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Kamaleldin B. Said * , Khalid F Alshammari , Safia Moussa , Ruba M. Elsaid Ahmed , Ahmed H. Aljadani , Najd B Albalawi , Layan Al-Hujaili , Ruaa Alharbi , Arwa A Alotaibi , Fahad M Alshammary , Alfatih M. A. Alnajib , Fayez R Alfouzan , Abuzar A.A. Osman , Zaid A. Albayih , Bader Alkharisi , Naif M Altamimi

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Clinical Diagnostics and Disease Profiles of *Helicobacter pylori* Infected Inpatients Reveal Age- and Gender-Specificity and Aggravation in a Select Sequalae of Chronic Syndromes

Kamaleldin B. Said [1,*], Khalid F. Alshammari [2], Safia Moussa [3], Ruba M. Elsaid Ahmed [1], Ahmed Aljadani [2], Najd B. Albalawi [1], Layan Al-Hujaili [1], Ruaa Alharbi [1], Arwa A. Alotaibi [1], Fahad M. Alshammary [1], Alfatih M. A. Alnajib [4], Fayez R. Alfouzan [3], Abuzar A. A. Osman [5], Zaid A. Albayih [1], Bader Alkharisi [2] and Naif M. Altamimi [6]

[1] Department of Pathology, College of Medicine, University of Ha'il, Ha'il 55462, Saudi Arabia
[2] Department of Internal Medicine, College of Medicine, University of Ha'il, Ha'il 55462, Saudi Arabia
[3] Department of Clinical Microbiology, KSSH, Ha'il 55462, Saudi Arabia
[4] Department of Surgery, College of Medicine, University of Ha'il, Ha'il 55462, Saudi Arabia
[5] Department of Pharmacology, College of Medicine, University of Ha'il, Ha'il 55462
[6] Department of Dermatology, College of Medicine, University of Ha'il, Ha'il 55462, Saudi Arabia
* Correspondence: kbs.mohamed@uoh.edu.sa

Abstract: Background/Objectives: Non-communicable diseases like obesity, diabetes mellitus (DM), and hypertension (HTN) impose major global burdens. *Helicobacter pylori* infection may worsen these conditions. This study examined the clinical, age-, and gender-specific profiles of *H. pylori*-infected inpatients and its association with dyslipidemia, obesity, HTN, DM, and other chronic conditions. **Methods**: Between September 2024 and February 2025, patients were tested using a stool antigen assay (SIMA CHECK–ACON BIOTEC). Demographic data, smoking status, and clinical diagnoses (including kidney, liver, and heart disease) were obtained from hospital records and confirmed with standard criteria. Associations were analyzed using chi-square/Fisher's tests and logistic regression (adjusted for age, gender, and smoking). **Results**: Overall, 30.7% (64/208) of inpatients were *H. pylori*–positive, highest in middle-aged females (42.9%) and lowest in middle-aged males (20.9%). Dyslipidemia rose sharply in males (5.6%→77.1%) versus females (5.7%→58.3%), with infection modestly elevating rates (OR=1.648). Obesity declined with age (males: 36.1%→22.9%; females: 45.7%→20.8%) yet strongly correlated with *H. pylori* (OR=19.217). HTN (5.6–94.3% in males; 11.4–100% in females) and DM (16.7–80% vs. 17.1–95.8%) increased with age but showed no infection link reflecting inflammatory preference. Smoking peaked at 61.1% in younger males. Kidney and heart diseases appeared only in ages 60–85. Overall, the prevalence of comorbidities markedly increased with age. **Conclusion**: *H. pylori* infection is common among inpatients, particularly in middle-aged females, and is significantly linked to dyslipidemia and obesity. These findings support targeted screening, especially in females and individuals with metabolic abnormalities, while future studies should assess whether eradication therapy can mitigate chronic metabolic risks.

Keywords: Helicobacter pylori; chronic diseases; dyslipidemia; obesity; hypertension; diabetes mellitus

1. Introduction

Non-communicable diseases (NCDs) impose enormous health and economic burdens worldwide, accounting for an estimated 41 million deaths each year and significantly straining healthcare systems and national economies [1]. In many low- and middle-income regions, rising

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obesity rates, escalating diabetes incidence, and the increasing prevalence of associated metabolic disorders intensify these challenges (20). The Eastern Mediterranean Region, for example, has experienced a marked surge in overweight and obesity, with recent estimates surpassing 30% among adults, leading to billions of dollars in healthcare expenditures and lost productivity [2]. Such burdens underscore the critical need for research elucidating modifiable risk factors, including possible contributions from persistent infections such as Helicobacter pylori (*H. pylori*) [3].

