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Exploring the Link Between *Helicobacter Pylori* Infection Severity and Metabolic Disruptions: The Role of IL-17F as a Key Biomarker

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ABSTRACT

Helicobacter pylori is a Gram-negative, microaerophilic bacterium that colonises. Aims to evaluate the relationship between *H. pylori* infection severity and metabolic disorder. A case-control study was conducted with 180 patients, who were divided into three groups: (Group 1) severe gastritis, (Group 2) moderate gastritis, and (Group 3) healthy controls. In serum, *H. pylori* was detected using nephelometry immunoassay, and enzyme-linked immunoassay (ELISA) determined interleukin-17F (IL-17F). The severe gastritis patients had significantly higher *H. pylori* levels compared to those of moderate gastritis patients (mean difference: 47.083 AU/ml, $p < 0.05$) and healthy controls (mean difference: 117.033 AU/ml, $p < 0.05$). The control group had significantly lower amounts of *H. pylori* than those in the moderate gastritis group (mean difference: -69.950 AU/ml, $p <0.05$). The levels of IL-17F were also significantly higher in the severe gastritis group than the moderate gastritis group (mean difference: 27.9467 ng/ml, $p < 0.05$) and the control group (mean difference: 59.9967 ng/ml, $p < 0.05$). The levels of HbA1c in the severe gastritis group were higher than those in both the moderate gastritis group (mean difference: 3.1217%, $p <0.05$) and the control group (5.0883%, $p <0.05$). The mean levels of RBS in severe gastritis were higher compared to both moderate gastritis (51.800 mg/dl, $p < 0.05$) and the control group (122.833 mg/dl, $p < 0.05$). Results revealed that higher infection severity resulted in higher concentrations of these markers, indicating the contribution of H. pylori to metabolic dysfunction and immune response. These results underscore the need for *H. pylori* eradication to enhance metabolism and immunity.

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1. INTRODUCTION

Helicobacter pylori (H. pylori) is a Gram-negative, microaerophilic bacterium that colonizes the gastric mucosa and infects roughly one-half of the world's population [1]. Worldwide, *H. pylori* has different rates of prevalence, with rates over 70% in Asia, Africa, and South America and less than 40% in North America, Europe, and Australia [2]. Modern studies have reported a global reduction in prevalence from 50.8% in the 1980s–1990s to 43.4% in 2011–2022, but with persistent variability among areas and regions [1]. In the Middle East, especially Iraq, the prevalence of infection has been on the rise, with reported prevalence between 50 and 70% among symptomatic patients [3]. For instance, 63% of adults with dyspeptic symptoms tested positive for *H. pylori* infection in a cross-sectional study in Duhok province by serology and stool antigen testing [3]. An infection rate of 50% was also reported in another study in Zakho City; it was more common among older individuals and in those of lower socio-economic status [4].

H. pylori infection results in chronic inflammation of the gastric mucosa and progression from superficial gastritis to more advanced forms, including atrophic gastritis and intestinal metaplasia, which is associated with increased gastric cancer risk [5]. Gastritis severity is histologically classified by mononuclear and polymorphonuclear cellular infiltration, glandular atrophy, and mucosal destruction [6]. The pathogenicity of *H pylori* is related to the host immune response and its induced inflammation, and H pylori virulence factors, CagA and VacA, act to modify the host immune defence and gastric epithelial integrity [7].

Recent studies have proposed a possible association between *H. pylori* infection and systemic metabolic abnormalities, particularly glucose metabolism [8]. Observational research has also shown that *H. pylori*-infected persons frequently exhibit higher fasting blood glucose and glycated haemoglobin (HbA1C) when compared to uninfected controls, indicating potential involvement in insulin resistance and type 2 diabetes mellitus (T2DM) [9]. These alterations in metabolism might be related to systemic inflammation, oxidative stress, and disturbances in the release of gastric hormones such as ghrelin and leptin [10]. Hemoglobin A1C is a marker of average blood glucose over the past 2 to 3 months and is an established tool in the clinical diagnosis and diabetes monitoring [11]. The random blood sugar (RBS) estimates only a single point in time and is generally employed for primary screening [9]. The association between H. pylori infection and glucose metabolic control is still an area of active investigation, and some evidence indicates that glucose metabolism can improve after *H. pylori* eradication [12].

