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The Relationship Between Helicobacter Pylori And Interleukin 1 Beta Level In Iraqi Patients With Diabetic And Thyroid Disorder

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Abstract The study was conducted on 88 influencers, 46 of whom were male, 42 samples from Haddith that were still present and still visible, diabetes, those infected with H. pylori, and uninfected persons, and their ages were between (35-70) years from (1/1/2023 - 1/1/2024) From Al-Diwaniyah General Hospital in Al-Diwaniyah Governorate, where numbers were collected from visitors and healthy people and were separated by the Central Croatian device, then the biochemical test was measured, which showed (Interleukin-1-beta, FBS, T3, T4, TSH).
The results of the current research showed a significant increase in the levels of (Interleukin-1-beta, FBS.) in the blood serum of patients compared to the control group, with no significant differences in the levels of (T3, T4, TSH) in both groups, and at its level, the probability of $P \leq 0.05$.

Key words / Interleukin-1-beta, FBS,T3,T4,TSH.

Introduction

Helicobacter pylori (H. pylori) infection has been increasingly recognized as a potential contributor to autoimmune thyroid diseases (AITD). Multiple studies have demonstrated a positive correlation between H. pylori infection and AITD, with particular emphasis on the role of cytotoxin-associated gene A (CagA) positive strains [(1,2)]. The presence of CagA-expressing H. pylori has been linked to more severe progression of AITD, suggesting a strain-specific impact on thyroid autoimmunity. This association is thought to be mediated through molecular mimicry, where bacterial antigens cross-react with thyroid tissues, and through the modulation of the host immune response. The resulting chronic inflammation and altered cytokine profiles may contribute to the initiation or exacerbation of thyroid autoimmunity, highlighting the complex interplay between infectious agents and endocrine disorders.
The prevalence of Helicobacter pylori (H. pylori) infection among individuals with diabetes has been a subject of significant research interest. A comprehensive meta-analysis has revealed a pooled prevalence of 54% in patients with diabetes [(3)]. This finding suggests that more than half of diabetic individuals may harbor H. pylori, highlighting the potential importance of this bacterial infection in the context of metabolic disorders. The prevalence rate in diabetic populations is notably higher than in the general population, raising questions about the bidirectional relationship between H. pylori infection and diabetes. Factors such as altered gastric motility, changes in glucose metabolism, and impaired immune responses in diabetic patients may contribute to this increased prevalence. Understanding the epidemiology of H. pylori infection in diabetic populations is crucial for developing targeted screening and management strategies.
The relationship between H. pylori infection and autoimmune disorders extends to type 1 diabetes mellitus (T1DM), where a significant association with thyroid autoimmunity has been observed. Research has revealed elevated levels of H. pylori IgG antibodies in T1DM patients, along with increased thyroid-stimulating hormone (TSH), anti-thyroglobulin, and anti-thyroid peroxidase antibodies [(4)] . This constellation of findings suggests a potential synergistic effect between H. pylori infection and the autoimmune processes underlying both T1DM and thyroid dysfunction. Moreover, a study focusing on pediatric populations with AITD found a high prevalence of H. pylori infection, which was associated with lower triiodothyronine (T3) levels [(3)] . These observations underscore the potential impact of H. pylori on thyroid hormone balance, particularly in younger individuals with developing immune systems.
In contrast to diabetic populations, individuals with thyroid disorders exhibit an even higher prevalence of H. pylori infection. Studies have reported strikingly high rates, with 93.3% prevalence in cases of Hashimoto's thyroiditis (HT) and 92.7% in Graves' disease (GD) [(1)]. These figures underscore a potentially significant relationship between H. pylori colonization and autoimmune thyroid conditions. The near-universal presence of H. pylori in these thyroid disorders suggests that the bacterium may play a role in the pathogenesis or progression of autoimmune thyroid diseases. The mechanisms underlying this high prevalence may involve shared genetic susceptibilities, environmental factors, or direct effects of H. pylori on thyroid function. These findings emphasize the need for healthcare providers to consider H. pylori infection when managing patients with autoimmune thyroid disorders.
The presence of H. pylori infection has been consistently associated with autoimmune thyroid diseases, particularly Hashimoto's thyroiditis and Graves' disease [(5)]. This significant positive relationship suggests that H. pylori may contribute to the development or exacerbation of thyroid autoimmunity. Several mechanisms have been proposed to explain this association, including molecular mimicry between H. pylori antigens and thyroid tissues, bystander activation of autoreactive T cells, and modulation of the host immune response. The chronic inflammatory state induced by H. pylori infection may also play a role in breaking thyroid self-tolerance. Furthermore, the potential role of specific

H. pylori virulence factors, such as CagA, in triggering or perpetuating thyroid autoimmunity warrants further investigation. Understanding these associations could lead to novel approaches in the prevention and treatment of autoimmune thyroid diseases.

