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Thyroid and Reproductive Hormone Profiles in Relation to *Helicobacter pylori* Stool Antigen Status: A Cross-Sectional Comparative Study from Al-Bayda, Libya

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Index Terms

Helicobacter pylori; stool antigen; thyroid hormones; Libya

Abstract

Background: *Helicobacter pylori* infection has been linked to extragastric endocrine disturbances, but evidence on thyroid and reproductive hormones is inconsistent and local data are lacking. We compared serum thyroid and reproductive hormone profiles between individuals with and without *H. pylori* infection in eastern Libya.
Methods: In this cross-sectional comparative study (January–June 2025), [64] adults with gastrointestinal complaints were classified by stool antigen test into H. pylori-positive (n = [32]) and H. pylori-negative (n = [32]) groups. Serum thyroid-stimulating hormone (TSH), triiodothyronine (T_3), thyroxine (T_4), follicle-stimulating hormone (FSH), luteinizing hormone (LH) and prolactin (PRL) were measured by immunoassay. Groups were compared using independent-samples t-tests and Mann–Whitney U tests, with Cohen's d, rank-biserial correlation and 95% confidence intervals as effect-size measures. **Results:** TSH, T_3 and T_4 were numerically lower in the positive group (e.g., TSH 1.46 ± 0.86 vs 1.73 ± 0.86 µIU/mL) but did not differ significantly by either test (all $p > 0.05$). LH (8.77 ± 1.39 vs 8.78 ± 2.41 mIU/mL) and PRL (12.96 ± 5.13 vs 14.92 ± 8.43 ng/mL) were also similar. FSH was higher in the positive group (9.01 ± 2.13 vs 8.20 ± 2.37 mIU/mL); this difference was significant by the Mann–Whitney U test ($p = 0.018$; rank-biserial $r = 0.34$) but not by the t-test ($p = 0.162$; Cohen's $d = 0.35$, 95% CI −0.14 to 0.85) and did not survive correction for six comparisons.
Conclusions: *H. pylori* stool antigen status was not associated with a generalized disturbance of thyroid or reproductive hormones. The isolated FSH signal should be regarded as exploratory and requires confirmation in larger, sex- and age-stratified cohorts, ideally with pre- and post-eradication measurements.

الصورة الهرمونية للغدة الدرقية والهرمونات التناسلية في علاقتها بحالة مستضد البراز لبكتيريا Helicobacter pylori: دراسة مقطعية مقارنة من مدينة البيضاء، ليبيا

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الكلمات المفتاحية

بكتيريا الملوية البوابية, هرمونات الغدة الدرقية, ليبيا

الملخص

الخلفية: ارتبطت عدوى بكتيريا Helicobacter pylori باضطرابات الغدد الصماء خارج المعدة، إلا أن الأدلة المتعلقة بهرمونات الغدة الدرقية والهرمونات التناسلية متضاربة، كما أن البيانات المحلية شحيحة. قارنّا في هذه الدراسة مستويات هرمونات الغدة الدرقية والهرمونات التناسلية في مصل الدم بين الأفراد المصابين بعدوى H. pylori وغير المصابين في شرق ليبيا.

المنهجية: في هذه الدراسة المقطعية المقارنة (يناير–يونيو 2025)، صُنِّف 64 بالغًا يعانون من شكاوى هضمية، بحسب اختبار مستضد البراز، إلى مجموعة موجبة لبكتيريا H. pylori (العدد = 32) ومجموعة سالبة (العدد = 32). قيست في المصل مستويات الهرمون المنبّه للدرقية (TSH)، وثلاثي يودوثيرونين (T_3)، والثيروكسين (T_4)، والهرمون المنبّه للجريب (FSH)، والهرمون الملوتن (LH)، والبرولاكتين (PRL) بطريقة المقايسة المناعية. قورنت المجموعتان باستخدام اختبار (t) للعينات المستقلة واختبار مان–ويتني (U)، مع استخدام معامل كوهين (d) وارتباط الرتب الثنائي وفترات الثقة 95% كمقاييس لحجم الأثر.