Interleukin-17F (IL-17F) is a proinflammatory cytokine secreted by Th17 cells and is crucial in mucosal immunity and inflammatory activities [13]. IL-17F enhances the recruitment of neutrophils and induces other cytokines, resulting in tissue inflammation and protection against bacterial infection [14]. Increased levels of serum and gastric mucosal IL-17F have been observed in *H. pylori*-infected patients and are associated with the severity of gastritis [15]. Furthermore, IL-17F gene variants are associated with susceptibility to gastric inflammation and cancer induction [16].

Demographic factors, including sex and body mass index (BMI), affect *H. pylori* infection risk and severity [17]. Epidemiological studies have shown that men are more likely to be infected and develop more severe gastric inflammation than women, possibly related to hormonal and lifestyle differences [18].

Given these multifactorial interactions, this study aims to investigate the association between *H. pylori* infection severity and metabolic markers, including HbA1C, RBS, and IL-17F levels, while controlling for gender, age, and weight differences.

2. METHOD

Study design: The study was a case-control. All samples were collected from October 2024 to March 2025. The research included 180 individuals (90 men and 90 women), divided into three groups. The first group included 60 patients with a confirmed diagnosis of *H. pylori*-positive with severe gastritis, while the second group contained 60 who had *H. pylori*-positive with moderate gastritis. The third group was 60 individuals who were healthy controls.

Inclusion criteria: The research subjects included patients diagnosed with severe and moderate gastritis with confirmed *H. pylori* infection. Also, healthy individuals were included as an apparent control for comparison cases.

Exclusion criteria: Subjects who experienced the following conditions were omitted from the study. Patients employing anti-inflammatory medications that exclude steroids. Patient with concurrent inflammatory bowel disease. Cardiovascular illnesses comprise maladies such as coronary artery disease, peripheral vascular disease, hypertension, and stroke. Individuals diagnosed with rheumatoid arthritis.

Quantitative detection of *Helicobacter pylori* using a Nephelometry immunoassay: The *Helicobacter pylori* infection was diagnosed according to the protocol procedure delineated by Hipro (Hipro Biotechnology Co., Ltd.). It utilizes Nephelometry Immunoassay technology to quantitatively detect *Helicobacter pylori* antibodies in human serum. This approach relies on the unique interaction between antibodies in the patient's blood and *H. pylori* antigens, resulting in the development of immune complexes. Immune complex luminescence, when exposed to a focused light beam. A nephelometric analyzer quantifies the variation in scattered light intensity, while the antibody concentration is determined using a standard calibration curve derived from known concentrations. This exposure offers a precise and accurate method to evaluate the immune response to *H. pylori infection*. It has been extensively used in diagnosing *H. pylori*-related gastritis and peptic ulcer disease.

Measurement of human IL-17F using ELISA: The methods and principles for Human Interleukin 17F (IL-17F) using the ELISA Kit from BT Laboratory are: The ELISA concept (Enzyme-Linked Immunosorbent Assay) involves a plate pre-coated with the antibody IL-17F. The sample containing IL-17F is introduced to the wells and adheres to the immobilized antibody. A biotinylated anti-IL-17F antibody is added, binding to the biotinylated antibody via a Streptavidin-HRP conjugate. After washing to remove residual unbound components, a substrate solution is introduced and reacts to generate a colorimetric change corresponding to the concentration of IL-17F, which is detected at a wavelength of 450 nm. The test entails introducing the samples and standards into the designated wells, incubating them with the reagent solution, performing a wash, and then adding the substrate solution. The data is recorded as absorbance at OD, and the findings are analyzed with a standard curve derived from known quantities of IL-17F.

