BMC Gastroenterology

RESEARCH

Open Access

Prevalence of H. Pylori in inflammatory bowel disease patients and its association with severity

Abdullah D. Alotaibi[1,2*], Abdullah A. Al-Abdulwahab[1,2], Mona H. Ismail[1,2], Jaber M. AlElyani[1,2], Turki A. Alamri[1,2], Raed M. Alsulaiman[1,2], Ibrahim A. Alhafid[1,2], Ibrahim M. Alzahrani[1,2], Reem S. AlSulaiman[1,2*], Arwa Althubaity[1,2], Sarah H. Buhulaigah[1,2], Abdulaziz A. AlQurain[1,2] and Abdulaziz M. Alrezuk[1,2]

Abstract

Introduction One key area of interest in gastroenterology research is the relationship between Helicobacter pylori (H. pylori) and Inflammatory bowel disease (IBD). Several studies have shown varying results regarding the prevalence of H. pylori in IBD patients and its impact on disease progression, severity, and overall outcome.

Method This is a prospective cohort study conducted at King Fahad University Hospital in Al Khobar, Saudi Arabia from November 2023 to May 2024 to determine the prevalence of H. pylori in IBD patients and its association with severity. The study included 2 arms for comparison which are IBD patients and control group, IBD will be further classified to CD and UC. Prevalence of H. pylori infection and severity of the disease was compared between these groups.

Results A total of 360 patients were included in the study which were divided equally into IBD group and control group. The IBD was subdivided into CD with 91 cases and UC with 89 cases. H. Pylori was significantly higher in control group (23.3%) compared with UC cases (13.2%) p value: 0.048. H. pylori infection was significantly high in smokers p value = < 0.0001. The presence of autoimmune disease was significantly associated with H. Pylori infection (16.4%) p value: 0.023.

Conclusion H. pylori infection was significantly higher in the control group in comparison to IBD group. In addition, smoking and autoimmune disease were significantly associated with H. pylori infection. Finally, the overall association between severity of CD, UC and medication use with H. Pylori were insignificant.

Keywords Crohn's disease, Ulcerative colitis, Helicobacter pylori infection, Prevalence

*Correspondence:
Abdullah D. Alotaibi
adotaibi@iau.edu.sa
Reem S. AlSulaiman
rsolaiman615@gmail.com
[1]Department of Internal Medicine, College of Medicine, Imam Abdulrahman Bin Faisal university, Dammam, Eastern Province, Saudi Arabia
[2]King Fahad University Hospital, Shura Street, Al Aqrabiyah, Al Khobar 34445, Saudi Arabia

Introduction

Inflammatory bowel disease (IBD), which includes Crohn's disease (CD) and ulcerative colitis (UC), is characterized by periods of flare-ups followed by remission. [1–2] The unpredictable nature of IBD necessitates ongoing monitoring and management to prevent long-term complications. [3–4] Research suggests that both genetic predisposition and environmental factors contribute to the development of IBD [2–5]. Understanding these factors is critical not only for prevention but also for individualizing treatment plans based on a patient's unique background.

One key area of interest in gastroenterology research is the relationship between Helicobacter pylori (H. pylori) and IBD. Several studies have shown varying results regarding the prevalence of H. pylori in IBD patients and its impact on disease progression, severity, and overall outcome.

Interestingly, a specific case-control study demonstrated that H. pylori infection rates were considerably lower in UC patients compared to a control group. This observation suggests that H. pylori may play a protective role against UC development [6]. The precise influence of H. pylori on IBD as a whole remains an area of uncertainty [7–9]. Additionally, several studies have suggested a potential protective effect of H. pylori infection against the development of IBD [10–12]. However, a case-control study concluded that H. pylori eradication therapy was not associated with an increased risk of developing IBD [13]. The findings highlight the complexity of the relationship between H. pylori infection and IBD, indicating that further research is necessary to clarify this association [9]. Nonetheless, several nuances in this relationship are yet to be fully explored, especially the impact of H. pylori infection on IBD severity. Consequently, the primary aim of our study was to delve into the prevalence of H. pylori in IBD patients and discern its correlation with the disease's severity.

To our knowledge, no studies have been conducted in Saudi Arabia or the Gulf region on the prevalence of H. pylori infection in IBD patients and its impact on disease severity. Therefore, this study aims to investigate the prevalence of H. pylori in IBD patients and examine its potential correlation with disease severity Using CADAI and Myo score [14, 15].

