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Helicobacter pylori Stool-Antigen Positivity and Associated Factors Among Adult Outpatients Referred for Testing: A Hospital-Based Cross-Sectional Study

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Abstract

Background/Objective: *Helicobacter pylori* is a gastric pathogen capable of lifelong colonization of the human stomach and is one of the most prevalent chronic bacterial infections worldwide. This study estimated *H. pylori* stool-antigen positivity among adult outpatients referred for testing at a single public hospital and examined clinical, familial, sociodemographic, and environmental characteristics associated with a positive result. Methods: This single-center, hospital-based cross-sectional study included 200 adult outpatients at Ranya Training Hospital who were treated from 1 June to 1 August 2026. Data were collected through structured, face-to-face, interviewer-administered interviews, and stool-antigen positivity was assessed using a qualitative fecal-antigen immunochromatographic assay. Recent use of proton-pump inhibitors, potassium-competitive acid blockers, bismuth compounds, and antibiotics was not recorded. The total number of referrals during the study period was unavailable; 20 of 220 eligible patients who were approached did not complete the study and could not be characterized or compared with participants. Adjusted prevalence ratios (aPRs) were estimated using modified Poisson regression with robust variance. Results: Of the 200 participants, 110 tested positive (55.0%; 95% confidence interval [CI] 48.1–61.7). Positivity was associated with a composite previous gastrointestinal-history item (aPR 1.56, 95% CI 1.22–1.99), self-reported family history of *H. pylori* infection or peptic ulcer disease (aPR 1.54, 95% CI 1.22–1.94), and rural residence (aPR 1.32, 95% CI 1.03–1.68). One contrast between municipal tap water and filtered water was observed (aPR 0.69, 95% CI 0.51–0.94), but this hypothesis-generating finding warrants testing of whether differences in water handling, storage, or source switching contribute to the observed contrast. Conclusions: These findings describe a clinically referred sample, not population-level prevalence. The GI-history item should not be interpreted as an etiologic risk factor. The key limitation was that recent medication exposure was not recorded, which may have affected stool-antigen detection and between-group comparisons; changes in water source after symptom onset were also not recorded.

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Keywords: *Helicobacter pylori*; stool-antigen test; associated factors; family history; drinking water; rural health; gastrointestinal diseases; Iraq; cross-sectional study

1. Introduction

Helicobacter pylori is a gastric pathogen capable of lifelong colonization of the human stomach and is one of the most prevalent chronic bacterial infections worldwide. A systematic review and meta-analysis covering the period of 1980–2022 estimated that the global prevalence decreased from 58.2% in the 1980–1990 period to 43.1% in the 2011–2022 period [1]. Earlier pooled estimates placed the global prevalence at approximately 4.4 billion infected individuals [2]. Persistent colonization drives chronic active gastritis and is causally linked to peptic ulcer disease and gastric adenocarcinoma [3]. Prevalence differs markedly between and within countries and varies with socioeconomic circumstances, sanitation, household crowding, geography, and other environmental exposures [4,5].

The routes of transmission are multiple and not fully established. Familial and household clustering has been consistently described, supporting a role for shared household exposures and person-to-person spread, particularly during childhood [6–9]. Environmental pathways, including exposure through food and water, have also been investigated, and both remain plausible contributors in areas with varying sanitation and water infrastructure [10,11]. Distinguishing these pathways is difficult in observational studies because household membership, socioeconomic status, sanitation, and water source are strongly correlated [12]. The present study did not obtain microbiological samples from food, drinking water, or household contacts; therefore, it could not identify or confirm any transmission route.

Regional studies differ substantially in recruitment, age composition, diagnostic method, and statistical analysis. In Duhok, a hospital-based study of 100 children reported 28% IgG seropositivity [13]. In Sulaimani, 86 patients referred to a specialist gastroenterology center because of dyspepsia were assessed using urea breath testing; 58 underwent esophagogastroduodenoscopy, whereas 28 received the breath test alone [14]. A separate Iraqi study examined symptomatic *H. pylori* infection and associated factors in Diyala Province [15], which included 194 symptomatic outpatients aged 15–70 years and used serum anti-*H. pylori* IgG ELISA, with associations assessed by chi-square and binomial tests. Because this study measured seropositivity rather than stool-antigen positivity, its estimate is not directly comparable with the present study. A later Duhok study included 259 adults who visited hospitals, used an IgG ELISA, and reported 40.0% seropositivity with unadjusted chi-square comparisons [16]. The Kirkuk series included 266 patients of all ages with gastrointestinal complaints or disorders recruited from two hospitals, private clinics, and medical laboratories; it used a rapid lateral-flow stool-antigen assay and reported chi-square and t-test comparisons without multivariable adjustment [17]. The studies differ in population, sampling frame, testing indication, assay, and analysis, so their positivity proportions are not directly comparable. Serology and stool-antigen testing measure different targets: IgG serology detects the host antibody response, which may persist after eradication, whereas a stool-antigen assay detects bacterial antigens in feces [18,19]. The present study adds a local estimate of stool-antigen positivity among adult outpatients referred for testing at a single public hospital in Ranya, together with adjusted association estimates and sensitivity analyses. It does not estimate community prevalence or establish a transmission mechanism. A structured comparison of the present study with the regional reports is provided in Supplementary Table S5.

Literature-guided variable selection was performed in the present study. Previous gastrointestinal disease was designated as the primary explanatory factor because *H. pylori* is closely linked to gastric mucosal inflammation and upper gastrointestinal disorders [20]. Family history was included because of well-documented familial clustering, and drinking-water sources were examined because environmental exposure has repeatedly been proposed as a transmission pathway [10,11]. Sex, body mass index (BMI), place of residence, and other sociodemographic characteristics were examined as covariates. Therefore, this study aimed to estimate the *H. pylori* stool antigen positivity rate among adults referred for testing at the outpatient department of Ranya Training Hospital and to examine the clinical, familial, sociodemographic, and environmental factors associated with a positive test result in this population.

2. Materials and Methods

2.1. Study Design and Setting

A hospital-based analytical cross-sectional study was conducted at the outpatient department of Ranya Training Hospital, Ranya, Kurdistan Region, Iraq, from 1 June to 1 August 2026. Ranya is a district town that serves both urban residents and surrounding rural communities, and the hospital is the district's principal public referral facility.