Measurement of glycated hemoglobin (HbA1c) using the turbidimetric assay: The HbA1c turbidimetric immunoassay was applied according to the protocol kit delineated by BIOLABO, which utilizes photometric measurement of turbidity, which correlates to an antigen-antibody response, using the end-point technique at 600 nm to quantify HbA1c in whole blood. Assay procedure: Reagents are produced for each standard and sample, introduced into designated tubes, mixed, and incubated at 37°C. Following incubation, the absorbance was measured at 600 nm using the appropriate spectrophotometer. Determining findings necessitates the establishment of a standard sample curve to evaluate HbA1c concentration.

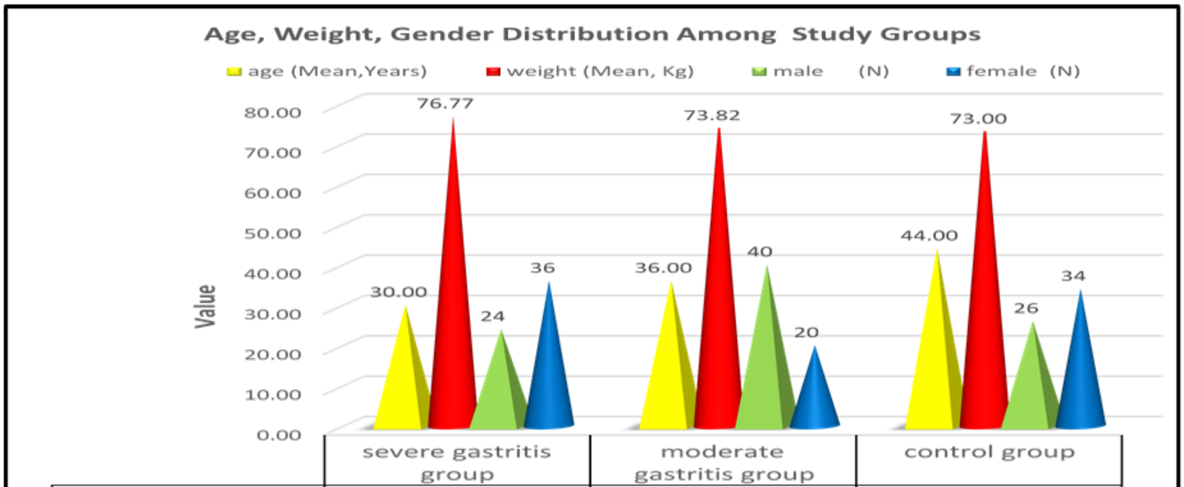
Quantitative glucose measurement using GOD-PAP technology: The Glucose GOD-PAP technique, developed by BIOLABO, operates on the idea that glucose is enzymatically oxidized by glucose oxidase (GOD) to produce gluconic acid and hydrogen peroxide. This hydrogen peroxide spontaneously reacts with 4-amino-antipyrine (PAP) and chloro-4-phenol in the presence of peroxidase (POD), forming a red-hued quinone imine complex. The intensity of the red color, which is directly proportional to the glucose content in the sample, is measured at 500 nm. The assay relies on three reagents: R1 (comprising enzymes and phosphate buffer), R2 (chromogen), and R3 (glucose standard). The reagents are combined and incubated, followed by the absorbance measurement to conduct the test. This approach is used for the quantitative determination of glucose in human serum.

Statistical analysis: Statistical analysis was performed in Statistical Package for the Social Sciences (SPSS) to determine the association between the degree of H. pylori infection and some metabolic and immune biomarkers. Descriptive statistics were used to summarize demographic variables, with ANOVA used to test for differences among groups on age, weight, H. pylori levels, IL-17F, HbA1c, and RBS. Where group differences were observed, post-hoc Tukey tests related to the HSD tests were conducted. Pearson's correlation measured the strength and direction of the relationships between variables. The influence of H. pylori titer on HbA1c and IL-17F levels was estimated by regression analysis. In addition, decision tree analysis was applied to spot some trends and relationships of infection severity with the biomarker levels.