Methods

Study design and setting

This study was a prospective observational cohort study conducted at King Fahad University Hospital (KFHU) in Al-Khobar, Saudi Arabia, from November 2023 to May 2024. The study aimed to determine the prevalence of Helicobacter pylori (H. pylori) infection among patients with inflammatory bowel disease (IBD) and its association with disease severity. The study was conducted following the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines to ensure transparency and methodological rigor. [16 Cuschieri S. The strobe guidelines. Saudi Journal of Anaesthesia. 2019;13(5):31. doi:https://doi.org/10.4103/sja.sja_543_18]

Study population and eligibility criteria

Inclusion criteria

1. Adults aged ≥ 18 years.
2. Confirmed diagnosis of IBD (Crohn's disease [CD] or ulcerative colitis [UC]) based on clinical, radiological, and histopathological findings.
3. Patients attending the Gastroenterology (GI) clinic at KFHU.
4. Availability of stool samples for *H. pylori* testing.

Exclusion criteria

1. Individuals younger than 18 years.
2. Pregnant women.
3. Patients with malignancy or prior gastrointestinal surgery.
4. Use of proton pump inhibitors (PPIs), antibiotics, or bismuth compounds within four weeks before testing.
5. Patients who declined to participate.

Study groups

Participants were classified into two main groups:

1. IBD Group: Included patients diagnosed with Crohn's disease (CD) or ulcerative colitis (UC).
2. Control Group: Included individuals without gastrointestinal disorders, matched for age and sex, attending other outpatient clinics at KFHU.

The prevalence of H.pylori infection and its association between smoking and autoimmune diseases were compared between these two groups.

Data collection and variables

Data were collected from electronic medical records and structured patient interviews. The following variables were included:

- Demographics: Age, gender, marital status, education level.
- Clinical Characteristics:
  - IBD subtype (CD/UC).
  - Disease duration.

  - Medication use (5-ASA, steroids, azathioprine, biologics).
  - Smoking status (non-smoker, current smoker, ex-smoker).
  - Presence of autoimmune diseases.

- H. pylori Status: Determined using the Foresight EIA stool antigen test.
- Disease Severity Assessment:
  - Ulcerative Colitis Severity: Mayo Scoring System (0–12 points).
  - Crohn's Disease Activity: Crohn's Disease Activity Index (CDAI) (> 450 severe, < 150 remission).

Outcome measures

1. Primary Outcome: Prevalence of *H. pylori* infection in IBD vs. control groups.
2. Secondary Outcomes:

- Association of *H. pylori* infection with IBD severity (Mayo/CDAI scores).
- Association of *H. pylori* infection with smoking and autoimmune diseases.
- Association of *H. pylori* with IBD medication use.

Study size and sample calculation

The required sample size for this study was determined using a standard formula for estimating proportions in cohort studies: ss = Z^2 x (p) \ x (1-p) / c^2.

Statistical analysis

All analyses were conducted using IBM SPSS Statistics version 26.

1. Descriptive Statistics:

- Categorical variables were reported as frequencies (%).
- Continuous variables were presented as mean ± standard deviation (SD) or median (IQR) where appropriate.

2. Comparative Analysis:

- The Chi-square test (χ^2) or Fisher's exact test was used for categorical variables.
- The t-test or Mann-Whitney U test was used for continuous variables.

3. Multivariate Analysis:

- Logistic regression analysis was conducted to estimate the odds ratio (OR) with 95% confidence intervals (CI) for factors associated with *H. pylori* infection while adjusting for potential confounders (age, smoking, autoimmune disease, medication use).

4. Missing Data Handling:

- Multiple imputation (MI) was used for missing variables when appropriate.
- Sensitivity analysis was conducted to assess the impact of missing data on study outcomes.

5. Methods for Controlling Confounders:

- Stratification: The study categorized participants into IBD and control groups and further stratified IBD into Crohn's disease (CD) and ulcerative colitis (UC) subgroups to reduce confounding by disease type.
- Statistical Adjustments: Logistic regression analysis was used to adjust for confounders, calculating adjusted odds ratios (ORs) with 95% confidence intervals (CIs) to control for multiple variables simultaneously.
- Exclusion Criteria: The study excluded individuals under 18 years old, pregnant women, patients with malignancy, and those using proton pump inhibitors (PPIs) to minimize confounding effects.
- Matching: The control group was selected from individuals without gastrointestinal disorders, reducing selection bias and confounding due to unrelated digestive conditions.
- Multivariate Analysis: Variables such as smoking status, autoimmune disease presence, and medication use were analyzed separately to assess their independent impact on H. pylori infection.

