin >50 µg/g, n (%)	90 (33.6)	71 (53.4)	19 (14.1)	<0.001
Calprotectin >250 µg/g, n (%)	32 (11.9)	32 (24.1)	0 (0.0)	<0.001

Values are presented as mean ± SD, median (IQR), or n (%), with percentages calculated within columns. The P value for sex represents the overall comparison and is reported on the parent row. FOB, fecal occult blood; IQR, interquartile range; SD, standard deviation.

3.2 Fecal calprotectin distribution

Fecal calprotectin showed a pronounced right-skewed distribution in both groups. Median concentration was 63.0 µg/g (IQR 11.8-224.0) among H. pylori-positive participants and 17.0 µg/g (IQR 6.4-34.5) among negative participants (Mann-Whitney P<0.001). The rank-biserial correlation was 0.409, indicating moderate distributional separation. Normal concentrations occurred in 46.6% of positive participants and 85.9% of negative participants. Intermediate concentrations occurred in 29.3% and 14.1%, respectively, and all 32 values >250 µg/g were observed in the positive group (Fisher's exact P<0.001).

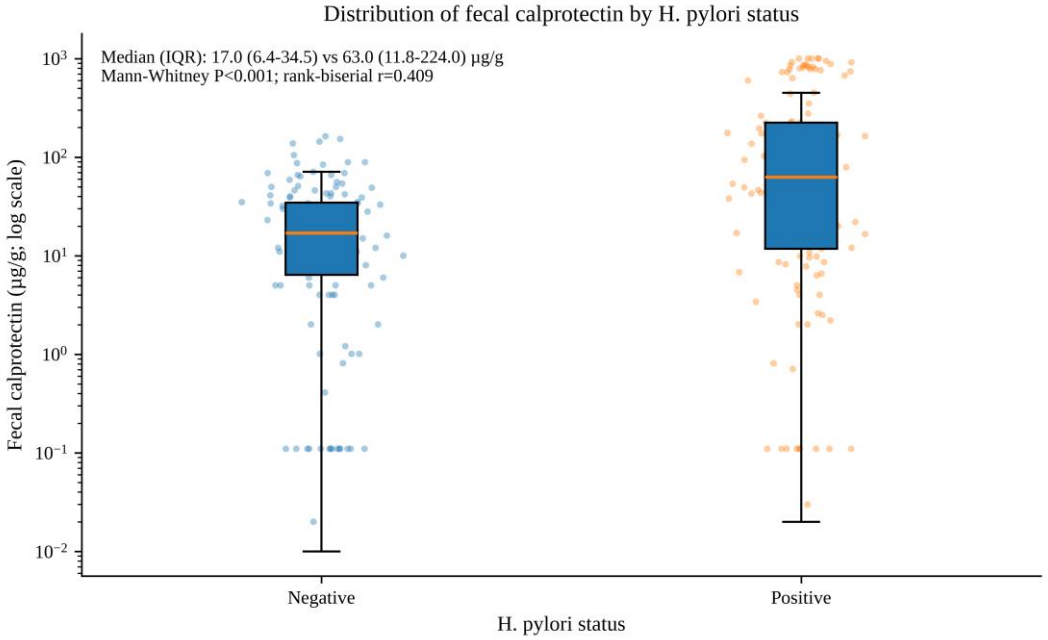

Fig. 1. Distribution of fecal calprotectin according to Helicobacter pylori infection status.

Figure 1. Distribution of fecal calprotectin according to H. pylori status. The logarithmic axis shows the pronounced right-skew while retaining differences across lower and higher concentration ranges. Four values reported as >1000 µg/g were top-coded at 1000 µg/g for graphical presentation.

3.3 Multivariable association with H. pylori positivity

After adjustment for age, sex, and FOB, elevated fecal calprotectin remained independently associated with H. pylori positivity. Participants with concentrations >50 µg/g had 6.89-fold higher adjusted odds of belonging to the H. pylori-positive group (adjusted OR 6.89, 95% CI 3.77-12.60; P<0.001). Age, sex, and FOB positivity were not independently associated with the outcome (As shown in the Table II).