3. RESULTS

Dimorphic characteristics of the age, weight, and gender distributions according to the study groups. The first group had severe gastritis, the second had moderate gastritis, and the third group was a control group. Shown as Figure 1. The average age was lowest in the severe gastritis group (30 y), and the average weight was highest in the severe gastritis group (76.77 kg). Sex did differ among the groups, with the severe and control groups containing more females and the moderate group more males.

Results of the ANOVA test showed a significant difference between groups for all of the studied variables. A significant difference was found between the groups regarding age, with a p-value lower than 0.05, as shown in Table 1. Showing that age has a statistically significant effect between the groups. Furthermore, weight showed marked disparity between the groups, with a P value 0.000, presenting a noticeable statistical difference. Similarly, for *H. pylori* levels, a significant difference was found among the groups ($P < 0.05$), indicating that *H. pylori* levels varied significantly among the study groups. A highly significant difference was observed in markers of IL17F contents, and the effect was substantial between the groups. In addition, for HBAIC, the analysis results showed remarkable differences among the groups, which makes it clear that the variable has had an effect. The differences between the groups for the RBS levels likewise indicated statistically significant results, P=.000, which confirmed the existence of substantial statistical divergence. The laboratory values significantly differed between the groups for all studied variables, with overt and clinical relevance for the respective variables.



	severe gastritis group	moderate gastritis group	control group
age (Mean,Years)	30.00	36.00	44.00
weight (Mean, Kg)	76.77	73.82	73.00
male (N)	24	40	26
female (N)	36	20	34

Figure 1: Dimorphic distributions of age, weight, and gender among the study groups.

The table1: presents the ANOVA results for group comparisons across the variables (age, weight, *H. pylori*, IL17F, HBAIC, and RBS). The results show significant differences between the groups for all variables, indicating notable effects and statistical variability across the studied parameters.

Table 1: ANOVA Results for Group Comparisons of Demographics and Biomarkers

ANOVA						
Variables		Sum of Squares	df	Mean Square	F	Sig.
age (year)	Between Groups	5920.000	2	2960.000	909.583	0.000
	Within Groups	576.000	177	3.254		
	Total	6496.000	179			
weight (kg)	Between Groups	471.144	2	235.572	58.258	0.000
	Within Groups	715.717	177	4.044		
	Total	1186.861	179			
H. pylori (AU/ml)	Between Groups	416132.878	2	208066.439	42292.679	0.000
	Within Groups	870.783	177	4.920		
	Total	417003.661	179			
IL17F (ng/ml)	Between Groups	108156.374	2	54078.187	8679.138	0.000
	Within Groups	1102.856	177	6.231		
	Total	109259.230	179			
HBAIC %	Between Groups	790.074	2	395.037	434.805	0.000
	Within Groups	160.811	177	0.909		
	Total	950.886	179			
RBS (mg/dl)	Between Groups	456340.044	2	228170.022	36266.248	0.000
	Within Groups	1113.600	177	6.292		
	Total	457453.644	179			

The post-hoc ANOVA (Tukey HSD) multiple comparisons among pairs of groups showed that the groups were significantly different regarding the variables. Shown as Table 2. Age (years) in the Severe gastritis group was substantially younger than moderate gastritis (mean difference = -6.000 years, p = 0.000) and the control group (mean difference = -14.000 years, p = 0.000). The mean ages of the control campers were lower than those of the moderate gastritis groups (mean difference = 8.000 years, p = 0.000). Weight was significantly higher in the Severe gastritis group than in the control group (mean difference = 3.767 kg, p = 0.000) and the moderate gastritis group (mean difference = 2.950 kg, p = 0.000). The average weight for the patients with moderate gastritis was not much different compared to the control, the same as the patients with moderate weighing mild and moderate gastritis patients separately and as whole versus the power did not have significant weight differences (for moderate versus control a mean difference was - 0.817 kg, p = 0.070-approaching statically substantial).