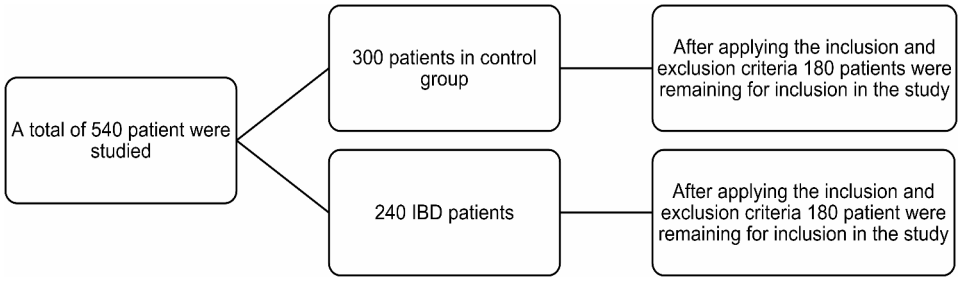
Ethical considerations

- Approval: The study protocol was reviewed and approved by the Institutional Review Board (IRB) of Imam Abdulrahman Bin Faisal University (IRB-2023-01-437).
- Informed consent: Written informed consent was obtained from all participants before enrollment.
- Confidentiality: Data anonymity and confidentiality were maintained following institutional and ethical guidelines.

Results

Demographic and clinical features

A total of 360 patients were included in the study which were divided equally into IBD group and control group.



The IBD were subdivided into CD with 91 cases and UC with 89 cases. As shown in (Table 1), Majority of cases in IBD 54 (30%) {UC 25.3%; CD 34.8%} were aged between 18 and 25 years. A low proportion of H. pylori infection was found in IBD group 25 (13.9%) {UC 13.2%; CD 14.6%} as compared to control group 42 (23.3%). Autoimmune diseases were more frequent in controls (12.2%) compared to UC (6.6%) and CD (5.6%).

Correlation between H. pylori infection and IBD

Table 2 examines the correlation between Helicobacter pylori (H. pylori) infection, smoking history, and

Table 1 Demographic and clinical features. (Number +%)

		IBD		Control
		UC *n* = 91	CD *n* = 89	
Gender	Male	49 (53.8%)	57 (64%)	87 (48.3%)
	Female	42 (46.2%)	32 (36%)	93 (51.7%)
Age	18–25	23 (25.3%)	31 (34.8%)	19 (10.6%)
	26–30	12 (13.2%)	22 (24.7%)	20 (11.1%)
	31–35	12 (13.2%)	16 (18%)	35 (19.4%)
	36–40	10 (11%)	10 (11.2%)	28 (15.6%)
	41–45	10 (11%)	4 (4.5%)	26 (14.4%)
	46–50	6 (6.6%)	2 (2.2%)	19 (10.6%)
	51–55	6 (6.6%)	0 (0%)	15 (8.3%)
	56–60	8 (8.8%)	1 (1.1%)	8 (4.4%)
	> 60	4 (4.4%)	3 (3.4%)	10 (5.6%)
Marital status	Single	30 (33%)	46 (51.7%)	34 (18.9%)
	Married	55 (60.4%)	40 (44.9%)	130 (72.2%)
	Divorced	2 (2.2%)	1 (1.1%)	8 (4.4%)
	Widow	4 (4.4%)	2 (2.2%)	8 (4.4%)
Educational status	Primary	0 (0%)	1 (1.1%)	5 (2.8%)
	Secondary	3 (3.3%)	1 (1.1%)	2 (1.1%)
	High school	27 (29.7%)	30 (33.7%)	23 (12.8%)
	Diploma	5 (5.5%)	4 (4.5%)	16 (8.9%)
	Bachelor	51 (56%)	52 (58.4%)	118 (65.6%)
	Master	4 (4.4%)	1 (1.1%)	15 (8.3%)
	PhD	1 (1.1%)	0 (0%)	1 (0.6%)
H. Pylori status	Positive	12 (13.2%)	13 (14.6%)	42 (23.3%)
	Negative	79 (86.8%)	76 (85.4%)	138 (76.7%)
Autoimmune disease	Yes	6 (6.6%)	5 (5.6%)	22 (12.2%)
	No	85 (93.4%)	84 (94.4%)	158 (87.8%)
Smoking hx	Non-smoker	76 (83.5%)	77 (86.5%)	147 (81.7%)
	Current smoker	13 (14.3%)	9 (10.1%)	24 (13.3%)
	Ex. Smoker	2 (2.2%)	3 (3.4%)	9 (5%)

Abbreviations:

CD: Crohn's Disease

UC: Ulcerative Colitis

IBD: Inflammatory Bowel Disease

H. Pylori: Helicobacter pylori

Table 2 Correlation between H. pylori in IBD compared to control group

		IBD (number+%)		Control	P-values	
		UC	CD		UC Vs Control	CD Vs Control
H. Pylori	Yes	12 (13.2%)	13 (14.6%)	42 (23.3%)	0.048	0.095
	No	79 (86.8%)	76 (85.4%)	138 (76.7%)		
Smoking hx	Non-smoker	76 (83.5%)	77 (86.5%)	147 (81.7%)	0.539	0.599
	Current smoker	13 (14.3%)	9 (10.1%)	24 (13.3%)		
	Ex. Smoker	2 (2.2%)	3 (3.4%)	9 (5%)		
Autoimmune Disease	Yes	6 (6.6%)	5 (5.6%)	22 (12.2%)	0.151	0.090
	No	85 (93.4%)	84 (94.4%)	158 (87.8%)		