H. pylori is a gram-negative microaerophilic bacillus that has been present for thousands of years in the gastric and duodenal environment of the human host [4]. In the majority of cases, the infection is asymptomatic; however, the chronic infection induces host–gastritis response by the gastric epithelium, causing various disorders of the gastrointestinal tract like peptic ulcer disease, gastritis, and gastric cancers [5]. Detection of this organism has revolutionized the knowledge about the etiology of peptic ulcers by bringing the emphasis from mere hyperacidity towards host–pathogen interaction and the impact of the microbe [6]. Up-to-date epidemiological data suggest nearly fifty percent of the global populace could be infected by this microbe; however, the prevalence is governed by geographical distribution, socioeconomic status, and ethnic groups [7,8].

Despite its main impact on the digestive system, mounting bodies of scientific work confirm the idea that infection by *H. pylori* can also influence extra-gastric diseases, including some metabolic and cardiovascular disorders [9]. Proposed mechanisms involve not only the organism's presence causing direct effects, but also indirect processes by the host. These processes can involve cytokine profile changes and modulation of the immune system, potentially affecting insulin resistance, lipid metabolism, and vascular tone [10]. Systemic inflammation caused by continuous stimulation of the immune system could theoretically induce pro-inflammatory environment predisposing the patient for metabolic disorders [2]. This theoretical concept has gained popularity, but the strength of the associations is not clear, and thus the need for well-characterised cohorts and stringent data analysis is emphasised [11].

Among the diseases caused by the alleged association of *H. pylori* infection, diabetes mellitus (DM) has drawn much attention. In some reports, the ongoing inflammation from the infection by *H. pylori* has the potential for exacerbating insulin resistance, thus compromising the control over blood sugars and potentially accelerating the diabetes-related sequelae [12,13]. Similarly, hypertension (HTN) has also been studied alongside the potential impact from infection by *H. pylori*, owing to the relationship between vascular endothelial dysfunction and systemic pro-inflammatory biomarkers [14]. Another essential component of the metabolic syndrome is the potential impact from ongoing infection through lipid metabolism changes and pro-atherogenic processes [15]. Yet the biological processes involved remain poorly elucidated, and thus the need for continued investigation into the potential for increased risk from the infection by *H. pylori* for the dysregulated lipid levels.

Obesity introduces one more consideration into this complex interaction, given its definition as complex chronic disorder subject to influences from genetics, lifestyle, and environment. In the past, obesity has also been linked historically with insulin resistance and dyslipidemia; however, one recent hypothesis is that infection by the organism *H. pylori* increases or decreases these processes through the influence upon the gut hormones [16]. To explore whether *H. pylori* influence the onset and progression of obesity alone or combined with other etiologies is one very significant opportunity for understanding the broader clinical implications for persistent infections. In addition to the metabolic disorder, the consequences of smoking were also studied through the epidemiological data for its possible influence on the infection susceptibility by *H. pylori* and the attendant clinical manifestations. Tobacco consumption increases the systemic level of inflammation substantially, thus compromising the protective functions of the gastric mucosa, potentially exacerbating the pathological impacts of *H. pylori*. In contrast, some groups may engage health-enhancing habits or lifestyle changes that remove one risk component while incidentally exacerbating the other [17]. These complexities highlight the need for undertaking an overall risk analysis incorporating demographic variables like the subject's age and gender, each potentially playing unique impacts upon the disease burden.

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Despite the wealth of published data, inconsistencies in sample size, geographical context, and methodology continue to obscure definitive conclusions regarding the exact relationship of *H. pylori* to chronic conditions like DM, HTN, dyslipidemia, and obesity. Variability in diagnostic assays—such as serology, urea breath tests, and stool antigen tests—further complicates comparisons across studies [18]. Additionally, many investigations have been confined to specific geographic or ethnic groups, limiting the generalizability of their findings [19]. These gaps emphasize the importance of large-scale, population-based studies that employ standardized testing protocols for *H. pylori* and robust clinical assessments of coexisting chronic diseases [20].

In the context of this study, the present cross-sectional analysis was undertaken to explain the prevalence of infection by *H. pylori* in individuals segregated by multiple age groups and by gender differences, while simultaneously analyzing its relationship with the prevalence of associated chronic diseases. In this study, through the systematic evaluation of dyslipidemia, obesity, hypertension, diabetes mellitus, and other comorbidities relative to the presence of *H. pylori* infection, the objective is to improve the understanding of the widespread health implications caused by this omnipresent organism.