TABLE II. MULTIVARIABLE LOGISTIC REGRESSION WITH H. PYLORI POSITIVITY AS THE OUTCOME

Predictor	Adjusted OR	95% CI	P value
Elevated calprotectin (>50 µg/g)	6.89	3.77-12.60	<0.001
Age, per year	1.009	0.991-1.027	0.348
Sex, male vs female	0.73	0.42-1.26	0.257
FOB positive	1.51	0.55-4.18	0.428

Model n=268; likelihood-ratio P<0.001; McFadden pseudo-R^2=0.140. Female sex was the reference category. CI, confidence interval; OR, odds ratio; FOB, fecal occult blood.

3.4 Exploratory ROC analysis

The AUC for continuous fecal calprotectin was 0.705 (bootstrap 95% CI 0.638-0.767), corresponding to moderate discrimination within this sample. At the predefined threshold >50 µg/g, sensitivity was 53.4% and specificity 85.9%. At the sample-derived Youden threshold of ≥74 µg/g, sensitivity was 48.9% and specificity 93.3%. The higher threshold increased specificity but further reduced sensitivity (As shown in the Table III).

TABLE III. EXPLORATORY DIAGNOSTIC-PERFORMANCE ESTIMATES FOR FECAL CALPROTECTIN

Metric/threshold	Estimate	Sensitivity	Specificity
Area under the curve	0.705 (95% CI 0.638-0.767)	-	-
>50 µg/g	Clinical threshold	53.4%	85.9%
>=74 µg/g	Sample-derived Youden threshold	48.9%	93.3%

AUC, area under the curve; CI, confidence interval. The Youden threshold was derived from the current sample and is presented for exploratory purposes.

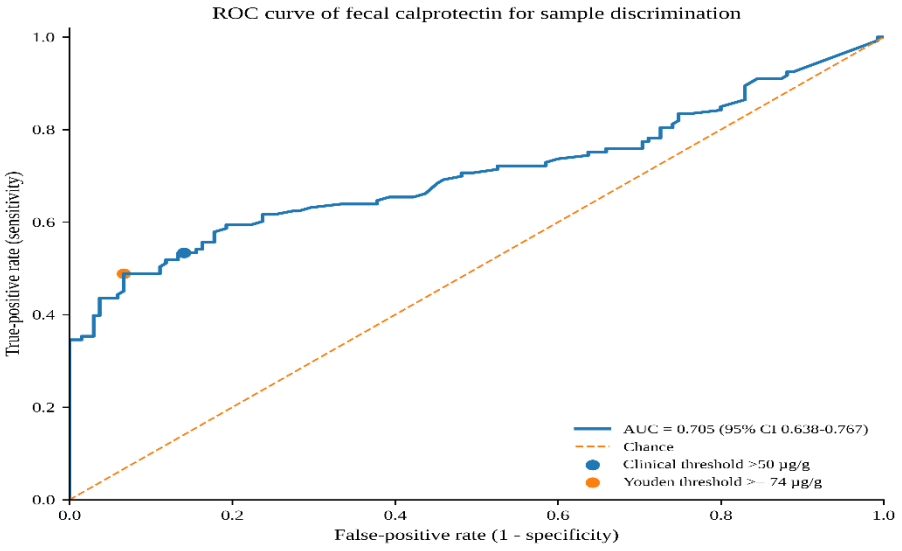

Fig. 2. Receiver operating characteristic (ROC) curve of fecal calprotectin for discriminating Helicobacter pylori infection.

Figure 2. Receiver operating characteristic curve for fecal calprotectin as a discriminator of H. pylori status in the analytical sample. The AUC represents discrimination within this sample and should not be interpreted as evidence of replacement diagnostic accuracy.

4. DISCUSSION

H. pylori- positive participants showed median levels of calprotectin concentration in their stool samples almost four times the level in H. pylori-negative participants, together with a moderate rank biserial effect size and greater occurrence of calprotectin levels >50 µg/g. Adjusted estimate was similar to unadjusted association indicating that the observed pattern was not affected by any potential confounders such as age, gender, and FOB. Values of >250 µg/g were seen in 24.1% of H. pylori-positive participants compared to zero percent in H. pylori-negative participants.