CD: Crohn's Disease

UC: Ulcerative Colitis

IBD: Inflammatory Bowel Disease

H. Pylori: Helicobacter pylori

Table 3 H. pylori infection status and its association to smoking history and autoimmune disease

		H. pylori + Ve	H. pylori -Ve	*P* value
Smoking History	Non-smoker	33 (49.3%)	267 (91.1%)	< 0.0001
	Current Smoker	29 (43.3%)	17 (5.8%)	< 0.0001
	Ex. Smoker	5 (7.5%)	9 (3.1%)	0.09
Autoimmune Disease	Yes	11 (16.4%)	22 (7.5%)	0.023
	No	56 (83.6%)	271 (92.5%)	

Table 4 Comparison of activity scores between inflammatory bowel disease (IBD) patients with and without H. pylori infection

		IBD patients with H. pylori infection	IBD patients without H. pylori infection	*P* value
CDAI Severity score for CD	Clinical remission	6 (46.2%)	31 (40.8%)	0.72
	Mild	5 (38.5%)	21 (27.6%)	0.43
	Moderate	1 (7.7%)	20 (26.3%)	0.15
	Severe	1 (7.7%)	4 (5.3%)	0.73
UC Myo Severity score	Normal or Inactive	8 (66.7%)	28 (35.4%)	0.06
	Mild	2 (16.7%)	25 (31.6%)	0.29
	Moderate	2 (16.7%)	15 (19%)	0.85
	Severe	0 (0%)	11 (13.9%)	0.17

CD: Crohn's Disease

UC: Ulcerative Colitis

IBD: Inflammatory Bowel Disease

H. Pylori: Helicobacter pylori

autoimmune diseases in IBD patients compared to control group. H. pylori infection was less prevalent in UC (13.2%) and CD (14.6%) patients compared to controls (23.3%). The difference between UC and controls was statistically significant ($p = 0.048$), whereas the CD vs. control comparison showed a trend but did not reach significance ($p = 0.095$). Autoimmune diseases were more common in controls (12.2%) than in UC (6.6%) and CD (5.6%) patients, though the differences were not statistically significant ($p = 0.151$ for UC vs. control, $p = 0.090$ for CD vs. control).

Association of H. pylori infection with smoking history and autoimmune disease

The relation between H. pylori infection with smoking and autoimmune disease in shown in (Table 3). In terms of smoking status, H. pylori-positive individuals had a significantly lower percentage of non-smokers (49.3%) compared to H. pylori-negative individuals (91.1%), with a highly significant p-value (< 0.0001). Current smoking was much more prevalent in H. pylori-positive individuals (43.3%) compared to H. pylori-negative individuals (5.8%), also with strong statistical significance ($p < 0.0001$). Ex-smokers showed a slight, non-significant trend toward higher H. pylori infection ($p = 0.09$). In terms of autoimmune diseases, the prevalence were higher in H. pylori-positive individuals (16.4%) compared to H. pylori-negative individuals (7.5%), with a statistically significant p-value (0.023).

Activity scores in IBD patients with and without H. pylori infection

Table 4 compares disease activity scores between inflammatory IBD patients with and without H. pylori infection,

Table 5 Correlation between H. pylori infection with medications

		IBD patients with H. pylori infection	IBD patients without H. pylori infection	*P.* value
Conventional	5-ASA	4 (57.1%)	31 (51.7%)	0.8
	Steroids	0 (0%)	3 (5%)	0.57
	Sulfasalazine	0 (0%)	1 (1.7%)	0.75
	Azathioprin	1 (14.3%)	13 (21.7%)	0.67
	5-ASA + Azathioprin	2 (28.6%)	10 (16.7%)	0.47
	Azathioprin + Steroids	0 (0%)	2 (3.3%)	0.65
Biologic	TNF-blocking agent (Infliximab or adalimumab)	7 (58.3%)	41 (68.3%)	0.505
	Vedolizumab	0 (0%)	1 (1.7%)	0.65
	JAK inhhibitor (Upadacitinib or Tofacitinab)	2 (16.7%)	14 (23.3%)	0.62
	Thiopurines + Infliximab	3 (25%)	4 (6.7%)	0.053
Both	Azathioprine + TNF inhibitor	5 (83.3%)	29 (82.9%)	0.98
	Sulfasalazine + TNF inhibitor	1 (16.7%)	1 (2.9%)	0.15
	Steroid+ TNF inhibitor	0 (0%)	2 (5.7%)	0.55
	5-ASA + TNF inhibitor	0 (0%)	3 (8.6%)	0.46

Abbreviation:

TNF: anti-tumor necrosis factor

JAK: Janus kinase inhibitor

CD: Crohn's Disease

UC: Ulcerative Colitis

IBD: Inflammatory Bowel Disease

H. Pylori: Helicobacter pylori

using the Crohn's Disease Activity Index (CDAI) for Crohn's disease (CD) and the Ulcerative Colitis (UC) Mayo severity score.

