International Journal of Reproduction, Contraception, Obstetrics and Gynecology
Taheri MS et al. Int J Reprod Contracept Obstet Gynecol. 2025 Sep;14(9):2846-2852
www.ijrcog.org
pISSN 2320-1770 | eISSN 2320-1789

DOI: https://dx.doi.org/10.18203/2320-1770.ijrcog20252708

Original Research Article

Evaluation of the relationship between thyroid hormones and endometriosis: a clinical investigation

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Received: 24 May 2025
Revised: 05 July 2025
Accepted: 07 July 2025

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ABSTRACT

Background: Endometriosis is a chronic gynecological condition that affects reproductive-aged women. This study evaluated the correlation between thyroid hormone levels and endometriosis severity.
Methods: A cross-sectional study was conducted at Shohada Tajrish Hospital and Atieh Hospital from September 2024 to April 2025. Participants diagnosed with endometriosis underwent clinical, ultrasound, and thyroid hormone assessments. Statistical analyses were used to explore correlations between hormone levels and endometriosis severity.
Results: Significant associations were found between anti-TPO levels and UBESS stage 2. Pain severity was higher in moderate disease. However, TSH, T3, and Free T4 levels showed no significant correlations with disease stage or pain severity. Adhesion levels were associated with nodule presence and uterosacral ligament thickness.
Conclusions: Elevated anti-TPO levels may indicate autoimmune involvement in moderate endometriosis. Structural markers on ultrasound, such as nodule count and USL thickness, may predict disease severity.

Keywords: Adhesion, Anti-TPO, Endometriosis, Thyroid hormones, Ultrasound markers

INTRODUCTION

Endometriosis is a chronic gynecological condition characterized by the ectopic implantation and growth of endometrial-like tissue outside the uterine cavity, primarily involving the pelvic peritoneum, ovaries, and rectovaginal septum.[1-3] It affects approximately 10% of women of reproductive age globally and poses a substantial public health challenge due to its marked impact on quality of life.[4] Clinically, endometriosis presents with chronic pelvic pain, dysmenorrhea, dyspareunia, and infertility, all of which contribute to significant morbidity. The pathogenesis is multifactorial and not yet fully understood, involving genetic susceptibility, hormonal dysregulation, immune dysfunction, and environmental influences.[5,6]

Several theories have been proposed to explain the pathophysiology, with Sampson's theory of retrograde menstruation being the most widely accepted. However, this theory does not fully account for all clinical manifestations, prompting alternative explanations such as coelomic metaplasia and lymphovascular dissemination.[7,8] Endometriosis is classified into three main types based on the location and depth of lesions: superficial peritoneal lesions, ovarian endometriomas, and deep infiltrating

endometriosis (DIE).[9] These subtypes may reflect different underlying pathogenic processes, thereby necessitating individualized diagnostic and therapeutic approaches.

Thyroid hormones- particularly triiodothyronine (T3) and thyroxine (T4)- play essential roles in female reproductive health, influencing menstrual cycle regulation, follicular development, ovulation, and endometrial receptivity.[10,11] Disruptions in thyroid function, such as hypothyroidism or hyperthyroidism, can adversely affect reproductive outcomes by altering the hypothalamic-pituitary-ovarian axis, modifying levels of sex hormone-binding globulin, and affecting gonadotropin-releasing hormone pulsatility.[12]

Recent research suggests a possible link between thyroid dysfunction and endometriosis. Altered levels of T3, T4, and TSH have been documented in patients with endometriosis, along with a higher prevalence of autoimmune thyroid disorders in this population.[13,14] The underlying mechanisms may include hormonal imbalance, immune modulation, and inflammatory pathways that contribute to endometrial cell proliferation, implantation, and angiogenesis.[15] Although a causal relationship has not been definitively established, these findings underscore the potential value of thyroid function assessment in the management of endometriosis.

Evaluating thyroid hormones as potential biomarkers carries important clinical implications. Non-invasive biomarkers could enhance early diagnosis, support disease monitoring, and enable personalized treatment strategies.[16] Moreover, elucidating the interaction between thyroid function and endometriosis may reveal novel therapeutic targets within hormonal and immunological systems, potentially improving reproductive outcomes for affected women.