It should be stressed that the findings presented above do not prove that H. pylori is the only factor which causes the increase in calprotectin concentration. Both infection status and biomarker levels were assessed at one time point and the database used for this analysis lacked patients with inflammatory bowel disease, infectious enteritis, medication intake, and other factors which may have led to the elevation of neutrophil markers in stool samples.

An OR of 6.89 is suggestive of a fairly strong association, but, to some degree, its size might be accounted for by the design of the constructed sample. Having approximately equal numbers of both positive and negative cases increases efficiency when making group comparisons; however, it does not reflect the prevalence of the infection within an unselected clinical population. Adjustment was limited to the variables available without any missing values. Residual confounding may still be possible due to the absence of information on the presence of symptoms, endoscopic findings, activity of gastritis, body mass index, smoking, medications intake, recent antibiotic treatment, and comorbidities of the gastrointestinal tract.

The direction of the associations described in the present study is in accordance with several other recent publications. Aksoy et al. demonstrated increased fecal calprotectin levels in the children suffering from H. pylori-positive gastritis and showed the association of calprotectin concentration with gastric neutrophil activity [6]. Villalba-Davila et al. also demonstrated a marked increase in the levels of calprotectin among the children infected with H. pylori and associated it with the higher incidence of colonoscopy [10].

The findings are also congruent with the prospective study of adults conducted in 2026 by Mazzawi and Al Haj Mahmoud, which showed an elevated level of fecal calprotectin in infected adults and reduced concentrations after eradication therapy [11]. Prospective assessment is a more temporally reliable approach than cross-sectional comparison because a decrease following effective treatment is more indicative of a role played by H. pylori-induced inflammation. Reversibility could

not be examined in the current research, but the revealed difference between groups would warrant further examination prospectively in the same clinical context.

Weaker or even non-existent associations have been demonstrated by other authors. Rafeey et al. found an elevated fecal calprotectin in H. pylori-infected children; however, the difference was minor and diagnostic capacity of the biomarker was poor [7]. Demirbas et al. noted that there was an elevated concentration in patients with chronic gastritis, although there was no obvious association with H. pylori and it was rather associated with acute gastritis [8]. Montalto et al. revealed no significant elevation in adults with chronic gastritis, although neutrophils were severely infiltrated [9]. Sykora et al. also reported that H. pylori infection is associated with abdominal pain in children, not with calprotectin [13].

Such variations among studies might be due to biological and methodological differences. Fecal calprotectin is based on the presence of neutrophil protein in the stool, not a particular organism. Concentration will depend on inflammatory activity of stomach, duodenum or intestine, platform used for analysis, sample storage, upper level of detection, age, symptoms, and presence of gastrointestinal disease. Those studies where histological grading is included will be able to discriminate better between infection and inflammation activity, while excluding the presence of lower gastrointestinal disease and medications will lead to less significant group differences. It may be assumed, then, that the effect found is a consequence of H. pylori infection, underlying disease, or both.

Calprotectin is secreted mainly by neutrophils and is stable in feces [5]. Inflammation in stomach caused by H. pylori is accompanied by epithelial stimulation and secretion of cytokines followed by neutrophils and mononuclear cell recruitment. More severe inflammation is characterized by greater number of inflammatory cells in the mucosa and will lead to higher calprotectin release from those cells, which is confirmed by findings of Aksoy et al. [6].

The data presently available cannot ascertain that the identified calprotectin is derived from gastric inflammation. The protein secreted in the upper gastrointestinal tract needs to traverse the intestine, and it is possible for H. pylori infection to co-exist with duodenitis, enteritis, changed flora, or inflammatory bowel disease. Fecal calprotectin is therefore an indicator of neutrophil activity in the gastrointestinal tract that occurred more often among positive subjects, but not the quantification of gastric H. pylori infection.