Regarding CDAI Severity Score: Clinical remission rates were similar between H. pylori-positive (46.2%) and H. pylori-negative (40.8%) patients ($p = 0.72$). In addition, mild and moderate disease activity was more frequent in H. pylori-negative patients, but the differences were not statistically significant ($p > 0.05$). Severe disease was slightly more common in H. pylori-negative patients (5.3% vs. 7.7%), but this was not statistically significant ($p = 0.73$).

Regarding UC Mayo Severity Score: A higher proportion of H. pylori-positive patients had normal or inactive UC (66.7%) compared to H. pylori-negative patients (35.4%), with a trend toward significance ($p = 0.06$). Mild and moderate UC severity was comparable between groups, with no significant differences. Severe UC was observed only in H. pylori-negative patients (13.9%), but the difference was not statistically significant ($p = 0.17$).

The overall association between severity of CD, UC and H. Pylori was insignificant.

Correlation between H. Pylori infection and medication use in IBD patients

Table 5 explores the relationship between H. pylori infection and medication used in IBD patients. For conventional therapies, no significant differences were found in the use of 5-ASA, steroids, sulfasalazine, or azathioprine between infected and uninfected groups ($p > 0.05$). Among biologic therapies, TNF inhibitors were slightly less common in infected patients (58.3% vs. 68.3%, $p = 0.505$). JAK inhibitors and vedolizumab showed no significant differences. Notably, the combination of thiopurines and infliximab was more frequent in infected patients (25% vs. 6.7%), however not approaching statistical significance ($p = 0.053$). In summary, there was no significant relationship between H. Pylori infection and medication used in IBD.

Discussion

This prospective study was conducted to determine the prevalence of H. Pylori infection in inflammatory bowel disease patients and its association with severity of the disease.

There are many key findings in the present study. First, H. pylori infection was significantly higher in the control group in comparison to IBD group. Second, smoking and autoimmune disease was significantly associated with H. pylori infection. Third, the overall association between severity of CD, UC and H. Pylori was insignificant. Finally, the association between H. pylori infection and medication used in IBD was also insignificant.

According to literature many authors looked into the prevalence of H. pylori in patients with inflammatory bowel disease and whether it's more in this group

or in general population. Sonnenberg A et al. (2012) conducted a study in Portland and found that H. pylori was inversely associated with IBD 0.48 (0.27–0.79) for Crohn's disease, 0.59 (0.39–0.84) for ulcerative colitis and 0.43 (0.15–0.95) for control group [17]. Similarly Jin X et al. (2013) investigated the relationship between Helicobacter Pylori infection and Ulcerative Colitis and discovered that the infection rate in the UC group was 30.5% which is significantly lower than the control group which was 57.0% [18]. Furthermore, Ali I et al. (2022) conducted a study in Palestine and the aim was to study the relationship between UC and H. Pylori infection. Results showed that the overall positive rate for H. pylori infection in UC patients was 14.3%, which was considerably lower than the 41.9% in the control group (P value 0.003) [6]. Other studies also had the same findings [8, 19] In our study H. Pylori was significantly higher in control group 42 (23.3%) compared with UC cases 12 (13.2%) $p = 0.048$. Similarly, H. pylori infection was higher in control group compared to CD group but didn't reach significance (14.6%) $p = 0.098$.

Numerous studies have been looking into the association between H. pylori infection and inflammatory IBD, and a significant number of studies suggested that H. pylori may offer a protective effect against IBD. The most recent and comprehensive meta-analysis to investigate the protective effect was conducted by Castano-Rodriguez et al. analyzing 40 studies. The study's total sample comprised 6130 IBD patients and 74,659 non-IBD controls. The overall analysis, covering all IBD patients revealed a negative association, indicating a 57% lower likelihood of IBDs (OR: 0.43, 95% CI 0.36 to 0.50, $p < 0.0000000001$) [20]. In addition, Zhong Y et al. (2021) conducted A meta-analysis, and their findings demonstrated that there is a negative correlation between H. Pylori and IBD. Moreover, they noticed that eradication of H. pylori in IBD patients made them 1.41 times more likely to relapse [21]. Furthermore, according to Bartels LE et al. (2015), H. pylori infection is a preventative factor against the development of CD because it is associated with a decreased prevalence of CD in those who are positive for it [22]. Several mechanisms have been proposed to explain the protective role of Helicobacter pylori (H. pylori) against inflammatory bowel diseases (IBDs), including Crohn's disease (CD) and ulcerative colitis (UC). These mechanisms primarily involve immune modulation, gut microbiota alterations, and epithelial interactions. One of the key mechanisms by which H. pylori exerts its protective effect against IBDs is through immune regulation [20]. The infection induces regulatory T cells (Tregs), which play a crucial role in suppressing excessive immune activation [19]. In addition, Chronic inflammation in IBD is driven by an upregulation of pro-inflammatory cytokines, particularly those associated with Th1 and Th17 responses, such as tumor necrosis factor-alpha (TNF-α), interleukin-17 (IL-17), and interleukin-23 (IL-23). H. pylori infection has been shown to downregulate these pro-inflammatory pathways, leading to a dampened immune response and reduced susceptibility to inflammation-induced intestinal damage [23–25]. Other studies didn't find relation between H.pylori and IBD, Abd El-Wahab EW et al. (2021) conducted study in Egypt and the findings of the study neither support the negative correlation nor deny it. He also investigated H. pylori effect on IBD progression beside the association, and their results were approximately (49.5%) of IBD patients who had evidence of H. Pylori infection [21]. H. pylori's protective effects might simply be the result of other confounding factors, such as the existence of innate genetic or environmental factors that promote IBD development in some people and H. pylori acquisition in others [23, 24].