2.2. Participants, Recruitment and Eligibility

The study population comprised adults who attended the outpatient department and had a clinical indication or physician referral for *H. pylori* stool-antigen testing. Referral decisions were made by the attending outpatient physician on clinical grounds; the study did not impose a uniform symptom combination or severity threshold. The interviewer-administered questionnaire recorded six current gastrointestinal complaints: epigastric pain, heartburn, acid regurgitation, postprandial fullness, nausea, and abdominal distension. These symptoms were study variables, not additional eligibility criteria. The specific indication entered in the laboratory request and its relationship to the recorded symptoms were not retained in the analytical dataset. The research interview was designed to collect study variables; it did not document the clinician's exact reason for referral or whether testing was for initial diagnosis or post-treatment assessment. Objective clinical findings, including endoscopic, histological, or other instrumental gastrointestinal assessments, were not systematically available and were not required for inclusion. Eligible patients were approached for recruitment during the two-month study period. Because total referral volume and daily approach coverage were not retained, recruitment cannot be confirmed as consecutive. Eligibility required age $\geq$ 18 years, a clinical indication or referral for stool-antigen testing, and voluntary informed consent. Individuals younger than 18 years, those unwilling to participate, and those unable to complete the structured interview were excluded from the study.

Previous *H. pylori* infection and eradication treatment were not recorded as separate variables. The available analytical dataset and retained study records did not permit the referral indication or testing context—initial diagnosis versus post-eradication assessment—to be classified retrospectively; therefore, the analyzed sample represents a mixed referral population. To prevent duplicate enrolment, the research team checked the daily outpatient laboratory request register before inclusion and asked each patient whether they had previously participated in the study. Each participant was assigned a unique study identifier and was included only once in the analytical dataset.

Of the 220 eligible patients approached during study data collection, 20 declined to participate or did not complete the study procedures, yielding 200 participants (response rate 90.9%) with complete interview and laboratory data. The available records did not

permit the 20 non-participants to be divided reliably into those who declined and those who did not complete the procedures. No demographic or clinical information was collected from non-participants; therefore, participants and non-participants could not be compared. The study records identified the 220 eligible patients who were approached but did not contain the total number of all patients referred for *H. pylori* testing during the study period, including patients who were not screened or were ineligible. The referring outpatient department, clinical specialty, and individual physician were not retained. Consequently, recruitment coverage among all hospital referrals and department- or physician-specific selection could not be quantified, and the available records did not permit confirmation that every eligible referral on every clinic day was approached. All 200 participants with complete data were included in the analysis, and no observations were excluded because of missing data.

Recent exposure to proton-pump inhibitors, potassium-competitive acid blockers, bismuth compounds, or antibiotics was not captured by the structured questionnaire and was not used as an eligibility criterion for this study. No participant-level medication exposure variables were available in the analytical dataset. These exposures could not be reconstructed retrospectively from the collected questionnaire or analytical dataset. Linkable participant-level medication information was not available from the retained pharmacy or referral records; therefore, recent medication exposure could not be recovered retrospectively. These medications may reduce stool-antigen detection and cause false-negative results. Because their use may differ according to gastrointestinal history, any resulting misclassification cannot be assumed to be non-differential, and its magnitude and direction cannot be quantified from these data. The resulting estimate should be interpreted as the positivity rate within this clinically selected outpatient sample rather than as a population-level prevalence estimate for adults.

2.3. Sample Size Determination

The sample size was calculated using the single-population proportion formula $n = Z^2P(1 - P)/d^2$. Because no sufficiently reliable pre-study estimate was available for this clinically referred outpatient population, $P = 0.50$ was assumed. For a proportion, $P(1 - P)$ is maximized at 0.50; this assumption therefore yields the largest required sample size for the specified confidence level and margin of error. With a 95% confidence level ($Z = 1.96$) and an absolute margin of error of 7%, the minimum required sample was 196 participants. Allowing for approximately 10% non-response or incomplete participation, the recruitment target was approximately 218, rounded to 220. Of the 220 eligible individuals approached, 200 completed the study and were included in the analysis; thus, although the recruitment target was not reached as an analyzed sample, the final sample remained above the pre-specified minimum of 196. This calculation was designed to estimate the stool-antigen positivity proportion with specified precision. It was not an a priori power calculation for the multivariable association model or for detecting predictor-specific prevalence ratios; therefore, the study was not specifically powered to detect prespecified clinically meaningful associations. Regression findings are interpreted using effect estimates and 95% confidence intervals, with wide intervals indicating limited precision.

2.4. Data Collection Instrument and Administration

Data were collected directly from participants through structured, face-to-face interviews conducted by members of the research team in the outpatient department. The questionnaire was interviewer-administered, not self-completed; routine registration data were not used as the source for questionnaire responses. Before recruitment, interviewers received standardized instructions on questionnaire administration, neutral wording, re-

sponse options, accurate recording, and confidentiality. Interviews were completed before stool-antigen results became available, so interviewers were unaware of the laboratory outcome. The questionnaire was reviewed by representatives of the University of Raparin Research Center and Health Assurance Department for linguistic clarity, cultural appropriateness, and comprehensibility. It was not formally psychometrically validated; therefore, scale-level reliability statistics are not reported. The retained English-language variable labels and analytical codebook are provided in Supplementary File S1.

2.5. Variables and Operational Definitions

Variables were operationalized and coded in IBM SPSS Statistics (version 26.0; IBM Corp., Armonk, NY, USA) according to a standardized codebook.

Outcome. *H. pylori* stool antigen results were coded as follows: 1 = positive and 0 = negative.

Composite previous gastrointestinal (GI) history. The retained English-language codebook labelled this binary item "Previous GI disease" and coded it as follows: 0 = no and 1 = yes. According to the study's operational definition, a value of 1 represented a participant-reported previous physician diagnosis of chronic gastritis, peptic ulcer disease, gastroesophageal reflux, or recurrent dyspeptic episodes. The original verbatim interview prompt and condition-specific responses were not retained; therefore, the exact prompt cannot be reproduced and the individual conditions cannot be analysed separately. No frequency or duration threshold for recurrent dyspeptic episodes was documented in the retained codebook or analytical dataset; consequently, this component could not be standardized retrospectively. The study team did not retrieve or verify the original clinical records, and no endoscopic or histological confirmation was obtained. The questionnaire did not record when a diagnosis was made or whether the condition was active at the time of testing. This variable therefore represents a self-reported lifetime composite history without information on timing.

Self-reported family history (FAMHP). The English translation of the interview question was: "Has any of your first-degree relatives ever been diagnosed with *H. pylori* infection or peptic ulcer disease?" Responses were recorded as yes or no. The two conditions were combined in a single response item and could not be separated in the dataset; consequently, condition-specific counts and their overlap could not be reported. The questionnaire did not record the relative's precise relationship to the participant, whether the relative lived with the participant, or when the diagnosis occurred. The information was based solely on participant reports and was not verified against relatives' medical records. The variable therefore represents a composite self-reported family history and not confirmed household infection.

Self-reported psychological or mood-related history (PSYCH). An exploratory, non-validated variable assessed by self-report, covering diagnosed psychological or mood disorders (for example, generalized anxiety disorder or major depressive episodes) as well as non-specific emotional distress reported by the participant. No validated psychometric instrument was administered, and this variable must not be interpreted as a verified clinical diagnosis. Current clinical symptoms (SYMPT) and symptom count. Active gastrointestinal complaints (epigastric pain, heartburn, acid regurgitation, postprandial fullness, nausea, and abdominal bloating) were each recorded as separate binary items. Symptom_Count is a composite variable defined as the sum of these six indicators (range 0–6) and was examined, together with the additional variables listed below, in the unadjusted exploratory comparisons described in Section 3.4.