Positive FOBT was found more often among H. pylori-positive compared to H. pylori-negative subjects (12.0% vs. 5.2%). Nevertheless, the former finding was borderline (P=0.051), while no significant association was found when adjusted. As FOB testing is not specific for H. pylori-induced damage, the positive results can be caused by bleeding from different locations in the gastrointestinal tract. In the absence of endoscopy results and precise diagnosis, neither the location nor the significance of positive FOBT can be elucidated.

The positive finding of FOB test should hence be seen as secondary in nature and exploratory in character but not as proof of active H. pylori infection leading to occult gastrointestinal bleeding. Further research involving prospective studies, using endoscopy, ulcer condition, drugs used and retesting following successful elimination of H. pylori infection is required to find out if there is any association between FOB levels and H. pylori infection.

An AUC value of around 0.70 indicates moderate degree of separation and is not sufficient enough for use in diagnostics. While the threshold level of 50 µg/g identified only half of the positive subjects, the higher threshold based on sample values improved specificity while compromising sensitivity. A negative result of fecal calprotectin does not rule out the presence of H. pylori infection and similarly a positive one cannot pinpoint it.

Tests for the presence of H. pylori infection are needed. Guidelines from around the world recommend breath tests, stool tests, and appropriate invasive tests for both diagnosis and confirmation of successful eradication [2-4]. The current result has implications for interpretation rather than diagnosis; H. pylori and upper GI inflammation can be regarded as some of the possibilities where high fecal calprotectin is seen, especially if lower GI examination has not found a cause. Medication use should also be considered, as proton pump inhibitors and non-steroidal anti-inflammatory medications have been linked to high fecal calprotectin without abnormal colonoscopy results [14-15].

The study recruited 268 participants with complete data for H. pylori, fecal calprotectin, age, gender, and FOB. An active infection was determined based on the results of stool antigen test and 13C-urea breath test, which decreased the misclassification associated with the use of non-invasive test alone. The analysis presented distributional characteristics, effect sizes, adjusted estimates, pre-defined biomarker groups, and the results of exploratory ROC.

There are several aspects limiting the interpretation of the study. The recruitment through general practice centers with reference to one private laboratory decreases generalizability and means that the positive proportion found cannot be interpreted as the population prevalence. Cross-sectional design precludes inference regarding temporal relationship and causality. Information on inflammatory bowel disease, acute enteric infections, celiac disease, colorectal disorders, non-steroidal anti-inflammatory drugs, smoking, body mass index, endoscopic results, and gastritis activity was not available for adjustment. The number of patients with age <18 years was too small, and stratified results were descriptive. The four calprotectin values >1000 µg/g were top-coded to 1000 µg/g for numerical presentation. The ROC cut-off point obtained in the sample was not validated externally.

The future prospective studies should maintain the a priori two-test definition of active H. pylori infection, measure fecal biomarkers on the same day, and collect data on the diagnoses and medications systematically. The endoscopic and histological assessments will allow distinguishing between the presence of the infection and the activity of gastritis and accompanying intestinal diseases. Serial fecal calprotectin and FOB tests both before and after eradication of the infection will add more information about changes, reversibility, and origin of increased levels of biomarkers.
In the current clinical practice, fecal calprotectin cannot be considered the marker of lower gastrointestinal disease only. The positive H. pylori infection could cause increased concentrations; however, such evaluation should be performed in the clinical context. Such data do not support screening of H. pylori by fecal calprotectin and do not justify avoiding proper endoscopic and laboratory investigations in case of another gastrointestinal disease suspicion.

5. CONCLUSION

Among the symptomatic patients attending a primary care clinic, the patients positive for H. pylori showed increased levels of fecal calprotectin, along with significantly higher chances of having their levels above 50 µg/g compared with negative patients. However, even with adjustments made for age, sex, and FOB, discrimination and sensitivity were still low, indicating that fecal calprotectin is not suitable for diagnosing H. pylori infections on its own. Future prospective studies are needed to establish the cause-effect relationship.

Ethical Considerations

Written informed consent was obtained from all participants before enrolment. For participants younger than 18 years, consent was provided by a parent or legal guardian. Confidentiality and data privacy were maintained throughout the study, and all procedures followed applicable ethical principles.

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Conflicts of Interest:

The authors declare no potential conflicts of interest.

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