Smoking has been associated with acquisition and increased persistence of Helicobacter pylori infection, as well as with lower effectiveness of its eradication. Rajashekhar et al. found that 80% smokers were positive for H. pylori infection with ($p < 0.001$) compared to non-smokers [26]. Similarly, Cardenas et al. study concluded that the prevalence of H. pylori infection was higher among those who currently smoked (prevalence odds ratio = 1.9; 95% CI = 1.4–2.5). Elevated serum cotinine, a marker of current smoking, was likewise linked to a higher incidence of H. pylori infection (1.6; 1.3–2.0) [27]. In contrast, Ferro et al. found that there was no significant association between smoking (ever vs. never) and H. pylori seropositivity (adjusted odds ratio = 1.08; 95% CI: 0.89–1.32; adjusted PR = 1.01; 95% CI: 0.98–1.05) [28]. In our study the proportion of H. Pylori infected patients was significantly high in those cases who smoke cigarettes 29 (43.3%) $p < 0.0001$.

Chronic H. pylori infection could participate in the etiopathogenesis and maintenance of inflammatory activity in some autoimmune diseases [29] According to Ram et al. Anti-phospholipid syndrome, giant cell arteritis, systemic sclerosis, and primary biliary cirrhosis patients had higher serum levels of IgG against H. pylori, and anti-H. pylori antibodies were linked to higher prevalences of anti-dsDNA and anti-Ro/SSA antibodies [30]. In our study, it was found that H. Pylori infection was more prevalent in patients with autoimmune disease with a significant p value of 0.023.

In terms of Association of H. Pylori infection with IBD treatment strategy, Yufen Zhong (2021) found that there was no significant association between the medications used in IBD and H. Pylori infection [21]. Similarly, according to Abd El-Wahab EW et al. (2021), neither the H. pylori infection nor the IBD treatment strategy significantly altered the course of IBD [22]. Furthermore,

Ali, Iyad et al. found that there was no effect of the drugs observed in the treatment of UC on the positivity of H. pylori (P-value = 0.808) [31]. Our study also found no significant relationship between medication used in IBD and H. pylori infection.

In term of H. pylori infection and severity of IBD, Matsumura M et al. (2001) studied the association between H. Pylori infection in IBD and its association with medical treatment and severity in Japan. He found that there was no correlation between H. Pylori infection and severity of IBD. Furthermore, H. pylori- positive rate did not differ significantly with treatment options used in IBD [32]. Murad H et al. (2021) also studied the relation between H. Pylori infection and IBD severity. He found that Mayo score did not differ between the ulcerative colitis severity and H. Pylori positivity. However, there was strong correlation between CD H. pylori positive and severity of the disease by using CDAI score with (P value of 0.01) [33]. The overall association between severity of CD, UC and H. Pylori was insignificant in our study.

Conclusion

This prospective study was conducted to determine the prevalence of H. Pylori infection in inflammatory bowel disease patients and its association with severity of the disease. The study concluded three main findings: First, H. pylori infection was significantly higher in the control group in comparison to IBD group. Second, smoking and autoimmune disease were significantly associated with H. pylori infection. Finally, the overall association between severity of CD, UC and medication use with H. Pylori were insignificant.

Limitations

As the study is a single center study there will be limited generalizability, findings may not be applicable to broader populations due to differences in demographics, healthcare settings, or regional factors. In addition, the study may have a smaller sample size compared to multicenter studies, reducing statistical power and increasing the risk of type II errors.