Helicobacter pylori infection in individuals with diabetes mellitus has been associated with distinct alterations in cytokine profiles. Research has demonstrated significant changes in pro-inflammatory and anti-inflammatory cytokines, particularly interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-α), and interleukin-10 (IL-10) [(1)]. These cytokine alterations are of particular concern in the diabetic population due to their potential impact on renal function. The heightened inflammatory state induced by H. pylori infection, as evidenced by elevated IL-6 and TNF-α levels, may contribute to the progression of diabetic nephropathy. Conversely, changes in anti-inflammatory cytokines like IL-10 may compromise the body's ability to regulate inflammatory responses. The complex interplay between H. pylori infection, diabetes, and cytokine dysregulation underscores the need for comprehensive management strategies that address both glycemic control and potential infectious complications.

The complex interplay between H. pylori infection, cytokine dysregulation, and the pathogenesis of diabetic and thyroid disorders highlights the need for a multifaceted approach to patient care. The chronic inflammatory state induced by H. pylori, characterized by elevated pro-inflammatory cytokines, may exacerbate existing metabolic and autoimmune conditions. In diabetic patients, the increased inflammatory burden could accelerate the progression of microvascular complications, particularly diabetic nephropathy. For individuals with autoimmune thyroid diseases, the H. pylori-induced cytokine alterations may perpetuate or amplify the autoimmune response against thyroid tissue. Furthermore, the local cytokine changes in the gastric mucosa underscore the importance of vigilant monitoring of H. pylori-infected patients for the development of gastric pathologies. These findings collectively emphasize the need for comprehensive management strategies that address not only the primary diabetic or thyroid disorder but also consider the potential impact of H. pylori infection on disease progression and treatment outcomes. Through the high level (Interleukin-1-beta, FBS), the current research aims to The relationship between Helicobacter pylori and interleukin 1 beta level in Iraqi patients WITH diabetic and Thyroid Disorder

Collection of specimens

The study was conducted on 88 influencers, 46 of whom were male, 42 samples from Haddith that were still present and still visible, diabetes, those infected with H. pylori, and uninfected persons, and their ages were between (35-70) years from (1/1/2023 - 1/1/2024) From Al-Diwaniyah General Hospital in Al-Diwaniyah Governorate, where numbers were collected from visitors and healthy people and were separated by the Central Croatian device, then the biochemical test was measured, which showed (Interleukin-1-beta, FBS, T3, T4, TSH).

• **Estimating levels FBS in blood serum:**

The level of FBS in blood serum was measured by the Spanish company BioSystems, using an assay kit prepared using the enzymatic method followed by Young (2001) [(6)].

• **Estimating the levels of immune and physiological indicators in blood serum:**

The concentration of each of the levels of (IL-1β, T3, T4, and TSH) was measured by adopting the ELISA technique (Sandwich) and by following the ready-made steps indicated in the custom analysis kit, and it differs from one device to another and according to its manufacturer.

Statistical Analysis

The process of collecting data for the samples used for the study and analyzing them statistically was done using the SPSS system by extracting the arithmetic mean and standard deviation. The test was also used to analyze the differences between the group of patients and healthy people. Significant differences were chosen for these groups under a probability level of $P \leq 0.05$.

Result and Desiccation

• Measuring the levels of biochemical variables for the samples under study:

Table No. (1) shows the mean ± standard deviation of the biochemical variables for the samples under study

With H. pylori infection (Total=41; F=25 & M=16)						**Without H. pylori infection (Total= 47; F=21 & M=26)**				
	IL1-β (pg/mg protein)	**FBS (mg/dL)**	**T4 (µg/dL)**	**T3 (ng/dL)**	**TSH (µlU/mL)**	**IL1-β (pg/mg protein)**	**FBS (mg/dL)**	**T4 (µg/dL)**	**T3 (ng/dL)**	**TSH (µlU/mL)**
Female (n=46)	**93.14±32.90**	**224.44±65.87**	**17.42±4.63**	**194.58±41.63**	**0.19±0.08**	**57.47±29.57**	**231.95±101.90**	**17.40±7.52**	**196.36±79.39**	**0.15±0.10**
Male (n=42)	**90.90±50.02**	**221.25±113.66**	**18.84±9.48**	**198.27±98.54**	**0.18±0.10**	**69.87±25.21**	**212±49.56**	**17.32±2.63**	**195.22±13.46**	**0.17±0.08**

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The results showed a significant increase in both levels (Interleukin-1-beta, FBS). In the blood serum of patients infected with H. pylori and diabetes compared with the control group, there was a significant decrease in the level of (T3, TSH, T4) in both groups, as in the following figures.