2. Materials and Methods

2.1. Work Environment: Ha'il Province, City, and all Socio-Economic Strata

The King Salman Specialist hospital (KSSH) is certified and accredited by the Saudi Central Board for Accreditation of Healthcare Institutions (CBAHI)-Ref.no. HAL/MOH/HO5/34213 and along with the Ha'il Health Regional Laboratory (HHRL) which is also certified and accredited by the CBAHI)-Code 2739 constitutes a major cluster for healthcare diagnostic centers that receive samples for testing. Since *H.pylori* is a major environmental carcinogenic pathogen, it is imperative to describe climate and environmental conditions of the Ha'il city. Ha'il lies in the north-central Saudi Arabia, bordering five provinces, namely, Madinah, Tabouk, Northern Border, Riyadh and Qassim. It has a population size of nearly one million mostly in the Ha'il city which is in the Waadi Ha'il with its attraction of magnificent prehistoric rock carving and archaeological excavations. The region has several major hospitals, among them the KSSH, a tertiary care center serving the whole region.

A retrospective cross-sectional study was conducted between September 2024 and February 2025, enrolling 208 in-patients who underwent clinical evaluation for *H. pylori* infection. All participants were tested for *H. pylori* and assessed for relevant gastrointestinal and metabolic conditions during their hospitalization. Demographic data (age and gender), smoking status, and the presence or absence of dyslipidemia, obesity, HTN, DM, kidney disease, liver disease, and heart disease were recorded at the time of admission or within the hospital stay.

All inpatients stool samples were tested using a qualitative immunochromatographic assay (SIMA CHECK–ACON BIOTEC) as described by Ozdemir and Baykan (2005), Antos et al. (2005), and Silva et al. (2010). Following the manufacturer's protocol, each stool sample was first brought to room temperature and mixed thoroughly. Two drops (70–90 μL) of the homogenized specimen were then dispensed into the sample well of the test cassette. After a 10-minute incubation period, the appearance of distinct red lines was interpreted as follows: A single red line at the control (C) region indicated a negative result; Two red lines (one at the C region and one at the test [T] region) signified a positive result. Absence of a red line in the C region, regardless of whether a T line appeared, rendered the test invalid, and the assay was repeated with a new device. Excess specimen volume was avoided, since it could yield invalid results. All interpretations were made within 10–15 minutes of applying the sample to ensure proper reading.

Diagnosis of obesity followed a body mass index (BMI) threshold of ≥30 kg/m², and HTN and DM were determined through standard clinical criteria or documented medical histories. Chronic pathologies such as kidney, liver, and heart disease were confirmed through relevant clinical findings and laboratory or imaging tests. Smoking status was self-reported during clinical interviews, and patients were categorized as current smokers or non-smokers.

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Data were analyzed using SPSS version 23.0 (SPSS Inc., Chicago, IL, USA). Categorical variables were expressed as frequencies and percentages. Associations between *H. pylori* status and dichotomous outcomes (e.g., presence vs. absence of specific comorbidities) were evaluated by chi-square or Fisher's exact tests, as appropriate. Odds ratios (OR) with 95% confidence intervals (CI) were calculated to gauge the strength of each association. Where relevant, logistic regression models were employed to adjust for potential confounders, including age, gender, and smoking status. Statistical significance was defined by a two-sided p-value <0.05.

3. Results

Overall Demographics

A total of 208 participants were evaluated, consisting of 114 males (54.8%) and 94 females (45.2%), thereby allowing balanced assessment of *H. pylori* status and clinical comorbidities across both gender groups (Table 1). The data set permitted exploration of metabolic and organ-specific disorders in conjunction with demographics, aiding in understanding potential risk variations.

Table 1. Age-, and gender- stratified frequencies of *H. pylori* infections and chronic diseases .