Given the diagnostic challenges and variable presentation of endometriosis, this study aimed to determine the potential correlation between thyroid hormone levels and the severity of endometriosis. By assessing these parameters, we hope to advance the understanding of its pathophysiology and explore biomarker-based approaches to improve clinical management.

METHODS

This cross-sectional study was conducted between (blinded for review) at the departments of radiology and obstetrics/gynecology of our university hospital. Prior to enrolment, all participants provided written informed consent, and the study protocol was approved by the university's ethics committee (blinded for review). Female patients aged 18 to 45 years were included in the study. Participants were referred to the radiology department at (blinded for review) hospital and diagnosed with endometriosis based on clinical interviews, symptoms, signs, and transvaginal sonography (TVS) findings. All diagnoses were confirmed by laparoscopy and histopathological examination to ensure definitive diagnosis.

Exclusion criteria comprised pregnancy, previous surgical intervention for endometriosis, underlying oncological diseases, and the use of hormonal medications such as gonadotropin-releasing hormone (GnRH) analogs, oral contraceptives, or progestins within the three months preceding the study. Additionally, patients with other potential causes of chronic pain (e.g., chronic lumbar pain, arthritis) were excluded to eliminate confounding variables. General demographic information- including age, weight, height, body mass index (BMI), marital status, gravidity, parity, history of abortion, menstrual phase and pattern, and infertility history- was collected through patient interviews and review of medical records.

Ultrasound examination

Ultrasound examinations were performed using a Philips Affiniti 50G ultrasound machine equipped with vaginal (10-3 MHz), curved (5-1 MHz), and linear (12-3 MHz) transducers. An experienced radiologist conducted sonographic evaluations for all patients, which included assessment of the uterus and adnexa, evaluation of the sliding sign, detailed search for DIE, and detection of ultrasound markers such as regional tenderness. Ultrasound findings were scored according to the ultrasound-based endometriosis staging system (UBESS) and categorized into three stages: mild, moderate, and severe endometriosis.[17]

Assessment of pain severity

The severity of endometriosis-related pain symptoms- including dysmenorrhea, dyspareunia, and dyschezia- was assessed using a visual analog scale (VAS). The VAS is a 10 cm linear scale where 0 indicates no pain, 1-3 represents mild pain, 4-7 moderate pain, and 8-10 severe pain, with 10 being the worst imaginable pain.[18]

Laboratory assays

During the early follicular phase of the menstrual cycle (days 2-5), non-fasting blood samples were collected from all participants to assess thyroid hormones and anti-thyroid peroxidase (anti-TPO). Serum levels of triiodothyronine (T3), thyroxine (T4), and thyroid-stimulating hormone (TSH) were measured using chemiluminescent immunoassay methods (IMMULITE 2000 XPi, Siemens Healthcare Diagnostics Inc., USA). Thyroglobulin levels were determined using an enzyme-linked immunosorbent assay (ELISA) kit (Human TG ELISA Kit, Abcam, UK). Based on recent large-scale studies in Iran, the reference ranges applied were: T3 (0.8-2.0 ng/ml), T4 (5-12 µg/dl), TSH (0.4-4.5 µIU/ml), and anti-thyroid peroxidase antibodies (anti-TPO) (<60 IU/ml).[19,20] Patients were categorized based on whether their hormone levels were within, below, or above these normal ranges.

Statistical analysis

Data were summarized as mean ± standard deviation (SD) for quantitative variables and as frequencies and percentages for categorical variables. The normality of data distribution was assessed using the Kolmogorov-Smirnov test. Comparisons between groups were performed using the independent t-test or one-way analysis of variance (ANOVA) for numerical variables and the chi-square test for categorical variables. Pearson's correlation coefficient was used to evaluate the relationship between thyroid hormone levels and the severity of endometriosis. A p value of less than 0.05 was considered statistically significant. Statistical analyses were conducted using IBM SPSS Statistics for Windows, version 21.0 (IBM Corp., Armonk, NY, USA).