Author contributions

Abdullah D. Alotaibi was in charge of conceptualization, supervision of data collection, manuscript preparation, revision, and submission. Reem S. AlSulaiman was the corresponding author that was responsible for submission, analysing the data, and revision of the manuscript. Abdullah A. Abdelwahab, Mona H. Ismail, Jaber M. AlElyani, Turki A. Alamri, Raed M. Alsulaiman, and Abdulaziz M. Alrezuk contributed to manuscript preparation. Ibrahim A. Alhafid, Ibrahim M. Alzahrani, Reem S. AlSulaiman, Arwa Althubaity, Sara H. Buhulaigah, Abdulaziz A. AlQurain contributed to data collection, analysis, and editing the final manuscript paper.

Funding

This study did not receive any funding.

Data availability

No datasets were generated or analysed during the current study.

Declarations

Ethical approval

The study design, protocol, and informed consent were approved by Imam Abdulrahman Bin Faisal University Institutional Review Board (IRB-2023-01-437). The study is adhered to the Declaration of Helsinki.

Consent for publication

Not Applicable.

Competing interests

The authors declare no competing interests.

Received: 12 January 2025 / Accepted: 14 April 2025
Published online: 29 April 2025

References

1. Park JR, Pfeil SA. Primary care of the patient with inflammatory bowel disease. Med Clin North Am. 2015 Sept;99(5):969–87. https://doi.org/10.1016/j.mcna.2015.05.009.
2. Abraham C, Cho JH. Inflammatory bowel disease. N Engl J Med. 2009;361(21):2066–78. https://doi.org/10.1056/nejmra0804647.
3. Jarmakiewicz-Czaja S, Zielińska M, Sokal A, Filip R. Genetic and epigenetic etiology of inflammatory bowel disease: an update. Genes. 2022;13(12):2388. https://doi.org/10.3390/genes13122388.
4. Su H-J, Chiu Y-T, Chiu C-T, Lin Y-C, Wang C-Y, Hsieh J-Y, et al. Inflammatory bowel disease and its treatment in 2018: global and Taiwanese status updates. J Formos Med Assoc. 2019;118(7):1083–92. https://doi.org/10.1016/j.jfma.2018.07.005.
5. Trivedi PJ, Adams DH. Chemokines and chemokine receptors as therapeutic targets in inflammatory bowel disease; pitfalls and promise. J Crohn's Colitis. 2018;12(suppl2). https://doi.org/10.1093/ecco-jcc/jjx145.
6. Ali I, Abdo Q, Al-Hihi SM, Shawabkeh A. Association between ulcerative colitis and helicobacter pylori infection: A case-control study. Heliyon. 2022;8(2). https://doi.org/10.1016/j.heliyon.2022.e08930.
7. Kamm MA. Rapid changes in epidemiology of inflammatory bowel disease. Lancet. 2017;390(10114):2741–2. https://doi.org/10.1016/s0140-6736(17)32669-7.
8. Yu Y, Zhu S, Li P, Min L, Zhang S. Helicobacter pylori infection and inflammatory bowel disease: A crosstalk between Upper and lower digestive tract. Cell Death &Disease. 2018;9(10). doi:10.1038/s41419-018-0982-2
9. Papamichael K. helicobacter pyloriinfection and inflammatory bowel disease: is there a link? World J Gastroenterol. 2014;20(21):6374. https://doi.org/10.3748/wjg.v20.i21.6374.
10. Rokkas T, Gisbert J, Niv Y, O'Morain C. The association between helicobacter pylori infection and inflammatory bowel disease based on meta-analysis. United Eur Gastroenterol J. 2015;3(6):539–50. https://doi.org/10.1177/2050640615580889.
11. Wu X-W, Ji H-Z, Yang M-F, Wu L, Wang F-Y. helicobacter pylori infection and inflammatory bowel disease in Asians: A meta-analysis. World J Gastroenterol. 2015;21(15):4750–6. https://doi.org/10.3748/wjg.v21.i15.4750.
12. Luther J, Dave M, Higgins PDR, Kao JY. Association between helicobacter pylori infection and inflammatory bowel disease. Inflamm Bowel Dis. 2010;16(6):1077–84. https://doi.org/10.1002/ibd.21116.
13. Rosania R, Arnim UV, Link A, Rajilic-Stojanovic M, Franck C, Canbay A, et al. Helicobacter pylori eradication therapy is not associated with the onset of inflammatory bowel diseases. A case-control study. J Gastrointest Liver Dis. 2018;27(2):119–25. https://doi.org/10.15403/jgld.2014.1121.272.hpy.
14. Lewis JD, Chuai S, Nessel L, Lichtenstein GR, Aberra FN, Ellenberg JH. Use of the noninvasive components of the Mayo score to assess clinical response in ulcerative colitis. Inflamm Bowel Dis. 2008;14(12):1660–6. https://doi.org/10.1002/ibd.20520.
15. Best WR, Becktel JM, Singleton JW, Kern F Jr. Development of a Crohn's disease activity index. National cooperative Crohn's disease study. Gastroenterology. 1976;70(3):439–44.
16. Cuschieri S. The strobe guidelines. Saudi J Anaesth. 2019;13(5):31. https://doi.org/10.4103/sja.sja_543_18.
17. Sonnenberg A, Genta RM. Low prevalence of helicobacter pylori infection among patients with inflammatory bowel disease. Aliment Pharmacol Ther. 2012;35(4):469–76. https://doi.org/10.1111/j.1365-2036.2011.04969.x.