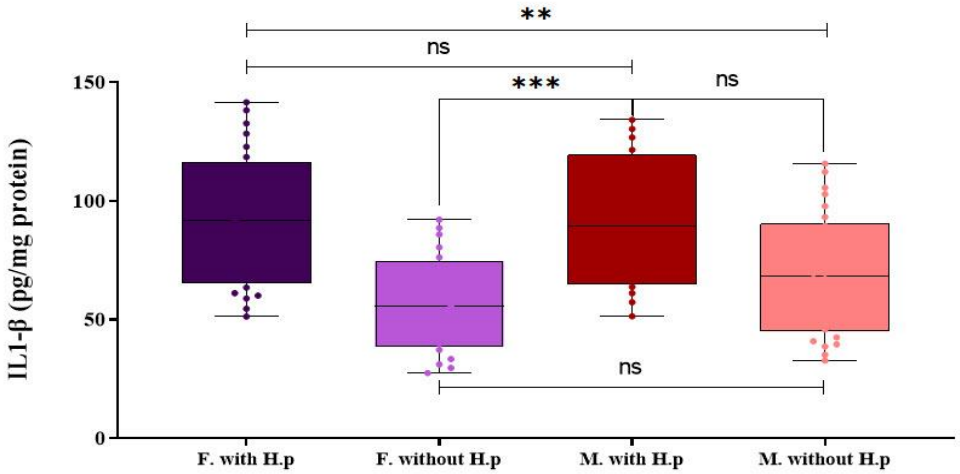

Figure (1) : IL-1β in the blood sera of the samples under study

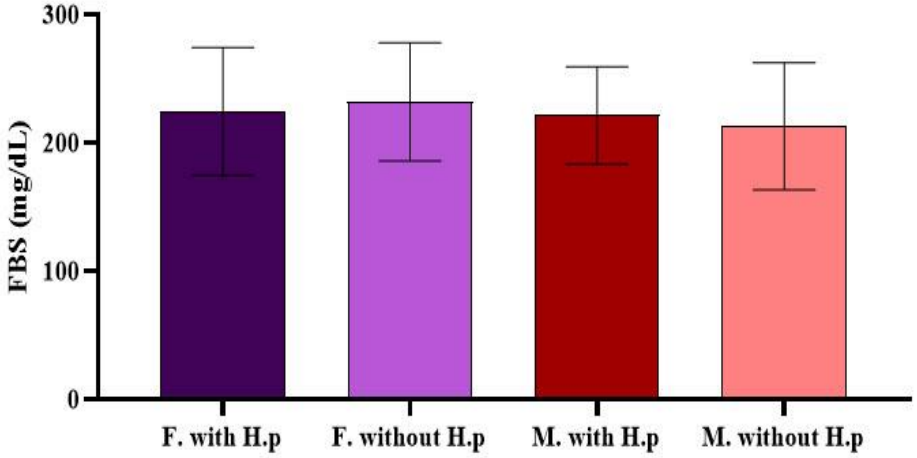

Figure (2) : FBS in the blood sera of the samples under study

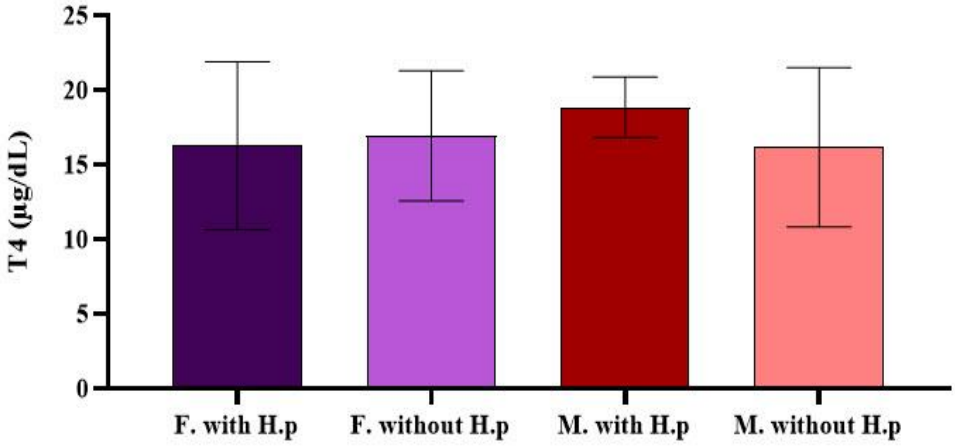

Figure (3) : T4 in the blood sera of the samples under study

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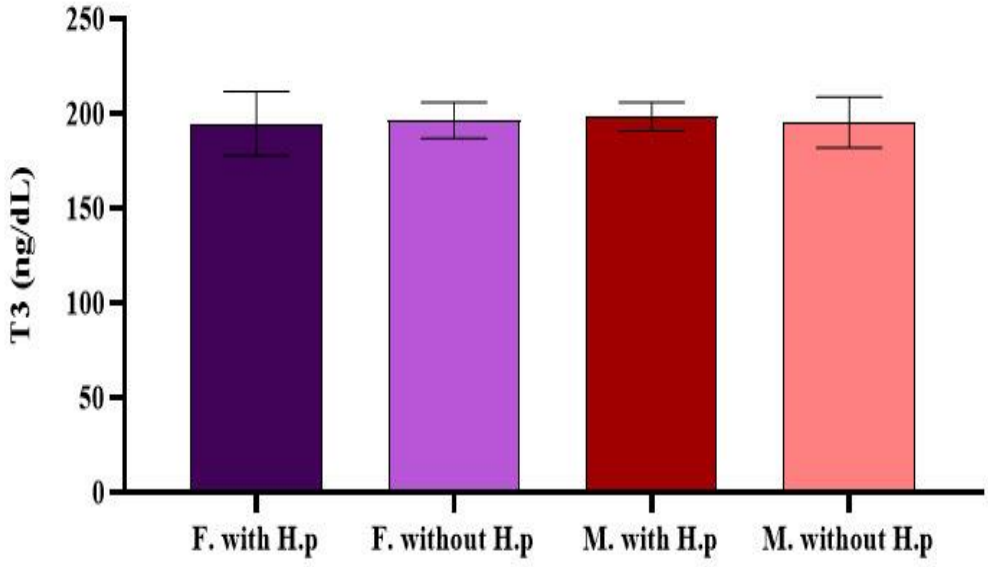

Figure (4) : T4 in the blood sera of the samples under study

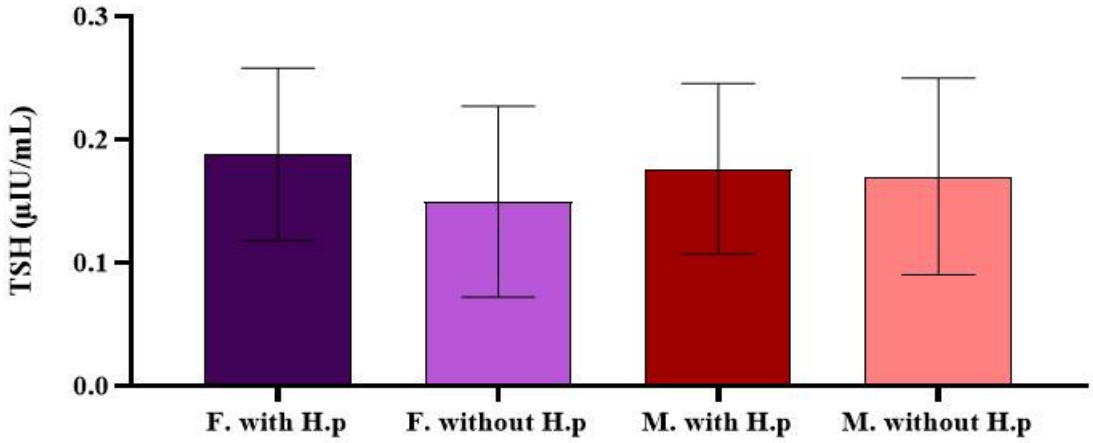

Figure (5) : T4 in the blood sera of the samples under study

Desiccation

The present investigation reveals a substantial augmentation in the circulating levels of the pro-inflammatory cytokines interleukin-1 beta (IL-1β) among individuals harboring Helicobacter pylori infection in comparison to uninfected controls. This elevated pro-inflammatory milieu was consistently observed across both male and female participants, underscoring a robust association between H. pylori infection and a systemic inflammatory response. These findings contribute to the growing body of evidence implicating H. pylori as a potential instigator of chronic inflammation.