In the severe gastritis, the *H. pylori* levels were higher than those of the control group (difference of mean = 47.083 AU/ml, p = 0.000. difference of mean = 117.033 AU/ml, p = 0.000, respectively. The control group had significantly lower *H. pylori* measurements than the moderate group (mean difference = -69.950 AU/ml, p = 0.000).
IL-17F of participants with severe gastritis was significantly higher than that with moderate gastritis (mean difference = 27.9467 ng/ml, p = 0.000) and the control group (mean difference = 59.9967 ng/ml, p = 0.000). The control group showed a significant decrease in IL-17F compared with the moderate gastritis group (mean difference: -32.0500 ng/ml, p = 0.000). The levels of HbA1c in severe gastritis were higher than those in the moderate gastritis group (mean difference = 3.1217%, p = 0.000) and the normal control group (mean difference = 5.0883%, p = 0.000). The HbA1c level of the control group was significantly lower than that of the moderate gastritis group (mean difference = -1.9667%, p = 0.000).

The RBS levels in the severe gastritis group were also significantly higher than in the moderate gastritis group (mean difference = 51.800 mg/dl, p = 0.000) and the control group (mean difference = 122.833 mg/dl, p = 0.000). Mean RBS levels were lower in the control group compared to the moderate gastritis group (mean differences, -71.033 mg/dl, p = 0.000).

Table 2: shows post-hoc comparisons among severe gastritis, moderate gastritis, and controls with Tukey HSD for age, weight, *H. pylori*, IL17F, HBAIC, and RBS. There were significant differences among the measures, with severe gastritis having higher measures for most attributes than moderate gastritis and control, respectively.

Table 2: Post-Hoc Tukey HSD Comparisons for Demographics and Biomarkers among pairs groups

Multiple Comparisons							
Tukey HSD							
Dependent Variable	(I) Group	(J) Group	Mean Difference (I-J)	Std. Error	Sig.	95% Confidence Interval Lower Bound	Upper Bound
age (year)	severe gastritis	moderate gastritis	- 6.000	0.329	0.000	- 6.78	- 5.22
		Control	- 14.000	.329	0.000	- 14.78	- 13.22
	Control	moderate gastritis	8.000	0.329	0.000	7.22	8.78
weight (kg)	severe gastritis	moderate gastritis	2.950	0.367	0.000	2.08	3.82
		control	3.767	0.367	0.000	2.90	4.63
	Control	moderate gastritis	- 0.817	0.367	0.070	- 1.68	0.05
H. pylori (AU/ml)	severe gastritis	moderate gastritis	47.083	0.405	0.000	46.13	48.04
		control	117.033	0.405	0.000	116.08	117.99
	Control	moderate gastritis	- 69.950	0.405	0.000	- 70.91	- 68.99
IL17F (ng/ml)	severe gastritis	moderate gastritis	27.9467	0.4557	0.000	26.869	29.024
		control	59.9967	0.4557	0.000	58.919	61.074
	Control	moderate gastritis	- 32.0500	0.4557	0.000	- 33.127	- 30.973
HBAIC %	severe gastritis	moderate gastritis	3.1217	0.1740	0.000	2.710	3.533
		control	5.0883	0.1740	0.000	4.677	5.500
	Control	moderate gastritis	- 1.9667	0.1740	0.000	- 2.378	- 1.555
RBS (mg/dl)	severe gastritis	moderate gastritis	51.800	0.458	0.000	50.72	52.88
		control	122.833*	.458	.000	121.75	123.92
	Control	moderate gastritis	- 71.033	0.458	0.000	- 72.12	- 69.95
*. The mean difference is significant at the 0.05 level.							