النتائج: كانت قيم TSH وT_3 وT_4 أقل عدديًا في المجموعة الموجبة (على سبيل المثال: TSH ± 1.46 0.86 مقابل (μIU/mL ± 0.86 1.73، لكنها لم تختلف معنويًا بأي من الاختبارين (جميع قيم .(p > 0.05 وكانت قيم LH (8.77 ± 1.39 mIU/mL) مقابل (8.78 ± 2.41 و12.96 ± 5.13 PRL مقابل 14.92 ng/mL) ± 8.43 متقاربة كذلك. وكان FSH أعلى في المجموعة الموجبة (9.01 ± 2.13 مقابل 8.20 mIU/mL) ± 2.37؛ وكان هذا الفرق معنويًا باختبار مان–ويتني (p = 0.018؛ معامل ارتباط الرتب الثنائي (r = 0.34 ولم يكن معنويًا باختبار t (p = 0.162؛ معامل كوهين d = 0.35، فترة الثقة 95%: من 0.14− إلى 0.85)، ولم تستمر دلالته بعد التصحيح لست مقارنات.

الاستنتاج: لم ترتبط حالة مستضد البراز لبكتيريا H. pylori باضطراب عام في هرمونات الغدة الدرقية أو الهرمونات التناسلية. ويُعدّ الارتفاع المنفرد في FSH نتيجة استكشافية تتطلب تأكيدًا في دراسات أكبر مصنَّفة بحسب الجنس والعمر، ويُفضَّل أن تتضمن قياسات قبل استئصال البكتيريا وبعده.

1. INTRODUCTION

Your goal is to simulate the usual appearance of papers in a The International journal of Engineering and Helicobacter pylori is a spiral, microaerophilic, Gram-negative bacterium that colonizes the human gastric mucosa and was first cultured from patients with gastritis and peptic ulceration by Marshall and Warren [1]. Approximately 44% of the world's population, some 4.4 billion people, were estimated to be infected in 2015, with the highest prevalence in developing regions [2]. Chronic infection is the principal cause of chronic gastritis and peptic ulcer disease and a major risk factor for gastric adenocarcinoma and mucosa-associated lymphoid tissue lymphoma [3]. Beyond the stomach, H. pylori has been associated with a range of extragastric manifestations, including hematological, metabolic and endocrine disturbances [4,5]. Proposed mechanisms include persistent low-grade systemic inflammation, cross-reactivity between bacterial and host antigens, and altered gastric acid secretion that impairs the absorption of minerals and other micronutrients [4,6]. The association with iron deficiency is the best documented of these effects [7,8]; by comparison, the endocrine consequences of infection are less well characterized. The thyroid is the endocrine organ most frequently linked to H. pylori. Meta-analytic and observational data indicate an association between infection and autoimmune thyroid disease [9,10], and the coexistence of H. pylori gastritis or autoimmune gastritis with Hashimoto's thyroiditis is well recognized [11]. Hypochlorhydria and atrophic gastritis also impair levothyroxine absorption, and treating H. pylori infection can reduce levothyroxine requirements [12,13]. Impaired absorption of micronutrients such as iron may further compromise thyroid hormone synthesis [14]. Whether infection is associated with circulating thyroid hormone concentrations in people without overt thyroid disease, however, remains unclear. The hypothalamic–pituitary–gonadal axis and pituitary prolactin secretion are likewise responsive to inflammation and stress [15–17], and prolactin has recognized immunomodulatory functions [18]. Despite this biological plausibility, the relationship between H. pylori and the gonadotropins, follicle-stimulating hormone (FSH) and luteinizing hormone (LH), and prolactin (PRL) has received little systematic investigation, and the available evidence is scarce and inconsistent. The stool antigen test is an accurate, inexpensive and non-invasive method that is recommended for the diagnosis of active infection [3,19,20]. Comparative studies in dyspeptic patients have examined hematological indices, electrolytes and lipids in relation to H. pylori status [21], but, to our knowledge, thyroid and reproductive hormones have not been evaluated together in a stool antigen–defined cohort, and no such data are available from Libya. The present study therefore compared serum TSH, triiodothyronine (T_3), thyroxine (T_4), FSH, LH and PRL between H. pylori-positive and H. pylori-negative individuals in the Al-Bayda region of Libya, and quantified the magnitude and precision of any differences using effect sizes and confidence intervals. The null hypothesis was that hormone concentrations do not differ according to H. pylori stool antigen status