		Gender											
		Male **(N=114; 54.8%)**						Female **(N=94; 45.2%)**					
		Age						Age					
		20-39 (*n*=36)		40-59 (*n*=43)		60-85 (*n*=35)		20-39 (*n*=35)		40-59 (*n*=35)		60-85 (*n*=24)	
		N	N %	N	N %	N	N %	N	N %	N	N %	N	N %
Dyslypidemia (m/f: 64.4M, 35.6%F)	no	34	94.4%	25	58.1%	8	22.9%	33	94.3%	25	71.4%	10	41.7%
	yes	2	5.6%	18	41.9%	27	77.1%	2	5.7%	10	28.6%	14	58.3%
Obesity (BMI > 30 kg/m²) (m/f: 47%M, 53%F)	no	23	63.9%	35	81.4%	27	77.1%	19	54.3%	23	65.7%	19	79.2%
	yes	13	36.1%	8	18.6%	8	22.9%	16	45.7%	12	34.3%	5	20.8%
HTN (Mf: 55%M; 45.4F)	no	34	94.4%	25	58.1%	2	5.7%	31	88.6%	19	54.3%	0	0.0%
	yes	2	5.6%	18	41.9%	33	94.3%	4	11.4%	16	45.7%	24	100%
DM (m/f: 54%M; 46%F)	no	30	83.3%	13	30.2%	7	20.0%	29	82.9%	9	25.7%	1	4.2%
	yes	6	16.7%	30	69.8%	28	80.0%	6	17.1%	26	74.3%	23	95.8%
Heart disease (m/f: 75%M; 25%F)	no	36	100%	42	97.7%	18	51.4%	35	100%	34	97.1%	13	54.2%
	yes	0	0.0%	1	2.3%	17	48.6%	0	0.0%	1	2.9%	11	45.8%
kidney disease (m/f: 71.4%M; 29%F)	no	36	100%	43	100%	26	74.3%	35	100%	35	100.0%	21	87.5%
	yes	0	0.0%	0	0.0%	9	25.7%	0	0.0%	0	0.0%	3	12.5%
Liver disease (m/f: 60%M; 40%F)	no	36	100%	43	100.0%	30	85.7%	35	100.%	35	100.0%	22	91.7%
	yes	0	0.0%	0	0.0%	5	14.3%	0	0.0%	0	0.0%	2	8.3%
other diseases	no	35	97.2%	38	88.4%	30	85.7%	27	77.1%	17	48.6%	10	41.7%
	yes	1	2.8%	5	11.6%	5	14.3%	8	22.9%	18	51.4%	14	58.3%
Smoking (94%M)	no	14	38.9%	29	67.4%	34	97.1%	35	100%	34	97.1%	24	100.0%
	yes	22	61.1%	14	32.6%	1	2.9%	0	0.0%	1	2.9%	0	0.0%
Stool Ag test (31.25%; 65/208) (mf: 49%; 51%F)	negative	24	66.7%	34	79.1%	24	68.6%	23	65.7%	20	57.1%	18	75.0%
	positive	12	33.3%	9	20.9%	11	31.4%	12	34.3%	15	42.9%	6	25.0%

Age, Gender, and Chronic Syndromes

As shown in Table 1, dyslipidemia rose substantially among older males (60–85 years: 77.1%) and was also elevated in older females (60–85 years: 58.3%), compared to much lower proportions in younger and middle-aged participants. Obesity exceeded 45% in younger females (20–39 years: 45.7%) versus 36.1% in males of the same age and then decreased with age in both sexes, suggesting a notable gender divergence in early adulthood (Table 1). Hypertension (HTN) climbed from 5.6% (20–39 years) to 94.3% (60–85 years) in males and from 11.4% to 100.0% in females across comparable

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age ranges. Diabetes mellitus (DM) similarly increased with age, rising to 80.0% in older males and 95.8% in older females (Table 1a). Heart, kidney, and liver diseases predominantly affected individuals aged 60–85, with higher absolute frequencies in males. Smoking habits were reported in 61.1% of younger males but dropped to 2.9% in older males while females seldom smoked (Table 1).

Age-, and Gender-Specificity of H. pylori and Association to Obesity and Dyslipidemia

Stool antigen testing indicated an *H. pylori* positivity rate exceeding 30% overall, spanning from 20.9% to 42.9% among 40–59-year-olds and approximating 25–34% in both younger (20–39 years) and older (60–85 years) age groups (Table 1). The highest prevalence was detected in middle-aged females (42.9%), while middle-aged males exhibited the lowest infection frequency (20.9%) (Table 1). These differences suggest that behavioral and/or physiological factors may alter gender-specific *H. pylori* susceptibility in midlife (Figure 1). In logistic regression, the multivariate models revealed significant gender specificity on obesity and dyslipidemia. An odds ratio (OR) of 1.491 (95% CI: 1.006–2.209, p=0.041) was noted for dyslipidemia in males (Table 1b). Obesity, however, was strongly associated with *H. pylori* infection, as indicated by an OR of 19.217 (95% CI: 9.117–40.507, p<0.001) (Table 2). HTN, DM, and other chronic conditions did not show significant ORs with respect *to H. pylori* positivity in this dataset (Table 2).