Primary drinking water source (WATER). The household's main current daily drinking water source was recorded as follows: 1 = municipal tap, 2 = groundwater or well, or 3 = filtered water. A review of the original dataset and codebook confirmed that the third

category represented filtered water only; the previous inclusion of bottled water in this label was incorrect and has been corrected throughout. This item only captured the main current source. The duration of use, switching source after the onset of symptoms, boiling, transport, and household storage practices were not recorded.

Sociodemographic characteristics: Age (years), sex, place of residence (urban or rural), educational attainment, occupation, household size, and self-rated economic status.

Anthropometric and nutritional status (NUTRI). The measured body mass index (kg/m^2) was classified according to the World Health Organization criteria as follows: 1 = underweight (<18.5), 2 = normal (18.5–24.9), 3 = overweight (25.0–29.9), and 4 = obese (≥30.0).

Lifestyle, dietary, and hygiene practices. Current cigarette smoking; alcohol consumption; usual meal location; consumption frequency of fast food (FAST), junk food (JUNK), and spicy food (SPICY); and hygiene indicators comprising handwashing before and after meals (HANDMEAL), washing of raw vegetables (VEGWASH), and handwashing after toileting (HANDTOIL).

2.6. Laboratory Assessment

Stool specimens were tested using the *H. pylori* Antigen Rapid Test Cassette (Feces), lot HP-2251014 (Weifang Kanghua Biotech Co., Ltd., Weifang, Shandong, China), a qualitative colloidal-gold lateral-flow immunochromatographic assay. The lot was manufactured on 31 October 2025, and had an expiry date of 30 October 2027. In the manufacturer's comparative evaluation of 1200 specimens against another commercially available *H. pylori* antigen rapid test, the positive percent agreement was 99.69% (95% CI 98.89–99.92%), the negative percent agreement was 99.45% (95% CI 98.41–99.81%), and the overall percent agreement was 99.58% (95% CI 99.03–99.82%). These manufacturer-declared agreement measures were not obtained against an independent reference standard and do not establish the diagnostic sensitivity or specificity of this kit in the present population. A Cochrane review reported a pooled stool-antigen sensitivity of 0.83 (95% CI 0.73–0.90) at a fixed specificity of 0.90; this estimate combined different tests and should not be taken as the performance of the present cassette [19]. The assay was not independently validated locally.

The specimens were processed according to the manufacturer's instructions for use (IFU-02-HP-Ag-1.0.2, version 2024.01, C/0). The test components and specimens were brought to room temperature before testing. For solid specimens, the sampling applicator was inserted into approximately six sites to collect approximately 50 mg of feces. For liquid specimens, approximately 50 µL was added to the extraction tube. The tube was thoroughly shaken and left for 2 min. Three drops of the processed specimen (approximately 80–100 µL) were applied to the sample well, and the results were read visually within 15–20 min and not interpreted after 20 min. The simultaneous appearance of the control (C) and test (T) lines was classified as positive, whereas a control line without a test line was classified as negative result. A visible control line served as the cassette's internal procedural control, indicating adequate specimen volume, membrane wicking, and procedural technique. Tests without a visible control line were considered invalid and were repeated using a new cassette. The assay provided a qualitative result and had no numerical positivity cut-off. Laboratory personnel performed and interpreted the stool-antigen tests without access to the participants' questionnaire responses and were therefore unaware of the recorded clinical, behavioral, and environmental information at the time of result interpretation. Participant-level collection-to-testing times and preanalytical storage records were not retained, and separate documentation of external control materials or independent duplicate readings was unavailable. The manufacturer's documentation describes a monoclonal anti-*H. pylori* antibody immobilized on the nitrocellulose membrane and goat anti-mouse IgG in the

control-line system. The instructions specified the storage of unopened test components at 4–30 °C, protection from sunlight, and no freezing; study-specific temperature logs were not retained. All 200 analyzed participants had an interpretable final result, but the frequency of initially invalid cassettes, if any, was not separately recorded.

2.7. Statistical Analysis

Descriptive and bivariate analyses and logistic regression sensitivity analyses were performed using IBM SPSS Statistics for Windows (Version 26.0; IBM Corp., Armonk, NY, USA). The modified Poisson primary analysis was implemented in Python 3.12 using NumPy 2.3.5 and SciPy 1.17.0 by solving the Poisson score equations and calculating an uncorrected HC0 robust sandwich covariance matrix. The primary-model specification and the SPSS syntax used for the logistic-regression sensitivity analyses are provided in Supplementary File S2 to support analytical reproducibility.

HC0 was the estimator implemented for the primary analysis; HC1/HC3 small-sample corrections were not evaluated, so some anticonservatism of confidence intervals cannot be excluded. Categorical variables were summarized as frequencies and percentages, and continuous variables as means with standard deviations (SD). The positivity rate is presented with a 95% confidence interval (CI) calculated using the Wilson score method. Associations between categorical variables and *H. pylori* status were examined using Pearson's chi-square test.

Because stool-antigen positivity was common (55.0%), the main reported multivariable analysis used modified Poisson regression with a log link and HC0 robust sandwich variance to estimate adjusted prevalence ratios (aPRs) with 95% confidence intervals. The model included six predictor terms represented by nine slope parameters: one each for composite previous gastrointestinal history, self-reported family history, place of residence, and sex; two for the three-category drinking-water-source variable; and three for the four-category BMI variable. Together with the intercept, the model therefore contained 10 regression coefficients. No term in the stated model was dropped during model fitting. With 110 positive outcomes, there were approximately 12.2 positive outcomes per slope parameter. This ratio is descriptive and does not by itself establish adequate precision; estimates were interpreted according to their effect sizes and 95% confidence intervals. Composite previous gastrointestinal history and self-reported family history were included as clinical and familial descriptive covariates; drinking-water source and residence represented environmental and geographical characteristics; and sex and BMI category were included as demographic and health-related covariates. Covariates were selected on epidemiological grounds rather than according to univariable statistical significance. Normal BMI was the reference category. Multi-category terms were tested jointly using robust Wald tests. The model was used to estimate associations rather than individual probabilities. Six fitted mean values exceeded 1, which can occur with the modified Poisson approach; these values were not interpreted as predicted probabilities.

A binary logistic regression model containing the same covariates was retained as a sensitivity analysis. Adjusted odds ratios (aORs) and the complete model diagnostics are reported in Supplementary Tables S2 and S6.