2. MATERIALS AND METHODOLOGY

This cross-sectional comparative study was conducted between January and June 2025 at Idea Medical Laboratory, Al-Bayda, Libya. A total of 64 adults aged 18–70 years were enrolled and classified into Helicobacter pylori stool antigen-positive and -negative groups (32 participants per group). Venous blood samples (10 mL) were collected from each participant, with 5 mL transferred into a plain tube for serum separation and 5 mL into an EDTA tube. Serum was separated by centrifugation at 1,500 × g for 10 min and stored at −80 °C until analysis. Blood samples were transported under refrigerated conditions (2–8 °C). Stool samples were collected in sterile containers, and H. pylori infection status was determined using a stool antigen assay according to the manufacturer's instructions. Serum concentrations of thyroid-stimulating hormone (TSH), triiodothyronine (T_3), thyroxine (T_4), follicle-stimulating hormone (FSH), luteinizing hormone (LH), and prolactin (PRL) were measured using the D10 analyzer, as specified in the laboratory records, following the applicable assay procedures and quality-control protocols. Statistical analyses were performed using JASP version 0.19.1.0. Data were expressed as mean ± standard deviation (SD), with minimum and maximum values. Between-group comparisons were conducted using the independent-samples t-test and Mann–Whitney U test. Effect sizes were evaluated using Cohen's d and rank-biserial correlation, as appropriate. Mean differences were calculated as the H. pylori-positive group minus the negative group, and statistical significance was set at $p < 0.05$. A Bonferroni-adjusted threshold of $p < 0.0083$ was also considered for the six hormonal comparisons.

3. RESULTS

The descriptive analysis demonstrated variable patterns in thyroid and reproductive hormone concentrations between

H. pylori-negative and H. pylori-positive participants. Serum TSH levels were higher in the negative group, with a mean of 1.732 ± 0.858 µIU/mL compared with 1.461 ± 0.859 µIU/mL in the positive group. The respective ranges were 0.525–4.790 and 0.488–3.720 µIU/mL. However, this difference was not statistically significant according to either the independent-samples t-test ($t = 1.268$, $p = 0.209$) or the Mann–Whitney U test ($p = 0.063$). The Cohen's d value of 0.316 indicated a small-to-moderate standardized difference. T3 concentrations were also higher in the H. pylori-negative group, with a mean of 1.434 ± 0.884 ng/mL compared with 1.223 ± 0.698 ng/mL in the positive group. The observed ranges were 0.450–4.830 ng/mL and 0.569–3.660 ng/mL, respectively. Statistical analysis showed no significant difference between the groups by either the independent-samples t-test ($t = 1.038$, $p = 0.303$) or the Mann–Whitney U test ($p = 0.177$). The corresponding Cohen's d was 0.259, representing a small effect size. Similarly, T4 levels were higher among H. pylori-negative participants, with a mean concentration of 5.838 ± 1.292 µg/dL compared with 5.453 ± 1.116 µg/dL in the positive group. The ranges were 4.380–9.750 µg/dL and 3.860–8.670 µg/dL, respectively. No statistically significant difference was observed using the independent-samples t-test ($t = 1.262$, $p = 0.212$) or the Mann–Whitney U test ($p = 0.222$). The Cohen's d value of 0.314 indicated a small-to-moderate standardized difference. FSH showed an opposite numerical pattern, with a higher mean concentration in the H. pylori-positive group (9.005 ± 2.130 mIU/mL) compared with the negative group (8.203 ± 2.373 mIU/mL). The respective ranges were 3.850–15.81 mIU/mL and 2.046–15.20 mIU/mL. The independent-samples t-test did not demonstrate a statistically significant difference ($t = -1.414$, $p = 0.162$). However, the Mann–Whitney U test indicated a statistically significant difference between the groups ($p = 0.018$), with an effect size of −0.344, corresponding to a small-to-moderate difference in the distribution of FSH concentrations. LH concentrations were nearly identical between the two groups. The mean LH concentration was 8.780 ± 2.407 mIU/mL in the H. pylori-negative group and 8.766 ± 1.392 mIU/mL in the positive group. The ranges were 3.840–18.50 and 5.170–12.08 mIU/mL, respectively. Neither the independent-samples t-test ($t = 0.027$, $p = 0.978$) nor the Mann–Whitney U test ($p = 0.442$) demonstrated a statistically significant difference. The Cohen's d value was 0.007, indicating a negligible standardized difference between the groups. PRL concentrations were higher in the H. pylori-negative group, with a mean of 14.920 ± 8.431 ng/mL compared with 12.960 ± 5.130 ng/mL in the positive group. The observed ranges were 5.140–42.50 ng/mL and 4.520–22.00 ng/mL, respectively. Nevertheless, the difference was not statistically significant according to the independent-samples t-test ($t = 1.078$, $p = 0.285$) or the Mann–Whitney U test ($p = 0.548$). The Cohen's d value of 0.268 indicated a small standardized difference. As expected from the diagnostic classification, HPS levels differed markedly between the two groups. The H. pylori-negative group showed a mean HPS level of 0.386 ± 0.245, with a range of 0.019–0.930, whereas the positive group showed a substantially higher mean of 10.680 ± 11.110, ranging from 1.400 to 57.15. This difference was highly statistically significant by both the independent-samples t-test ($t = -5.876$, $p < 0.001$) and the Mann–Whitney U test ($p < 0.001$). The Cohen's d value of −1.464 indicated a very large standardized difference between the two groups. The graphical presentations were consistent with the descriptive and inferential statistical findings. Figures 1–3 and Figures 4–7 illustrate the distributions of TSH, T3, and T4, showing substantial overlap between the two H. pylori status groups. Taken together, the observed differences were most evident for HPS and FSH, while TSH, T3, T4, LH, and PRL did not demonstrate statistically significant differences between the H. pylori-negative and H. pylori-positive groups.