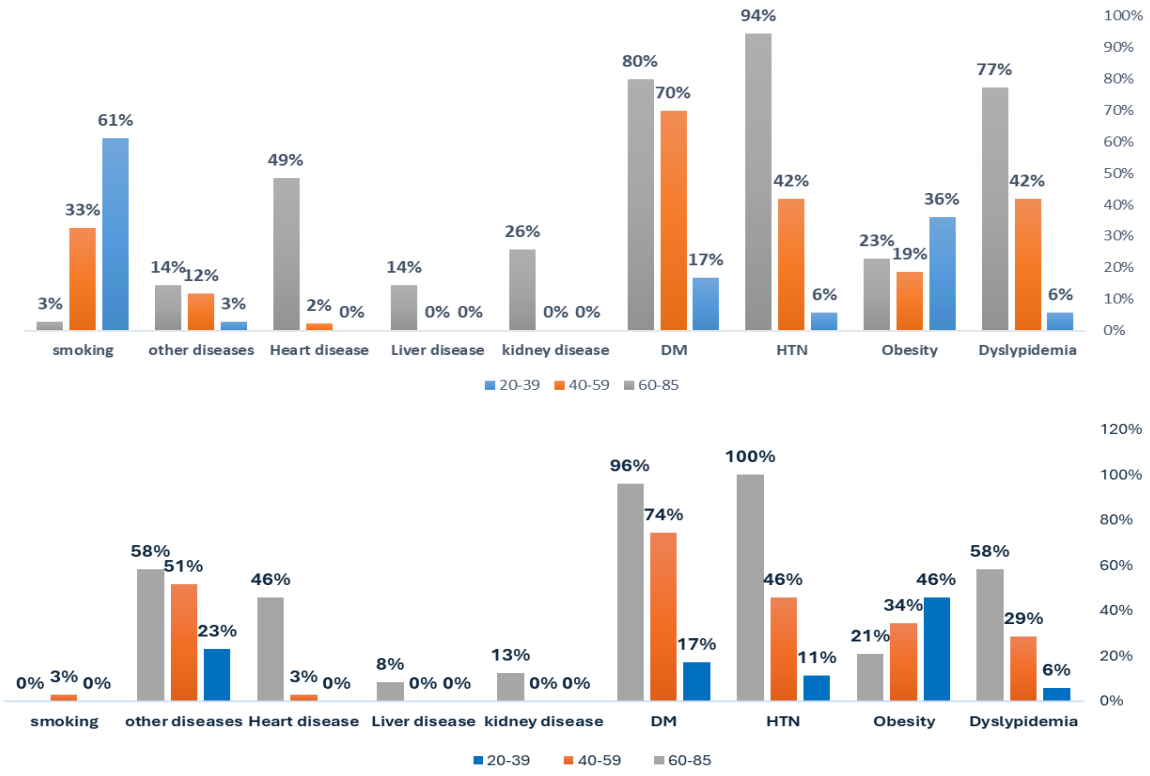

Figure 1. Prevalence of chronic conditions among study participants stratified by age and gender. The upper panel represents male participants, while the lower panel represents female participants across three age groups (20–39, 40–59, and 60–85 years).

Table 2. Logistic regression analysis for risk estimates of Helicobacter pylori infection and gender in chronic diseases.

Gender-Specific Association to Chronic Diseases					Risk of *H. pylori* Infection and Association in Chronic Diseases			
	Odd's Ratio	95% Confidence Interval		*p* Value	Odd's Ratio	95% Confidence Interval		*p* Value
Dyslipidemia	1.491	(1.006	2.209)	0.041	1.648	.900	3.017	.052

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Obesity	1.586	.872	2.882	.036	19.217	9.117	40.507	.000
Hypertension	1.103	.586	1.751	.002	.676	.373	1.225	1.672
DM	1.102	.634	1.914	.118	.819	.454	1.479	.437
Kidney diseases	.385	0.101	1.464	2.096	1.619	.494	5.307	.643
Liver diseases	.474	.090	2.500	.808	3.060	.665	14.088	2.260
Heart diseases	0.780	0.355	1.716	0.382	1.118	.491	2.546	.071
Smoking	0.022	0.003	0.167	0.000				
Other diseases					1.277	.654	2.490	.514[a]