The covariate rationale was organized as an explicit conceptual framework. Age, sex, socioeconomic status, education, and household size were considered background demographic and social characteristics; residence and drinking-water source represented geographical and environmental context; family history represented a familial marker; and composite previous gastrointestinal history represented the principal clinical factor. A formal directed acyclic graph was not prespecified. Because the study examined several associations rather than one exposure-specific causal effect, and because the temporal

ordering of gastrointestinal history, referral, and current test positivity was uncertain, a post hoc causal DAG was not used to claim identification of total causal effects. The primary model was kept parsimonious and focused on the principal clinical, familial, environmental, and demographic factors. The original logistic regression specification and three alternative logistic models were retained as sensitivity analyses: a parsimonious model containing composite previous gastrointestinal history, family history, drinking-water source, and residence; an expanded model adding age, household size, and economic status; and an expanded model using underweight/normal versus overweight/obese BMI. Education was not included in the primary model and remained exploratory because its five-category coding included small groups and would have added four indicator parameters. No term in the stated primary model was dropped during model fitting. Full logistic regression estimates with 95% CIs are provided in Supplementary Table S2 and are used only to assess sensitivity to effect measure and model specification.

Additional demographic, lifestyle, dietary, symptom-related, hygiene, and psychological or mood-related variables were examined in unadjusted exploratory analyses. For categorical variables, denominators, outcome counts, positivity percentages, crude odds ratios (ORs) with 95% CIs, and an overall p value are reported in Supplementary Table S4. Pearson's chi-square test was used when the expected cell counts were adequate. Fisher's exact test was used for sparse 2 × 2 tables (alcohol use, washing raw vegetables, and handwashing after toileting), and a Monte Carlo permutation version of the Pearson chi-square test with 500,000 replicates was used for education because one expected count was below five. Continuous variables were compared using Welch's independent-samples t-test and are reported with group means, standard deviations, mean differences, and 95% CIs. These analyses were not adjusted for multiple comparisons and were considered exploratory rather than confirmatory. No formal, dated statistical analysis plan specifying the model hierarchy was prepared before analysis; the terms primary and exploratory describe the analytical hierarchy used in the manuscript and should not be interpreted as preregistration or prospective specification. Reproducibility syntax and the questionnaire codebook are provided in the Supplementary Materials.

2.8. Ethical Considerations

Ethical approval was granted on 1 June 2026 by the University of Raparin Institutional Review Board, through the Research Center and Health Assurance Department, Ranya, Kurdistan Region, Iraq (approval no. UOR-REC/2026/045). Participant recruitment commenced only after approval had been granted and continued until 1 August 2026. Institutional permission was obtained from the Ranya Training Hospital. All participants received an explanation of the study's purpose and procedures and provided voluntary informed consent. Participants were informed that they could decline to participate or withdraw at any time without consequences to their clinical care. All data were handled confidentially and used only for research purposes. This study was conducted in accordance with the Declaration of Helsinki.

3. Results

3.1. Participant Characteristics

The 200 participants had a mean age of 35.34 ± 11.58 years (range 18–68). Females accounted for 58.5% of the sample, and 70.5% of participants lived in urban areas. University education was the most frequent educational category (42.0%), and most participants rated their economic status as sufficient (36.0%) or barely sufficient (52.0%). Normal BMI was the most common nutritional category (56.5%). Current smoking was reported by 10.0% of participants and alcohol use by 1.5%. Full baseline characteristics are presented in Table 1.

Table 1. Baseline characteristics of the 200 adult outpatients included in the analysis.

Characteristic	*n*	%
Age, years, mean ± SD (range)	35.34 ± 11.58 (18–68)	—
Sex: female	117	58.5
Sex: male	83	41.5
Residence: urban	141	70.5
Residence: rural	59	29.5
Education: no formal education	8	4.0
Education: primary	13	6.5
Education: secondary	41	20.5
Education: high school	54	27.0
Education: university	84	42.0
Economic status: sufficient	72	36.0
Economic status: barely sufficient	104	52.0
Economic status: insufficient	24	12.0
BMI: underweight (<18.5 kg/m^2)	11	5.5
BMI: normal (18.5–24.9 kg/m^2)	113	56.5
BMI: overweight (25.0–29.9 kg/m^2)	54	27.0
BMI: obese (≥30.0 kg/m^2)	22	11.0
Current smoker	20	10.0
Alcohol use	3	1.5

BMI, body mass index. Except for age, *n* is the number of participants in the category and percentages use all 200 analyzed participants as the denominator.

3.2. Stool-Antigen Positivity Rate and Bivariate Associations

Among the 200 adult outpatients referred for testing, 110 had a positive *H. pylori* stool antigen result and 90 had a negative result, corresponding to a positivity proportion of 55.0% (95% CI 48.1–61.7). A history of gastrointestinal disease was reported in 67 participants (33.5%). Positivity was 73.1% among those with such a history compared to 45.9% among those without ($\chi^2(1) = 13.39$, $p < 0.001$). Positivity also differed by drinking-water source ($\chi^2(2) = 6.15$, $p = 0.046$): 43 of 94 municipal tap-water users (45.7%), 42 of 66 well- or groundwater users (63.6%), and 25 of 40 filtered-water users (62.5%) tested positive for *H. pylori*. Drinking-water source was not associated with place of residence ($\chi^2(2) = 1.07$, $p = 0.586$); the complete cross-tabulation is provided in Supplementary Table S3. Neither sex ($\chi^2(1) = 1.58$, $p = 0.210$) nor BMI category ($p = 0.822$) was associated with *H. pylori* status in the unadjusted analyses, and rural residence was not significant before adjustment ($p = 0.156$). The bivariate results for all primary factors, including residence and BMI, are shown in Table 2, which also provides category denominators and Wilson 95% CIs for the positivity proportions.

Table 2. Bivariate associations between primary factors and *H. pylori* stool-antigen status among adult outpatients referred for testing.

Primary Factor	Negative, *n* (%)	Positive, *n*/N (%; 95% CI)	Crude PR (95% CI)	*p* Value
Composite previous GI history: no	72 (54.1)	61/133 (45.9; 37.6–54.3)	Reference	<0.001
Composite previous GI history: yes	18 (26.9)	49/67 (73.1; 61.5–82.3)	1.59 (1.26–2.02)	
Composite family history: no	71 (52.6)	64/135 (47.4; 39.2–55.8)	Reference	0.002
Composite family history: yes	19 (29.2)	46/65 (70.8; 58.8–80.4)	1.49 (1.18–1.89)	
Drinking water: municipal tap	51 (54.3)	43/94 (45.7; 36.0–55.8)	0.73 (0.53–1.01)	0.046
Drinking water: well	24 (36.4)	42/66 (63.6; 51.6–74.2)	1.02 (0.75–1.38)	
Drinking water: filtered	15 (37.5)	25/40 (62.5; 47.0–75.8)	Reference	
Sex: female	57 (48.7)	60/117 (51.3; 42.3–60.2)	Reference	0.210
Sex: male	33 (39.8)	50/83 (60.2; 49.5–70.1)	1.17 (0.92–1.51)	
Residence: urban	68 (48.2)	73/141 (51.8; 43.6–59.9)	Reference	0.156
Residence: rural	22 (37.3)	37/59 (62.7; 50.0–73.9)	1.21 (0.94–1.56)	