The marked elevation of IL-1β observed in H. pylori-positive individuals is consistent with the established paradigm that chronic H. pylori infection triggers a persistent inflammatory response within the gastric mucosa. These pro-inflammatory cytokines, as pivotal mediators of inflammation, have been extensively implicated in the pathogenesis of a spectrum of gastrointestinal disorders, including peptic ulcer disease and gastric adenocarcinoma, both of which are well-documented sequelae of chronic H. pylori infection. Our findings thus corroborate the notion that H. pylori-induced inflammation extends beyond the gastric mucosa to elicit a systemic inflammatory response. The observed positive association between H. pylori infection and elevated levels of IL-1β and TNF-α is corroborated by a substantial body of literature. Several studies have consistently reported increased circulating levels of these pro-inflammatory cytokines in individuals harboring H. pylori [7,8,9]. These findings collectively support the notion of a systemic inflammatory response triggered by H. pylori infection. It is noteworthy, however, that a subset of studies, exemplified by some stadies, have failed to detect a significant association between H. pylori status and cytokine levels [10,11].

Analyzing metabolic parameters and thyroid function tests in the context of H. pylori infection has yielded intriguing insights. Within the scope of our study, an intriguing trend was observed where fasting blood sugar (FBS) levels were elevated in participants with H. pylori infection compared to those without. However, it is critical to note that these differences did not reach statistical significance. This finding suggests that while there may be a tendency for higher

FBS in the presence of H. pylori, the effect is not pronounced enough to conclude a definitive impact of the infection on glycemic control.

The lack of significant differences in FBS levels aligns with the complex and sometimes contradictory findings in existing literature regarding the role of H. pylori in glucose metabolism. Some studies have proposed that H. pylori infection may contribute to insulin resistance and, consequently, elevated blood sugar levels. However, the mechanisms underlying this association are not fully understood, and the relationship appears to be influenced by multiple factors, including the host's immune response, genetic predispositions, and environmental influences.

Comparing our findings to those of other studies reveals a heterogeneous landscape. Research such as Eslami et al., 2017 has indicated a stronger correlation between H. pylori infection and increased FBS levels, suggesting a potential contributory role of the infection in the development of insulin resistance or type 2 diabetes [(12).] On the other hand, several stadies have found no significant association, which is more in line with our results [(13,14,15)]. The variation in these findings may be attributable to differences in study design, population demographics, the criteria for defining H. pylori infection, and the methods used to assess FBS.

Our study has demonstrated that thyroid-stimulating hormone (TSH) levels do not significantly differ between individuals with and without H. pylori infection, irrespective of gender. Similarly, thyroxine (T4) levels were consistent across all groups, suggesting that H. pylori infection may not influence T4 levels. Triiodothyronine (T3) levels, however, showed a slight variation, with females trending towards higher T3 levels than males, although the role of H. pylori infection in this variation remains ambiguous.

The consistent T4 and TSH levels across groups further suggest that thyroid function remains unaffected by the presence of H. pylori infection. The slight variation in T3 levels among females could indicate a gender-specific response to H. pylori infection or an unrelated gender difference in T3 levels. However, without a significant impact of H. pylori on T3, these findings suggest that the infection may not play a direct role in thyroid hormone dysregulation.

The findings of our study suggest that H. pylori infection does not significantly disrupt metabolic parameters or thyroid function in the studied population. This could imply that the inflammatory processes associated with H. pylori infection do not extend to altering the metabolic or thyroid pathways in a clinically meaningful way. It is possible that the inflammatory response elicited by H. pylori is localized or that systemic effects are too subtle to detect in thyroid and metabolic parameters. These insights are crucial for understanding the pathophysiological mechanisms underlying chronic conditions, as they suggest that other factors may be more influential in the dysregulation of metabolic and thyroid functions.

The present study aimed to investigate a potential correlation between Helicobacter pylori (H. pylori) infection and thyroid function, as assessed by the levels of thyroid-stimulating hormone (TSH), triiodothyronine (T3), and thyroxine (T4). Contrary to some previous speculations suggesting a possible link between these two conditions, our findings indicate no significant association between H. pylori infection status and thyroid hormone levels.

These findings align with a growing consensus in the scientific community that challenges the previously proposed link between H. pylori infection and thyroid dysfunction. While earlier studies hinted at a potential association, subsequent research, including the present investigation, has consistently failed to establish a causal relationship (16,17,18,19). This lack of consistent evidence suggests that other factors, such as genetic predisposition, environmental influences, or coexisting medical conditions, may play a more significant role in the development of thyroid disorders.

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