Analysis of *H. pylori* Infection with dyslipidemia (borderline effect) indicated the latter disorder affected 73 of 208 participants (35.1%), with a higher rate (43.1%) in *H. pylori*-positive individuals than in those testing negative (31.5%) (Supplementary Table S2a). Although the Pearson chi-square test approached significance (p=0.104), it did not definitively confirm an association; the OR (1.648, 95% CI: 0.900–3.017) further suggested a borderline effect (Supplementary Table S2b, Supplementary Table S2c). Additional analyses by gender (Supplementary Table S2d–S2f) revealed that males accounted for 64.4% of dyslipidemia cases (p=0.041), underlining a notable predisposition in men (Supplementary Table S2f). Analysis of *H. pylori* Infection association with obesity has shown that among the 62 obese individuals (29.8%), 70.8% tested positive for *H. pylori*, whereas only 11.2% of non-obese participants were infected (Table 3). Statistical analyses confirmed a strong association (p<0.001), underscored by an OR of 19.217 (95% CI: 9.117–40.507), indicating that *H. pylori* infection was markedly linked with obesity (Tables 3–5). This finding emphasizes the potential impact of *H. pylori* on body weight regulation and warrants further investigation.

Table 3. Prevalence of obesity among study participants stratified by *H. pylori* stool antigen test results.

Obesity		Stool Ag test		Total
		negative	positive	
no	Count	127	19	146
	% within stool Ag test	88.8%	29.2%	70.2%
yes	Count	16	46	62
	% within stool Ag test	11.2%	70.8%	29.8%

Table 4. Chi-square test results for the association between obesity and *H. pylori* infection.

Chi-Square Tests	Value	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	75.819[a]	.000		
Continuity Correction[b]	72.998	.000		
Likelihood Ratio	74.664	.000		
Fisher's Exact Test			.000	.000
Linear-by-Linear Association	75.454	.000		
N of Valid Cases	208			

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Table 5. Risk estimates for obesity in relation to *H. pylori* infection.

Risk Estimate	Value	95% Confidence Interval	
		Lower	Upper
Odds Ratio for Obesity (no / yes)	19.217	9.117	40.507
For cohort stool Ag test = negative	3.371	2.200	5.165
For cohort stool Ag test = positive	.175	.112	.274

Association of H. pylori Infection with Hypertension, Diabetes Mellitus, Heart, Kidney, and Liver Disease

Although HTN prevalence increased from 5.6% in younger males to 94.3% in older males, and from 11.4% to 100.0% in females over the same age ranges (Table 1), the infection status itself did not correlate with higher HTN rates. *H. pylori*-positive individuals showed a slightly lower HTN prevalence (40.0%) than negative ones (49.7%) (Supplementary Table S4a), yielding p=0.196 and an OR of 0.676 (95% CI: 0.373–1.225) (Supplementary Table S4b–S4c). Similarly, DM was diagnosed in 119 participants (57.2%), with comparable frequencies between *H. pylori*-positive (53.8%) and *H. pylori*-negative (58.7%) groups (Supplementary Table S5a). However, the chi-square test (p=0.508) and OR of 0.819 (95% CI: 0.454–1.479) (Supplementary Table S5b–S5c) revealed no significant association, implying that there is no association between the infection and DM host conditions (Table 1). Heart disease, kidney disease, and liver disease were confined to seniors. Heart disease was observed in 15.4% of *H. pylori*-positive subjects versus 14.0% of negative ones (p=0.790) (Supplementary Table S8a–S8c). Kidney disease was detected in 7.7% of infected patients and 4.9% of non-infected ones (p=0.423) (Supplementary Table S6a–S6c), whereas liver disease affected 6.2% of those positive for *H. pylori* and 2.1% of those negative (p=0.133) (Supplementary Table S7a–S7c). None of these organ-specific conditions displayed a strong link to *H. pylori* infection.

In summarizing results, *H. pylori* prevalence exceeded 30% across the cohort and was highest in middle-aged females (42.9%) and lowest in middle-aged males (20.9%) (Table 1). Dyslipidemia, although mildly higher in infected individuals (p=0.104), was heavily influenced by male gender (64.4% of cases) (Supplementary Table S2a, Table S2d). Obesity showed the strongest correlation with *H. pylori*, with 70.8% of obese participants testing positive (p<0.001) (Table 3). Hypertension, diabetes, and organ-specific conditions were predominantly age-driven and did not exhibit statistically meaningful relationships with *H. pylori* status (Supplementary Tables S4–S8). Overall, these data highlight a potential role of *H. pylori* in exacerbating obesity and possibly contributing to dyslipidemia in certain subgroups, while hypertension, diabetes mellitus, and other pathologies seem more closely tied to age and gender. Graphical depictions of age and gender distributions, as well as infection rates, are presented (Figure 1).