Table 2. *Cont.*

Primary Factor	Negative, *n* (%)	Positive, *n*/N (%; 95% CI)	Crude PR (95% CI)	*p* Value
BMI: underweight	6 (54.5)	5/11 (45.5; 21.3–72.0)	0.79 (0.41–1.54)	0.822
BMI: normal	48 (42.5)	65/113 (57.5; 48.3–66.2)	Reference	
BMI: overweight	26 (48.1)	28/54 (51.9; 38.9–64.6)	0.90 (0.67–1.22)	
BMI: obese	10 (45.5)	12/22 (54.5; 34.7–73.1)	0.95 (0.63–1.43)	

BMI, body mass index; CI, confidence interval; GI, gastrointestinal; PR, prevalence ratio. Negative and positive percentages are row percentages within each category. The positive column additionally provides the category denominator and Wilson 95% CI for the positivity proportion. Crude PRs are unadjusted and use no composite previous GI history, no composite family history, filtered water, female sex, urban residence, and normal BMI as the reference categories. Blank *p*-value cells indicate that the value shown on the first row applies to the overall comparison for that factor. The *p* values are from Pearson's chi-square tests. Composite family history denotes the self-reported family history of *H. pylori* infection or peptic ulcer disease in a first-degree relative.

3.3. Primary Modified Poisson Regression

The primary modified Poisson model converged after six Newton–Raphson iterations, and all coefficients and robust standard errors were finite. Every categorical level contained both positive and negative outcomes; complete outcome counts are provided in Supplementary Table S1. The drinking-water-source term was associated with positivity as a whole (robust Wald $\chi^2(2) = 6.39$, $p = 0.041$), whereas the overall BMI term was not (robust Wald $\chi^2(3) = 0.31$, $p = 0.958$). The logistic-regression sensitivity model converged without evidence of complete or quasi-complete separation and produced associations in the same direction. Its descriptive fit, discrimination, multicollinearity, and influence diagnostics are reported in Supplementary Table S6 and are not interpreted as predictive validation.

In the primary modified Poisson model, the composite previous gastrointestinal-history item was associated with higher prevalence of *H. pylori* positivity (aPR 1.56, 95% CI 1.22–1.99; $p < 0.001$), and the self-reported composite family-history variable was associated with higher prevalence (aPR 1.54, 95% CI 1.22–1.94; $p < 0.001$). Rural residence was associated with higher prevalence than urban residence (aPR 1.32, 95% CI 1.03–1.68; $p = 0.026$). The three-category water-source term was significant as a whole (robust Wald $\chi^2(2) = 6.39$, $p = 0.041$). In one pairwise contrast, municipal tap-water use had lower estimated prevalence than filtered-water use (aPR 0.69, 95% CI 0.51–0.94; $p = 0.017$); the well- or groundwater comparison was not statistically significant (aPR 0.91, 95% CI 0.68–1.21; $p = 0.517$). This single contrast warrants testing whether differences in water handling, storage, or source switching contribute to the observed association; water exposure was measured only as the main current source, without duration or source changes after symptom onset. It does not establish that municipal tap water is protective or differs from every alternative source. No statistically significant association was detected for sex (aPR 1.24, 95% CI 0.98–1.57; $p = 0.069$) or BMI category. Full estimates are given in Table 3 and displayed in Figure 1.

The crude prevalence ratio for rural versus urban residence was 1.21 (95% CI: 0.94–1.56). The corresponding adjusted prevalence ratio was 1.32 (95% CI 1.03–1.68; $p = 0.026$) in the primary modified Poisson model. The magnitude changed only modestly, and the confidence intervals remained compatible with considerable uncertainties.

Within the retained logistic regression sensitivity analyses, the odds-ratio estimates were broadly similar across alternative covariate specifications (Supplementary Table S2). As expected for a common outcome, their numerical magnitudes were larger than the adjusted prevalence ratios from the primary modified Poisson model and were not interpreted as prevalence ratios. In the parsimonious logistic model, the adjusted odds ratios (aORs) were 3.40 (95% CI 1.72–6.75) for composite previous gastrointestinal history, 3.04 (95% CI 1.54–5.99) for family history, 2.02 (95% CI 1.02–4.00) for rural residence, and 0.40 (95% CI 0.18–0.90) for municipal tap water compared with filtered water.

Table 3. Modified Poisson regression with robust variance for factors associated with *H. pylori* stool-antigen positivity.

Predictor (Comparison)	aPR	95% CI	*p* Value
Composite previous GI history: yes vs. no	1.56	1.22–1.99	<0.001
Composite family history: yes vs. no	1.54	1.22–1.94	<0.001
Residence: rural vs. urban	1.32	1.03–1.68	0.026
Drinking water: tap vs. filtered	0.69	0.51–0.94	0.017
Drinking water: well vs. filtered	0.91	0.68–1.21	0.517
Sex: male vs. female	1.24	0.98–1.57	0.069
BMI: underweight vs. normal	0.85	0.47–1.53	0.586
BMI: overweight vs. normal	0.98	0.74–1.30	0.877
BMI: obese vs. normal	0.97	0.63–1.48	0.873

aPR, adjusted prevalence ratio; BMI, body mass index; CI, confidence interval; GI, gastrointestinal. Normal BMI was the reference category. The model used a log link and HC0 robust sandwich variance. Each estimate was adjusted for all other variables in the model. The overall robust Wald tests were $\chi^2(2) = 6.39$, $p = 0.041$ for drinking-water source and $\chi^2(3) = 0.31$, $p = 0.958$ for BMI. Composite family history denotes the self-reported family history of *H. pylori* infection or peptic ulcer disease in a first-degree relative.