Pearson's correlation analysis indicates statistically significant associations between the variables (P = 0.000). Shown as Table 3. There is a strong reverse association of age with weight (− 0.591), H. pylori (− 0.948), IL17F (− 0.950), HBAIC (− 0.837), and RBS (− 0.951); age increases, then all these decreases. The weight has a moderate positive correlation with H. pylori (0.570), IL17F (0.592), HBAIC (0.550), and RBS (0.575), clearly indicating that the weight increases with these variables. Gastric *H. pylori* has a highly positive correlation with IL17F (0.990) and RBS (0.999), which suggests that higher levels of H. pylori are correlated with an increase in the levels of these markers. Likewise, IL17F correlates significantly with RBS (0.992) and HbA1C (0.894), signifying that the higher the IL17F, the higher the RBS and HbA1C. Strong positive correlation between HBA1C-RBS Pair-wise comparison of HBA1C with RBS also exhibits a strong positive relation (0.899), which indicates a strong relation between RBS and HBA1C. It is clear from the results that the variables under study are highly interdependent, and several significant correlations between factors were observed, which imply that they are related in meaningful ways.

Table 3: The Pearson correlation was performed between age, weight, *H. pylori*, IL17F, HbA1c, and RBS. Age had strong negative correlations with all other variables. Weight had a moderate positive association with *H. pylori*, IL17F, HbA1c, and RBS. Correlations. A significant positive association was identified between *H. pylori*, IL17F, HbA1C, and RBS.

Table 3: Pearson Correlation Analysis of Demographics and Biomarkers.

Correlations							
		age (year)	weight (kg)	*H. pylori* (AU/ml)	IL17F (ng/ml)	HBAIC %	RBS (mg/dl)
age (year)	Pearson Correlation	1	- 0.591	- 0.948	- 0.950	- 0.837	- 0.951
	Sig. (2-tailed)		0.000	0.000	0.000	0.000	0.000
weight (kg)	Pearson Correlation	- 0.591	1	0.570	0.592	0.550	0.575
	Sig. (2-tailed)	0.000		0.000	0.000	0.000	0.000
H. pylori (AU/ml)	Pearson Correlation	- 0.948	0.570	1	0.990	0.892	0.999
	Sig. (2-tailed)	0.000	0.000		0.000	0.000	0.000
IL17F (ng/ml)	Pearson Correlation	- 0.950	0.592	0.990	1	0.894	0.992
	Sig. (2-tailed)	0.000	0.000	0.000		0.000	0.000
HBAIC %	Pearson Correlation	- 0.837	0.550	0.892	0.894	1	0.899
	Sig. (2-tailed)	0.000	0.000	0.000	0.000		0.000
RBS (mg/dl)	Pearson Correlation	- 0.951	0.575	0.999	0.992	0.899	1
	Sig. (2-tailed)	0.000	0.000	0.000	0.000	0.000	
**. Correlation is significant at the 0.01 level (2-tailed).							

The included decision tree analyses demonstrate a robust and statistically significant relationship between *Helicobacter pylori* antibody levels and several metabolic and immune biomarkers, such as HbA1C, RBS, and IL-17F, shown in Figures 2 and 3. The first analysis *H. pylori* titers >120 AU/ml showed to have significantly positive influence on HbA1C levels (mean = 9.216%) as compared to the maintained lower titers which exhibited a negative impact on glycemic control, indicating well treatment compliance as the increase in titers it found within the category of advancement of infection severity (P = 0.000). IL-17F, another pro-inflammatory cytokine, was also significantly elevated due to increased *H. pylori* titers. Subjects with titers >120 AU/ml had significantly increased levels of IL-17 (mean = 114.893 ng/ml) compared with low and moderate antibody titers. Such a result highlights the *H. pylori*-related immune activation, which may participate in both gastric and systemic inflammation.

These findings highlight the possible contribution of *H. pylori* to gastrointestinal disease and the induction of systemic metabolic and immune abnormalities, suggesting its importance as a target for timely prevention and treatment.