Table 1 . "Descriptive Statistics of Thyroid, Reproductive, and Diagnostic Markers Stratified by *H. pylori* Stool Antigen Status"

Parameter	Group	Mean ± SD	Minimum	Maximum
TSH (µIU/mL)	Negative	1.732 ± 0.858	0.525	4.790
	Positive	1.461 ± 0.859	0.488	3.720
T_3 (ng/ml)	Negative	1.434 ± 0.884	0.450	4.830
	Positive	1.223 ± 0.698	0.569	3.660
T_4 (µg/dl)	Negative	5.838 ± 1.292	4.380	9.750
	Positive	5.453 ± 1.116	3.860	8.670
FSH (mIU/ml)	Negative	8.203 ± 2.373	2.046	15.20
	Positive	9.005 ± 2.130	3.850	15.81
LH (mIU/ml)	Negative	8.780 ± 2.407	3.840	18.50
	Positive	8.766 ± 1.392	5.170	12.08
PRL (ng/mL)	Negative	14.920 ± 8.431	5.140	42.50
	Positive	12.960 ± 5.130	4.520	22.00
HPS	Negative	0.386 ± 0.245	0.019	0.930
	Positive	10.680 ± 11.110	1.400	57.15

Table 2. "Comparative Analysis of Endocrine Parameters and *H. pylori* Stool Antigen Levels between Negative and Positive Groups: Parametric and Non-Parametric Test Results**".**

Parameter	Student t	p-value	Cohen's d	Mann-Whitney p	Effect Size
TSH	1.268	0.209 ns	0.316	0.063 ns	0.270
T_3	1.038	0.303 ns	0.259	0.177 ns	0.196
T_4	1.262	0.212 ns	0.314	0.222 ns	0.178
FSH	-1.414	0.162 ns	-0.352	**0.018 ***	-0.344
LH	0.027	0.978 ns	0.007	0.442 ns	0.113
PRL	1.078	0.285 ns	0.268	0.548 ns	0.088

HPS	-5.876	<0.001 ***	-1.464	<0.001 ***	-1.000

Comment: HPS showed the most highly significant difference ($p < 0.001$, Cohen's $d = -1.464$), which is expected as it is the diagnostic marker itself. FSH demonstrated a significant difference by Mann-Whitney test ($p = 0.018$, effect size = -0.344), with higher levels in the positive group. Thyroid hormones (TSH, T3, T4) showed no significant differences.

Figure 1: Distribution and density of Thyroid-Stimulating Hormone (TSH) levels (µIU/mL}) based on *Helicobacter pylori* stool antigen test results.