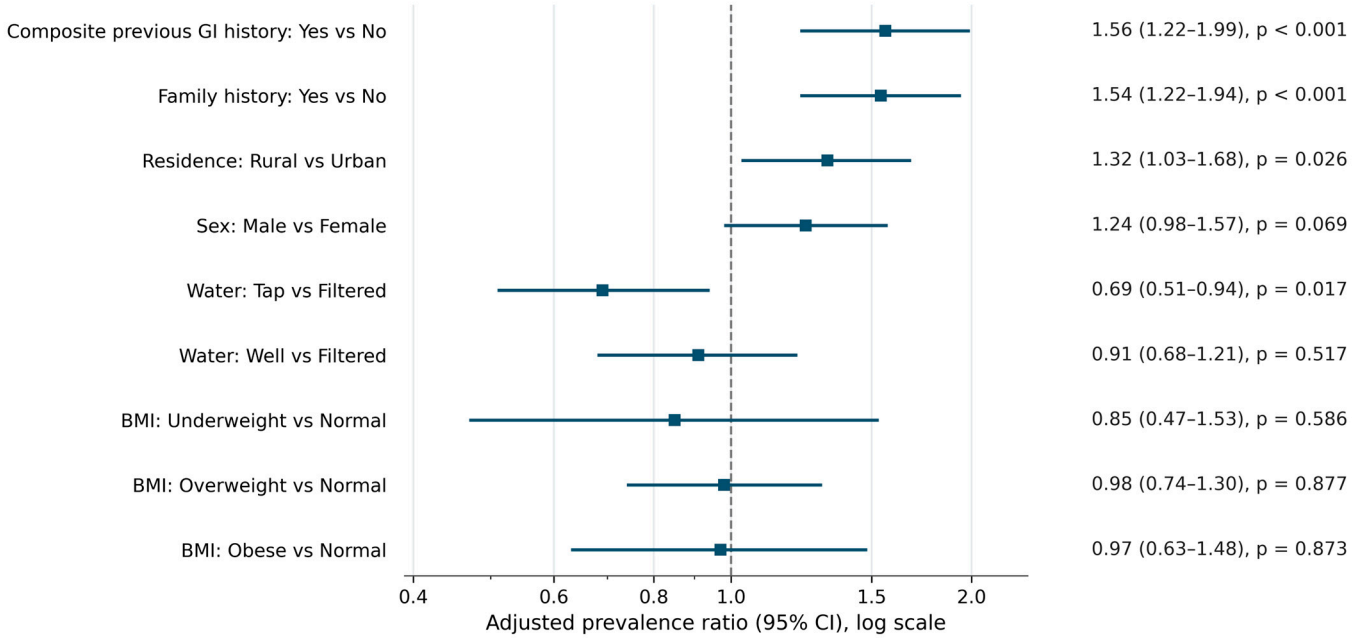


Figure 1. Adjusted prevalence ratios with 95% confidence intervals from the primary modified Poisson regression model for *H. pylori* stool-antigen positivity. The *x*-axis is on a logarithmic scale with decimal tick labels, and the vertical dashed line marks the null value (aPR = 1). Reference categories were filtered water for water-source contrasts, normal BMI for BMI contrasts, female sex, and urban residence. Family history denotes the self-reported family history of *H. pylori* infection or peptic ulcer disease in a first-degree relative.

After age, household size, and economic status were added to the expanded logistic regression sensitivity model, the estimates remained similar for composite previous gastrointestinal history (aOR 3.49, 95% CI 1.73–7.06), family history (aOR 3.15, 95% CI 1.56–6.37), rural residence (aOR 2.04, 95% CI 1.01–4.15), and municipal tap water compared with filtered water (aOR 0.38, 95% CI 0.16–0.90). No statistically significant association was detected for age per 10-year increase (aOR 1.15, 95% CI 0.87–1.53), household size per additional household member (aOR 0.94, 95% CI 0.78–1.14), or economic status. When BMI was collapsed to underweight/normal versus overweight/obese, the principal estimates again changed little: composite previous gastrointestinal history aOR 3.52 (95% CI 1.75–7.10), family history aOR 3.13 (95% CI 1.55–6.31), rural residence aOR 1.99 (95% CI

0.99–3.99), and municipal tap water aOR 0.38 (95% CI 0.16–0.88). No statistically significant association was detected for the simplified BMI term (aOR 0.83, 95% CI 0.43–1.61; $p = 0.589$). Thus, collapsing the small BMI categories did not materially alter the principal exposure estimates, although the residence estimate remained imprecise.

3.4. Additional Exploratory Comparisons

Unadjusted exploratory comparisons are reported in Supplementary Table S4, including denominators, outcome counts or group summaries, effect estimates, 95% CIs, and *p*-values. The symptom count was higher among stool-antigen-positive participants than among negative participants (mean ± SD, 2.88 ± 1.64 versus 2.11 ± 1.61; mean difference 0.77, 95% CI 0.32–1.23; Welch $p = 0.001$). Other exploratory estimates, including the non-validated self-reported psychological or mood-related variables, are retained in Supplementary Table S4 for transparency but are not emphasized. These analyses were unadjusted, were not corrected for multiple comparisons, and were intended to identify patterns requiring confirmation in independent studies. Alcohol use was reported by only three participants, making this comparison too imprecise for meaningful interpretation; the detailed Fisher's exact-test estimate is retained in Supplementary Table S4.

4. Discussion

4.1. Principal Findings

In this hospital-based sample, 55.0% of adult outpatients referred for testing had a positive *H. pylori* stool-antigen result. The adjusted analyses identified associations with the composite previous GI-history item, self-reported family history, rural residence, and one water-source contrast. The GI-history item is heterogeneous, its timing is unknown, and it may reflect clinical referral selection or manifestations of infection; it should not be interpreted as an etiologic risk factor. The water-source contrast was exploratory. Other questionnaire variables were examined only in unadjusted exploratory analyses and are not considered confirmatory.

The 55.0% positivity estimate is a local result for adult outpatients referred for testing at Ranya Training Hospital, not an estimate of population prevalence. The Duhok child study reported IgG seropositivity in 100 children [13], while the Sulaimani study assessed patients referred for dyspepsia using urea breath testing, with endoscopy in a subset [14]. A separate study concerned symptomatic *H. pylori* infection in Diyala [15]. The Duhok adult study included 259 hospital attendees and used IgG serology [16]. The Kirkuk stool-antigen study included children and adults recruited through hospitals, clinics, and laboratories and did not report multivariable adjustment [17]. These studies differed in sampling frame, age composition, clinical indication, assay, calendar period, and analysis; their estimates therefore should not be compared as if derived from a common population. The present study adds descriptive and adjusted association data from Ranya but does not demonstrate a transmission mechanism or support broad regional generalization.

4.2. Composite Previous Gastrointestinal History

The composite previous-GI-history item had the largest point estimate in the primary model, but it should be treated as a heterogeneous descriptive covariate rather than an etiologic risk factor. Chronic gastritis, peptic ulcer disease, gastroesophageal reflux, and recurrent dyspeptic episodes are clinically distinct, and their individual contributions cannot be separated. Some may be manifestations or consequences of *H. pylori* infection rather than factors that preceded infection [21]. The timing of the reported history is unknown, and the item may also reflect referral selection. The estimate applies only to the combined

self-reported history and does not show that gastrointestinal disease increases the risk of acquiring *H. pylori*.

However, this association should be interpreted with caution. Gastrointestinal history was a self-reported lifetime history of a previous physician diagnosis and was not independently verified; its timing and activity at the time of stool testing were unknown. In addition, initial diagnostic testing could not be distinguished from post-treatment tests. Therefore, the observed association describes a pattern within a clinically selected, mixed referral population and does not establish whether the gastrointestinal condition preceded the *H. pylori* stool antigen result or resulted from infection.