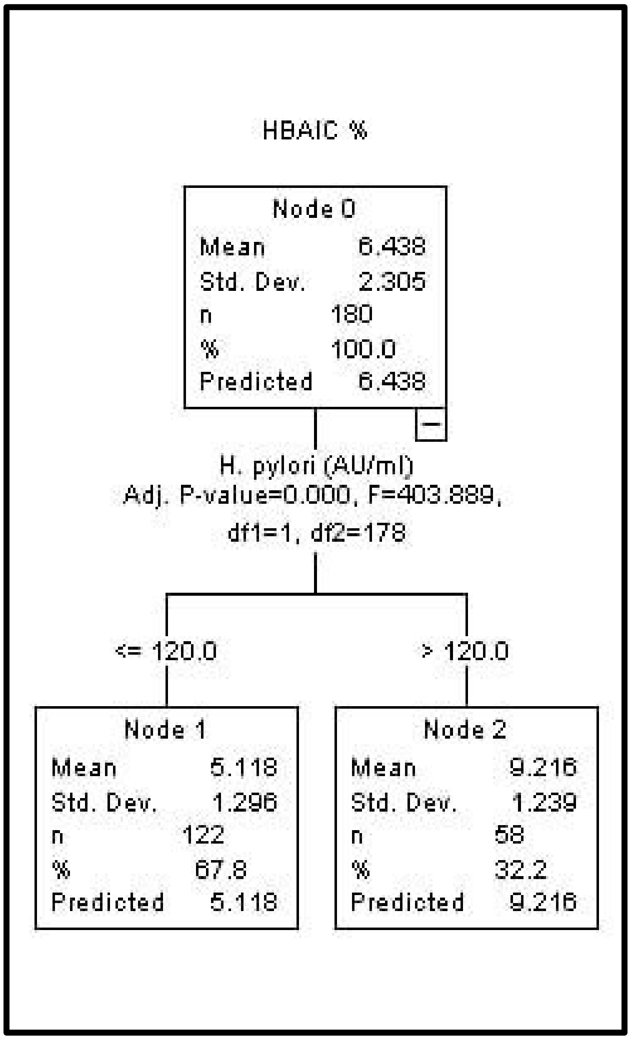


Figure 2: Association Between *Helicobacter Pylori* Infection Severity and HbA1C Levels: Evidence of a Metabolic Impact

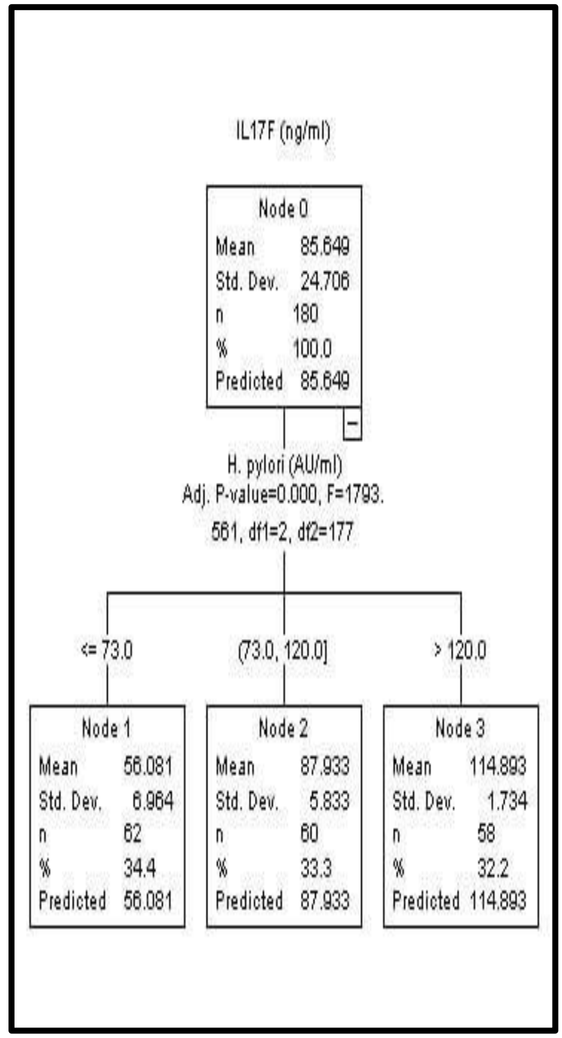


Figure 3: Elevated IL-17F Levels in Response to Increasing *Helicobacter pylori* Titers: Evidence of Immune Activation