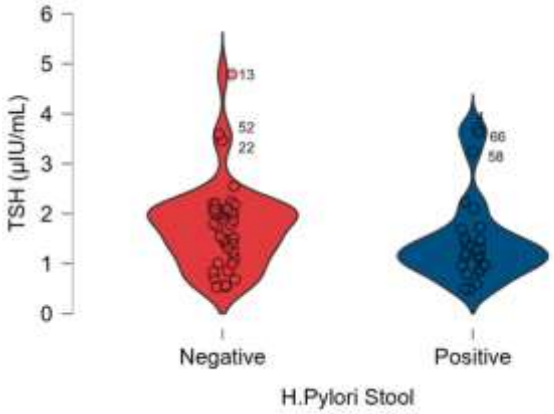


Figure 2: Distribution and density of Triiodothyronine (T_3) levels (ng/ml) based on *Helicobacter pylori* stool antigen test results.

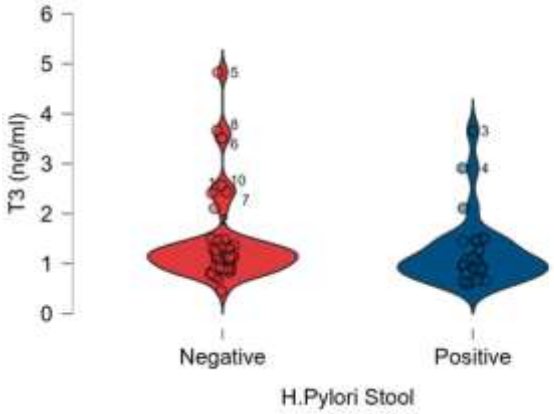


Figure 3: Distribution and density of Thyroxine (T_4) levels (µg/dl) based on *Helicobacter pylori* stool antigen test results.

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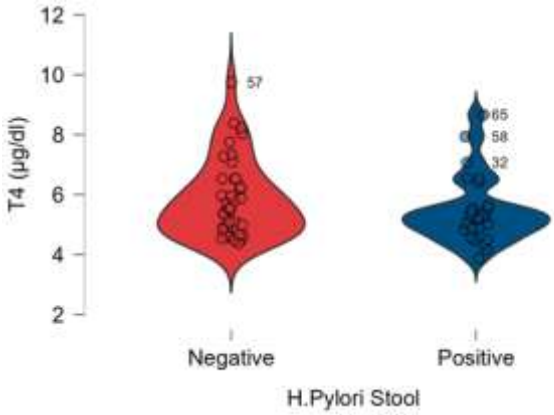


Figure 4: Distribution and density of Follicle-Stimulating Hormone (FSH) levels (mIU/ml) based on Helicobacter pylori stool antigen test results

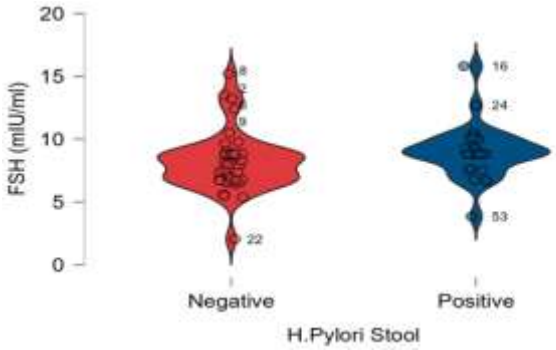


Figure 5: Distribution of Luteinizing Hormone (LH) levels (mIU/ml) based on *Helicobacter pylori* stool antigen test results.

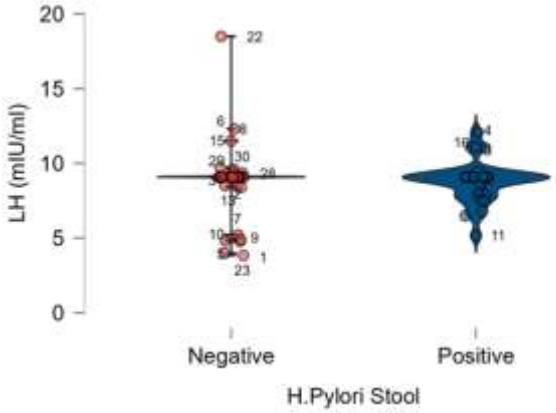


Figure 6: Distribution and density of Prolactin levels (ng/mL) based on *Helicobacter pylori* stool antigen test results.