Nevertheless, the direction of this association requires careful interpretation within the clinical referral pathway. A previous gastrointestinal diagnosis and recurrent gastrointestinal symptoms may themselves increase the likelihood of referral for *H. pylori* testing. Because the analysis was restricted to referred patients, selection arising from conditioning on referral may have created or amplified the observed association. The adjusted prevalence ratio therefore represents a mutually adjusted association within this clinically selected sample and should not be interpreted as an independent epidemiological risk relationship in the source population. This finding may reflect a combination of underlying disease history, current clinical presentation, and the process leading to testing. In addition, the cross-sectional design cannot establish whether GI disease preceded infection or arose as a consequence of it, and the history variable was based on participant reports of a previous physician diagnosis without verification of the original records, timing, or current activity.

4.3. Family History

The composite self-reported family-history variable—*H. pylori* infection or peptic ulcer disease in a first-degree relative—was associated with positivity in crude and adjusted analyses. However, the two conditions could not be separated, relatives' diagnoses were not verified, and neither co-residence nor diagnosis timing was recorded. Peptic ulcer disease is not specific to *H. pylori* and may also reflect NSAID use, smoking, or other causes. Therefore, this variable cannot be interpreted as confirmed infection among household members.

Although familial clustering of *H. pylori* infection has been described elsewhere [6–9], the present study provides no direct evidence of transmission. This may reflect shared environmental, socioeconomic, dietary, water, sanitation, or healthcare-related factors, prolonged interpersonal contact, or recall and reporting biases. Studies using verified diagnoses, paired testing of participants and relatives, information on co-residence and timing, and molecular strain comparison are required to evaluate household transmission.

4.4. Drinking-Water Source

The three-category drinking water source term was associated with *H. pylori* status in the primary model (robust Wald $\chi^2(2) = 6.39$, $p = 0.041$); however, the individual contrasts require cautious interpretation. Municipal tap water users had a lower adjusted prevalence than filtered water users, whereas the comparison between well water or groundwater and the reference category was not statistically significant. Thus, the result is driven by one contrast and does not show that municipal tap water differs from every alternative source, much less that it is microbiologically safer. Water source was not associated with residence ($\chi^2(2) = 1.07$, $p = 0.586$; Supplementary Table S3); however, residual confounding by unmeasured household and neighborhood conditions remains possible. No microbiological testing was performed on the water samples.

The interpretation of our results is further limited by the exposure measurements. A review of the original dataset and codebook confirmed that the third category represented filtered water only; the earlier reference to bottled water was a manuscript labeling error and

has been corrected. The questionnaire recorded only the main current source and did not capture the duration of use, changes after the onset of symptoms, boiling, transportation, or storage. Consequently, this variable describes a nominal current source rather than a detailed measure of water exposure. Reverse causation is plausible if participants changed their water sources because of previous gastrointestinal symptoms or diagnoses; however, this possibility cannot be examined with the available data. Water sources may also act as markers of socioeconomic status, housing, and local infrastructure. Environmental studies have detected *H. pylori* DNA in water-related samples [10,11,22], but such findings do not establish the viability, infectivity, or transmission pathway in the present population. Therefore, our results warrant testing whether differences in water handling, storage, source switching, or related socioeconomic conditions account for the association; they should not be interpreted as evidence of the microbiological quality or safety of any water source.

4.5. Residence, Sex and Body Mass Index

The crude prevalence ratio for rural versus urban residence was 1.21 (95% CI 0.94–1.56), whereas the adjusted prevalence ratio was 1.32 (95% CI 1.03–1.68). Although the point estimate increased modestly after adjustment, the confidence intervals indicated uncertainty. Therefore, the results suggest an association between rural residence and stool antigen positivity within this clinically selected sample but do not establish a robust or causal effect. The change between the crude and adjusted estimates should not be interpreted solely based on whether the corresponding *p*-values were above or below 0.05.

Rural residence may represent correlated characteristics such as household environment, sanitation, water access, and socioeconomic circumstances. It may also act as a proxy for access to testing: rural patients may present later or be referred when symptoms are more severe. This referral-selection mechanism is distinct from environmental exposure and may contribute to the observed association among patients who were tested. Although age, household size, and economic status were included in an expanded sensitivity model, residual confounding by these and other incompletely measured characteristics remains possible.

No statistically significant association was detected between sex and BMI values. The adjusted prevalence ratio for males was 1.24 (95% CI 0.98–1.57), and the BMI estimates were imprecise, including an aPR of 0.85 (95% CI 0.47–1.53) for underweight compared with normal BMI. Thus, the results are compatible with both no association and potentially meaningful associations in either direction and should not be interpreted as evidence of absence. Male sex has been associated with infection in some studies, including an adult survey from Duhok Province [16], whereas findings regarding lifestyle and metabolic factors have varied across populations [23–26]. Larger studies with adequately represented BMI categories are required for more precise estimation.

4.6. Exploratory Observations

The complete unadjusted exploratory results are presented in Supplementary Table S4. These analyses involved multiple unadjusted comparisons and were exploratory only. The higher symptom count among test-positive participants may reflect referral selection because symptoms preceded testing and may have prompted referral; it should not be interpreted as a measure of disease severity or as evidence of a causal relationship. The self-reported psychological or mood-related variable was not measured with a validated instrument and is retained in the Supplementary Materials for transparency, but is not interpreted further. None of the exploratory findings support a causal inference.

4.7. Strengths and Limitations

The principal strengths are complete data for all 200 analyzed participants, use of a noninvasive qualitative fecal-antigen assay, presentation of crude and adjusted associations, separation of the primary model from unadjusted exploratory comparisons, clear denominators and confidence intervals, and sensitivity analyses using alternative model specifications. The study provides recent stool-antigen positivity data from an underreported outpatient setting in Ranya. The response rate was 90.9% among the 220 eligible patients approached. Because total referral volume was unavailable and recruitment coverage could not be verified, the manuscript does not characterize recruitment as consecutive. No formal dated statistical analysis plan was prepared before analysis; the primary/exploratory terminology should not be interpreted as preregistration.

Several limitations apply. First, the cross-sectional design precludes any assessment of temporality and, therefore, any causal inference. Second, the participants were recruited from a single outpatient clinic and were referred for testing on clinical grounds; therefore, the positivity rate cannot be generalized to the adult population of Ranya or the wider Kurdistan Region. Previous gastrointestinal diagnoses and symptoms may have influenced entry into the referral pathway; conditioning the analysis on referral may therefore have created or amplified the association involving gastrointestinal history. The study cannot separate an underlying epidemiological relationship from the clinical process leading to testing. Third, drinking-water source, composite family-history variable, and psychological history were self-reported and were not independently verified. Duration of water-source use, switching after symptoms, boiling, transport, and storage were not recorded; no water samples were examined microbiologically. Family history combined *H. pylori* infection and peptic ulcer disease in first-degree relatives, without information on the specific condition, overlap, co-residence, or timing. Fourth, a major measurement limitation is that recent use of proton-pump inhibitors, potassium-competitive acid blockers, bismuth, or antibiotics was not recorded and no participant-level medication information could be recovered. These exposures may reduce stool-antigen detection and produce false-negative results [27–29], potentially lowering the observed positivity rate by an unknown amount. If medication use differed according to gastrointestinal history or other participant characteristics, the associations may also have been affected by differential misclassification, with an unknown magnitude and direction of bias. Fifth, the assay was not independently validated in this population.