4. DISCUSSION

The differences in demographics between the severe gastritis, moderate gastritis and control groups, and most notably, the differences in age, weight and gender were remarkable. In detail, the age of patients in the severe gastritis group was the youngest, and that of the normal control group was the oldest. This result agrees with studies that the infectious rate of *H. pylori* is higher in young people than in older people in developing countries due to the environment and the sanitary conditions; that is, early-life exposure is a significant cause[19]. Nonetheless, other studies, including those that reported re-infection or chronic *H. pylori* infection to account for higher rates in older patients, might explain the higher average age in the control group [20]. The interesting findings in the present study were that patients with severe gastritis were significantly heavier than those in the moderate gastritis and control groups. The findings agree with the study, which found that *H. pylori* infection can influence gastric emptying and appetite regulation and may drive weight gain, at least in a subset of patients, particularly in the presence of other metabolic disturbances such as obesity [21]. In contrast, the work reported that chronic *H. pylori* infection is more frequently related to loss of weight and malnutrition, caused by its gastrointestinal symptoms, similar to the general expectation of reduced body weight due to *H. pylori* infection [22]. The disagreement in weight results in this study and other reports could result from the interaction of *H. pylori* infection and other comorbid conditions, such as metabolic syndrome or obesity, that might modulate the body's response to the infection. Additional studies are necessary to elucidate the effect of *H. pylori* infection on weight and whether its impact is affected by the types of patients and comorbidities.

There was also a significant disparity in *H. pylori* levels between the groups, which were highly valued in the severe gastritis group. This result is consistent with another study, which reported the relationship since greater titers of *H. pylori* antibodies were related to more severe gastritis [23]. Furthermore, *H. pylori* is associated with developing superficial to more severe forms of gastritis, such as atrophic inflammation. These findings confirm the present study and highlight the association between *H. pylori* and the intensity of gastric inflammation and the involvement of more severe gastric disease [24]. Also, the results of this study are well in agreement with the idea that the extent of gastric inflammation, as affected by *H. pylori* infection, may depend on strain differences of the bacterium and the host's immune response [25].

Higher amounts of IL-17F were observed in severe gastritis, which agrees with other studies showing it is involved in inflammation in *H. pylori* infection [26]. IL-17F is a pro-inflammatory cytokine secreted by Th17 cells, and its high levels are frequently found in chronic *H. pylori*-infected gastritis [26]. Reinforce these observations by showing that IL-17F is critically involved in fine-tuning the host response to *H. pylori* and directly correlates with the degree of gastritis [27]. Similarly, a study has reported that high levels of IL-17F are associated with more prominent immune activation and gastric inflammation, especially in patients with more advanced gastritis [28].

The patients with severe gastritis were found to have higher HbA1c and RBS levels compared with the patients with moderate gastritis and controls. This result aligns with the recent data indicating an association between *H. pylori* infection and type 2 diabetes [29]. The persistent inflammatory response associated with the *H. pylori* infection is thought to be responsible for insulin resistance and disruption in glucose metabolism. The evidence linking *H. pylori* infection to high levels of HbA1c indicates that it may be involved in the pathogenesis of type 2 diabetes by promoting systemic inflammation, agreement with the other study observed that H. pylori-infected people often show elevated blood glucose levels, especially when presenting with metabolic syndrome [30].

5. CONCLUSION

This study concluded a significant correlation between the severity of *Helicobacter pylori* infection and changes in metabolic and immune biomarkers, including HbA1c, RBS, and IL-17F. Results revealed that higher infection severity resulted in higher concentrations of these markers, indicating the contribution of *H. pylori* to metabolic dysfunction and immune response. These results underscore the need for *H. pylori* eradication to enhance metabolism and immunity.

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