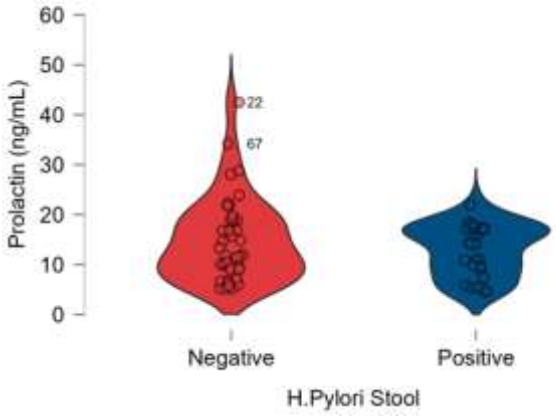
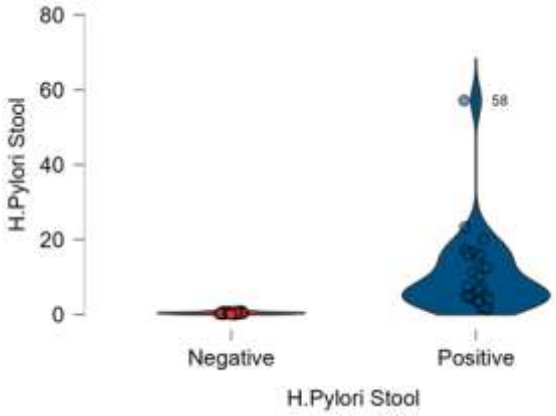


Figure 7: Distribution and density of (*H. pylori* Stool) levels based on *Helicobacter pylori* stool antigen test results.



4. Discussion

The present findings suggest that H. pylori infection was not associated with statistically significant changes in circulating TSH, T3, or T4 in this sample, despite slightly lower mean thyroid hormone values in infected participants, a pattern that is consistent with some studies reporting no clear differences in thyroid hormones by infection status or no change in FT4 [22], but differs from other reports showing lower T3 and TSH in infected older adults, lower TSH in adults with active stool-antigen-confirmed infection, or higher TSH in population-based analyses [23]. This inconsistency across studies likely reflects differences in population characteristics, infection assessment, and thyroid phenotypes, because longitudinal and subgroup data suggest that the association may emerge more clearly in selected groups such as elderly men with non-thyroidal illness syndrome, middle-aged and older women at risk of subclinical hyperthyroidism, or children with congenital hypothyroidism who showed lower T3 without changes in TSH or T4 [24]. The higher FSH observed in the positive group should be interpreted cautiously because it was supported only by the Mann–Whitney test, although limited prior evidence has also reported higher FSH in infected individuals, whereas broader reproductive evidence remains sparse compared with the thyroid literature [25]. A more consistent theme in the literature is the link between H. pylori and thyroid autoimmunity rather than simple hormone shifts, with studies showing higher TPO-Ab positivity, positive correlations with anti-TPO and anti-Tg, stronger associations for long-lasting infection, and higher prevalence in Hashimoto's thyroiditis and Graves' disease, particularly with virulent CagA-positive strains [26]. Proposed mechanisms include chronic low-grade inflammation, cytokine-mediated disruption of the hypothalamic-pituitary-thyroid axis, molecular mimicry, and immune activation [27], and interventional evidence further suggests that eradication may improve thyroid function and reduce thyroid autoantibody titers in selected hypothyroid patients [28]. As expected, the marked difference in stool antigen levels between groups mainly reflects the diagnostic classification itself rather than an independent endocrine association, and stool antigen testing is widely used to identify active infection and can distinguish active from chronic serologic positivity [29].

5. Conclusion

The present study demonstrated no statistically significant differences in thyroid hormone (TSH, T_3, and T_4), LH, or PRL concentrations between H. pylori-positive and -negative participants. Although FSH levels were higher in the infected group according to the Mann–Whitney U test, this finding did not remain significant after Bonferroni correction and should be interpreted cautiously. As expected, stool antigen levels were markedly elevated in the positive group, confirming the distinction between the diagnostic categories. Overall, these findings do not establish a clear association between H. pylori infection and alterations in thyroid or reproductive hormone profiles. Further studies involving larger populations and adjustment for relevant confounding factors are needed to clarify these potential relationships.

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