18. Jin X, Chen Y, Chen S, Xiang Z. Association between helicobacter pylori infection and ulcerative colitis-A case control study from China. Int J Med Sci. 2013;10(11):1479–84. https://doi.org/10.7150/ijms.6934.
19. Bartels LE, Dahlerup JF. Association of helicobacter pylori and Crohn's disease incidence: an inversion reaction? Dig Dis Sci. 2017;62(9):2217–9. https://doi.org/10.1007/s10620-017-4561-7.
20. Bretto E, Frara S, Armandi A, Caviglia GP, Saracco GM, Bugianesi E, Pitoni D, Ribaldone DG. Helicobacter pylori in inflammatory bowel diseases: active protagonist or innocent bystander?? Antibiot (Basel). 2024;13(3):267. https://doi.org/10.3390/antibiotics13030267.
21. Zhong Y, Zhang Z, Lin Y, Wu L. The relationship between helicobacter pylori and inflammatory bowel disease. Arch Iran Med. 2021;24(4):317–25. https://doi.org/10.34172/aim.2021.44.
22. Abd El-Wahab EW, Youssef EI, Hassouna E. helicobacter pylori infection in patients with inflammatory bowel diseases: A single-centre, prospective, observational study in Egypt. BMJ Open. 2022;12(5). https://doi.org/10.1136/bmjopen-2021-057214.
23. Kelly D, Conway S, Aminov R. Commensal gut bacteria: mechanisms of immune modulation. Trends Immunol. 2005;26(6):326–33. https://doi.org/10.1016/j.it.2005.04.008.
24. Raju D, Hussey S, Ang M, Terebiznik MR, Sibony M, Galindo–Mata E, et al. Vacuolating cytotoxin and variants in ATG16L1 that disrupt autophagy promote helicobacter pylori infection in humans. Gastroenterology. 2012;142(5):1160–71. https://doi.org/10.1053/j.gastro.2012.01.043.
25. Giuffrida P, Di Sabatino A. Targeting T cells in inflammatory bowel disease. Pharmacol Res. 2020 Sept;159:105040. https://doi.org/10.1016/j.phrs.2020.105040.
26. Rajashekhar V, Singh K, Sharma B, Vaiphei K, Ray P, Bhasin D. Helicobacter pylori infection in chronic smokers with Non ulcer dyspepsia. Trop Gastroenterology: Official J Dig Dis Foundation. 2000;21(22):71–2. doi:PMID: 10881628.
27. Cardenas VM, Graham DY. Smoking and helicobacter pylori infection in a sample of U.S. Adults. Epidemiology. 2005;16(4):586–90. https://doi.org/10.1097/01.ede.0000165365.52904.4a.
28. Ferro A, Morais S, Pelucchi C, Aragonés N, Kogevinas M, López-Carrillo L et al. Smoking and helicobacter pylori infection: an individual participant pooled analysis (stomach cancer pooling- stop project). Eur J Cancer Prev 2019 Sept;28(5):390–6. https://doi.org/10.1097/cej.0000000000000471
29. Magen E. helicobacter pyloriand skin autoimmune diseases. World J Gastroenterol. 2014;20(6):1510. https://doi.org/10.3748/wjg.v20.i6.1510.
30. Ram M, Barzilai O, Shapira Y, Anaya J-M, Tincani A, Stojanovich L, et al. Helicobacter pylori serology in autoimmune diseases– fact or fiction? Clin Chem Lab Med. 2013;51(5). https://doi.org/10.1515/cclm-2012-0477.
31. Ali I, Abdo Q, Al-Hihi SM, Shawabkeh A. Association between ulcerative colitis and Helicobacter pylori infection: A case-control study. Heliyon. 2022;8(2):e08930. https://doi.org/10.1016/j.heliyon.2022.e08930.
32. Matsumura M, Hatakeyama S, Sakurai T, Oishi T, Fujioka T, Matake H, et al. Prevalence of helicobacter pylori infection and correlation between severity of upper Gastrointestinal lesions and H. pylori infection in Japanese patients with Crohn's disease. J Gastroenterol. 2001;36(11):740–7. https://doi.org/10.1007/s005350170015.
33. Murad H, Rafeeq M, Mosli M, Gari M, Basheikh M. Effect of sequential eradication therapy on serum osteoprotegerin levels in patients with helicobacter pylori infection and co-existing inflammatory bowel disease. J Int Med Res. 2021;49(11):030006052110606. https://doi.org/10.1177/03000605211060648.

Publisher's note