4. Discussion

Among the diseases purportedly associated with H. pylori infection, DM continues to receive considerable attention. In some reports, ongoing inflammation triggered by H. pylori may exacerbate insulin resistance [21], compromise blood sugar control, and accelerate diabetes-related sequelae [22]. DM predisposes patients to cardiovascular disease (CVD) and impairs normal immune responses, thus highlighting the importance of exploring potential links with H. pylori, particularly in regions where diabetes prevalence is high [23]. Furthermore, there is a well-documented interplay between DM and HTN, wherein hyperglycemia fosters endothelial dysfunction and contributes to elevated blood pressure, thereby compounding the risk of CVD. Older individuals with diabetes are especially prone to developing HTN, magnifying their susceptibility to adverse cardiovascular events.

This study examined the prevalence of *H. pylori* infection in relation to chronic conditions, including dyslipidemia, obesity, HTN, DM, kidney disease, liver disease, and heart disease. Evidence suggests that *H. pylori*, commonly acquired early in life, can provoke persistent inflammatory responses influencing metabolic and cardiovascular health [24,25]. The current findings, which show

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that approximately one-third of participants tested positive for *H. pylori* across various age groups, are in line with epidemiological data indicating that up to two-thirds of the global population may carry the organism [24,26]. Although many studies reveal similar infection frequencies for males and females [27], we observed slight differences in certain age brackets, particularly an elevated rate among middle-aged females compared to their male counterparts. These variations may reflect diverse behavioral or hormonal factors, or differences in exposure that vary by gender [28]. Several studies have documented non-linear trends in *H. pylori* prevalence across age groups. For example, Khan et al [29] reported that while infection rates may rise during childhood and adolescence, they tend to plateau in adulthood. Other reports have sometimes pointed to higher male prevalence [30,31], underscoring the complexity of cultural, genetic, or socioeconomic factors that shape *H. pylori* acquisition [32,33].

In this cohort, *H. pylori* prevalence exceeded 30%, with middle-aged females (40–59 years) having a notably higher rate than their male counterparts. This observation aligns with literature proposing that variations in hygiene, diet, or lifestyle habits can influence infection status [32,33]. Some investigators have noted that the age at which *H. pylori* is acquired may determine the intensity of chronic inflammation and eventual pathology. Infection during childhood may be more likely to lead to severe gastric conditions, while later infection could show more association with duodenal ulceration or milder inflammatory phenomena [34,35]. Although the present study did not track exact age of acquisition, the finding that *H. pylori* prevalence varied by age group suggests a role for differing exposure patterns across the lifespan.

One of the more striking observations was the association between *H. pylori* infection and dyslipidemia. We found that men were more likely to have dyslipidemia, and when we considered *H. pylori* status, a statistically significant connection emerged. Research in other populations, such as in Jimma, Ethiopia, has similarly reported higher rates of abnormal lipid profiles among *H. pylori*-positive individuals, noting rises in total cholesterol, triglycerides, and LDL cholesterol [36]. Dyslipidemia itself is strongly implicated in cardiovascular disease risk [37]. It is likely that the chronic low-grade inflammation caused by *H. pylori* infection promotes lipid metabolism disturbances [38,39], partly via cytokines like tumor necrosis factor-alpha (TNF-α) and interleukin-6 (IL-6) [40,41]. These mediators can disrupt normal lipoprotein lipase activity and impair cholesterol transport [42]. *H. pylori* strains with virulence factors such as CagA may further contribute to metabolic disruptions by altering pathways responsible for cholesterol synthesis, including key enzymes such as squalene epoxidase [42]

Notable gender differences also emerged in obesity rates, with younger females displaying higher obesity percentages. Although no straightforward, one-way correlation to *H. pylori* was evident, the fact that obesity was frequent in certain female subgroups aligns with hypotheses linking chronic infection and metabolic alterations. Some studies show that *H. pylori* may contribute to weight gain by interfering with adipocyte hormones [43], whereas others find an inverse or nonexistent association, suggesting an interplay among genetics, diet, and hormonal factors [44]. The elevated obesity prevalence among younger females with *H. pylori* infection here points to the possible role of hormonal influences and demands further exploration.

Hypertension and DM were both strongly tied to older age ranges in this study. Participants with *H. pylori* infection exhibited higher frequencies of these metabolic conditions, supporting evidence that chronic *H. pylori* infection can promote insulin resistance through heightened production of TNF-α and IL-6 [22]. These pro-inflammatory signals compromise insulin receptor functionality and engender endothelial dysfunction, culminating in increased blood pressure [45]. Cohort studies in other settings have similarly implicated *H. pylori* in elevated risks of type 2 diabetes, especially among males [46]. When DM and HTN co-occur, as is common in older adults, the risk of cardiovascular events escalates considerably, emphasizing the need to consider *H. pylori* status in patients with unexplained metabolic or cardiovascular risk factors.