The manufacturer's performance figures describe agreement with another commercially available *H. pylori* antigen rapid test rather than diagnostic accuracy against an independent reference standard. They therefore cannot establish how this kit performed in the present sample, and the difference between observed stool-antigen positivity and true infection status is unknown in both magnitude and direction. A Cochrane review reported pooled stool-antigen sensitivity of 0.83 (95% CI 0.73–0.90) at a fixed specificity of 0.90, but pooled different tests and cannot establish the performance of this cassette [19]. The primary model used the uncorrected HC0 robust variance estimator; because HC1/HC3 corrections were not evaluated, small-sample anticonservatism cannot be excluded. Although age, household size, and self-reported economic status were included in an expanded sensitivity model, these characteristics may not fully capture socioeconomic conditions, household crowding, sanitation, or healthcare-access factors. Residual confounding therefore remains possible. The residence estimate was imprecise, and the underweight and obese BMI categories were small; the BMI-specific estimates cannot exclude potentially meaningful associations. Several exploratory variables also had small or imbalanced categories and should be interpreted cautiously.

Recruitment was limited to a two-month period; therefore, the study could not assess possible seasonal or other temporal variations in the composition of referrals or stool-antigen positivity. In addition, education was examined only in the exploratory analyses and was not included in the primary or expanded multivariable models; residual confounding by education may therefore remain despite the sensitivity analysis incorporating age, household size, and economic status. The sample size calculation addressed the precision of the positivity proportion rather than statistical power for the multivariate associations. Consequently, some association estimates, particularly those based on small categories, are imprecise, as reflected in their 95% confidence intervals; non-significant results do not establish the absence of potentially meaningful associations.

Although structured administration and interviewer masking of the laboratory results reduced the potential for differential questioning according to test status, the questionnaire was not formally validated. Self-reported psychological history, economic status, hygiene practices, and dietary behaviors are susceptible to recall, reporting, and social desirability bias, and measurement errors in these variables cannot be quantified. Testing performed for initial diagnosis could not be distinguished from testing performed after eradication therapy, and previous *H. pylori* infection and eradication treatment were not recorded as separate variables. The gastrointestinal history variable was based on participants' reports of a previous physician diagnosis without record verification or timing. In addition, the available records did not permit the 20 non-participants to be separated by the reason for non-participation. These limitations restrict the interpretation of the gastrointestinal history association and the assessment of participation bias.

4.8. Implications

The findings support the recording of previous gastrointestinal diagnoses, the composite reported family history, and relevant household and environmental characteristics during clinical assessment. However, they do not establish that infection was transmitted within families or show that testing relatives improves diagnosis, eradication success, symptoms, or other clinical outcomes. Because no relatives were tested and no clinical strategy was compared, family-centred testing remains a proposal for future evaluation rather than a recommendation arising from this study. Likewise, the water-source association should motivate better exposure measurement and direct microbiological investigation rather than attribution of infection to a particular source. Community-based probability samples are required to estimate population prevalence, while paired household studies or comparative clinical studies would be needed to evaluate household transmission and the potential benefits and harms of family-centred testing. No new intervention trial is required to support the present observational report, provided that its conclusions remain limited to the associations observed in the referred outpatient sample.

5. Conclusions

Among adult outpatients referred for *H. pylori* testing at Ranya Training Hospital, 55.0% had a positive stool-antigen result. The adjusted analysis identified associations involving the composite previous GI-history item, self-reported family history, and rural residence; the GI-history item is heterogeneous, has unknown timing, and may reflect referral selection rather than an etiologic risk factor. One drinking-water-source contrast was observed, but it is hypothesis-generating: it warrants testing whether differences in water handling, storage, or source switching account for the observed contrast and should not be interpreted as evidence of source safety or a transmission route. These findings describe a clinically selected sample and cannot be extrapolated to the general adult population of Ranya. Recent medication exposure and changes in water source after symptom onset were not recorded.

Community-based studies with probability sampling, medication assessment, detailed water-exposure measures, and direct environmental testing are needed.

Supplementary Materials: The following supporting information can be downloaded at https://www.mdpi.com/article/10.3390/diseases14110417/s1, Table S1: Outcome counts within each category of the primary model variables; Table S2: Logistic-regression sensitivity analyses of factors associated with *H. pylori* stool-antigen positivity ($n = 200$); Table S3: Joint distribution of primary drinking-water source and place of residence ($n = 200$); Table S4A: Unadjusted exploratory categorical comparisons with *H. pylori* stool-antigen status ($n = 200$); Table S4B: Unadjusted exploratory continuous-variable comparisons with *H. pylori* stool-antigen status ($n = 200$); Table S5: Design features of the present study and regional Iraqi reports discussed in the manuscript; Table S6: Descriptive diagnostics for the multivariable logistic-regression sensitivity model ($n = 200$); File S1: English-language variable labels and analytical codebook; File S2: Reproducibility specification for the primary model and syntax for logistic-regression sensitivity analyses.

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Institutional Review Board Statement: The study was conducted in accordance with the Declaration of Helsinki and approved by the University of Raparin Institutional Review Board through the Research Center and Health Assurance Department, Ranya, Kurdistan Region, Iraq (approval no. UOR-REC/2026/045), on 1 June 2026, before participant recruitment commenced later that day. Institutional permission was obtained from Ranya Training Hospital.

Informed Consent Statement: Informed consent was obtained from all the participants involved in the study. All participants were informed of the study's purpose and procedures and provided informed consent. Participation was voluntary, and participants could decline or withdraw at any time without any consequences.

Data Availability Statement: The de-identified participant-level dataset is not publicly available because the combination of demographic, clinical, and residential variables in this small, single-centre sample may permit deductive re-identification. The data may be made available by the corresponding author upon reasonable request and subject to an appropriate data-use agreement. Requests should include a clearly defined research question and a statistical analysis plan, and any approved use must preserve participant confidentiality.

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Conflicts of Interest: The authors declare no conflicts of interest.

Abbreviations

The following abbreviations are used in this manuscript:

aPR	Adjusted prevalence ratio
BMI	Body mass index
CI	Confidence interval

GI	Gastrointestinal
H. pylori	*Helicobacter pylori*
IgG	Immunoglobulin G
OR	Odds ratio
PPI	Proton-pump inhibitor
SD	Standard deviation
SPSS	Statistical Package for the Social Sciences
WHO	World Health Organization

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