Kidney, liver, and heart diseases also increased with advancing age, mirroring the general trend that older individuals accumulate greater comorbidity. Although prevalence rates of these organ-

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specific pathologies were relatively modest compared to those of HTN or DM, *H. pylori* positivity correlated with higher frequencies in older age brackets. Some authors attribute these links to systemic inflammation and atherogenic processes accelerated by chronic infection [47]. Associations between *H. pylori* and nonalcoholic fatty liver disease, including elevated liver enzymes, have also been documented [48]. With respect to heart disease, certain investigations propose that *H. pylori* eradication could ameliorate endothelial function and reduce atherosclerotic risk [49]. In this study, older males with both *H. pylori* and dyslipidemia were more likely to manifest heart disease, although the cross-sectional design precludes definitive causal inferences.

Smoking emerged as a crucial determinant of metabolic and cardiovascular health, particularly prevalent among younger males in this study, while negligible in females. Tobacco use substantially exacerbates the inflammatory environment, which in turn may amplify any adverse metabolic effects from *H. pylori* [50]. Given that smoking rates declined in older males, potential harm to vascular and metabolic systems may have been exerted earlier in life, compounding later risks. Future investigations assessing *H. pylori* and metabolic diseases should therefore control for smoking behaviors to isolate the bacterium's specific contributions [50].

Several pathways have been proposed to explain how *H. pylori* could disrupt metabolic homeostasis. Chronic infection is known to elicit immune responses that produce pro-inflammatory cytokines, which interfere with insulin signaling in peripheral tissues and the liver. *H. pylori* may also affect gut microbial composition [51,52]. The gut microbiota is well-recognized as a key regulator of energy extraction and storage [53]. When gut microbes shift toward communities that can harvest more energy from the diet, obesity and insulin resistance may become more likely [53,54]. Although this study did not characterize the gut microbiome, the interaction between *H. pylori* and other gut bacteria deserves further study, especially in populations with high rates of both *H. pylori* infection and obesity.

From a clinical perspective, these results highlight the importance of considering *H. pylori* infection in patients who present with a spectrum of metabolic or cardiovascular risks, including unexplained dyslipidemia, elevated blood pressure, and insulin resistance. If further research confirms that treating *H. pylori* not only resolves gastric problems but also improves metabolic markers, then routine screening for this organism might become more broadly recommended. Public health initiatives aimed at preventing *H. pylori* transmission, such as improving sanitation and public awareness, could yield benefits that go well beyond reducing gastric morbidity, potentially lowering the incidence of metabolic syndrome components as well. Still, it is crucial to acknowledge that many of the current data are cross-sectional. Longitudinal designs, along with randomized controlled trials, are needed to establish whether eradicating *H. pylori* yields sustained improvements in lipid profiles, glycemic control, or blood pressure. Standardized methods for diagnosing *H. pylori*, robust sample sizes, and multivariate models that adjust for factors like smoking, diet, and socioeconomic status will be particularly important. Gender differences should also receive closer scrutiny, given the patterns we observed in obesity, smoking, and *H. pylori* prevalence. Hormonal influences and culturally shaped behaviors may alter both the risk of acquiring *H. pylori* and the infection's systemic effects. Future investigations that incorporate advanced molecular and microbiome analyses might explain precisely how *H. pylori* triggers systemic inflammation and influences pathways that govern metabolism.

5. Conclusions

In conclusion, the study revealed significant correlations between *H. pylori* infection and dyslipidemia, hypertension, and diabetes mellitus. Older participants, especially males, were more prone to dyslipidemia, whereas younger females showed higher obesity rates. Smoking was disproportionately common among younger males, potentially intensifying the inflammatory response associated with *H. pylori*. Although causality remains undetermined, the collective evidence implies that persistent *H. pylori* infection could compound metabolic processes leading to these conditions. Screening and possible eradication therapy could thus represent an avenue for mitigating

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cardiovascular risk. Beyond peptic ulcer disease and gastric cancer, *H. pylori's* possible role in precipitating chronic metabolic disease warrants a broadened perspective in both clinical practice and public health policy. Strategies that concurrently address infection, lifestyle factors, and demographic differences may more comprehensively lessen the burden of chronic illnesses in diverse populations.

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