RESULTS

A total of 137 patients were included. Considering the UBESS classification, 66 patients had level 2 (moderate stage disease) and 71 cases had level 3 (higher stage disease). Table 1 shows the results of the independent t-test for comparing the variables age, TSH, free T4, anti-TPO, T3, and D3 between the control and experimental groups. Based on the results, only a significant difference in Anti TPO levels was observed between the control and experimental groups ($p<0.05$).

Table 1: Comparison of clinical and hormonal variables between UBESS stage 2 and stage 3 endometriosis patients.

Variable	Stage 2 (n)	Mean	SD	Stage 3	Mean	SD	t-statistic	P value
Age	66	34.38	6.78	71	33.73	6.83	0.555	0.58
TSH	66	2.54	1.57	61	2.29	1.13	1.01	0.31
Free T4	63	8.47	12.81	50	11.01	19.83	-0.82	0.413
Anti-TPO	41	64.66	120.64	34	15.30	19.07	2.58	0.013*
T3	12	21.92	36.02	11	7.73	18.73	1.19	0.24
D3	63	30.97	14.81	71	30.05	15.97	0.34	0.732

*p<0.05 was considered statistically significant.

Table 2: Distribution of dysmenorrhea severity between UBESS stage 2 and stage 3 endometriosis patients.

Severity of dysmenorrhea	Stage 2	%	Stage 3	%	Chi-square	P value
No pain	1	1.7	33	50.0	57.58	0.0001*
Mild pain	5	8.3	6	9.1		
Moderate pain	19	31.7	24	36.4		
Severe pain	35	58.3	3	4.5		

*p<0.05 was considered statistically significant.

Table 3: Comparison of clinical variables across dysmenorrhea severity levels in UBESS stage 2 and 3 patients (sample: age).

Variable	Group	Dysmenorrhea severity	Count	Mean	SD	F-statistic	P value
Age	Stage 2	No pain	33	34.18	7.18	0.16	0.918
		Mild pain	6	36.00	6.00		
		Moderate pain	24	34.08	7.01		
		Severe pain	3	35.67	1.52		
	Stage 3	No pain	1	27.00	-	0.98	0.405
		Mild pain	5	37.20	6.76		
		Moderate pain	19	33.11	6.23		
		Severe pain	35	34.66	6.66		

Table 4: Association between adhesion severity and laboratory parameters in endometriosis patients (ANOVA).

Variables	Adhesion	Count	Mean	SD	F-statistic	P value
TSH	No	8	1.89	1.41	0.44	0.722
	Mild	11	2.48	1.41		
	Moderate	20	2.36	1.10		
	Severe	22	2.29	0.93		
Nodule	No	8	0.56	1.59	5.75	0.001*
	Mild	12	3.71	5.16		

Continued.

Variables	Adhesion	Count	Mean	SD	F-statistic	P value
	Moderate	23	7.11	7.01		
	Severe	27	13.37	12.68		

*p<0.05 was considered statistically significant.

Table 5: Association between adhesion severity and clinical variables (chi-square test).

Variables	Response	No adhesion (%)	Mild (%)	Moderate (%)	Severe (%)	Chi-square	P value
USL thickness	No	75.0	33.3	13.0	11.1	16.45	0.001*
	Yes	25.0	66.7	87.0	88.9		
Right ovarian endometriomas	Prevalent	50.0	33.3	52.2	63.0	2.97	0.426
Left ovarian endometriomas	Prevalent	87.5	50.0	60.9	63.0	2.97	0.396

*p<0.05 was considered statistically significant.

Table 2 shows the results of the Chi-square test for examining the relationship between dysmenorrhea pain levels and the control and experimental groups. Based on the results, there was a significant relationship in dysmenorrhea pain levels between the control and experimental groups (p<0.05).

Table 3 represents ANOVA for comparing the relationship between dysmenorrhea pain levels and the variables age, TSH, free T4, anti-TPO, T3, and D3 in both the control and experimental groups. Based on the results, no significant relationship was found between dysmenorrhea pain levels and any of the variables in either the control or experimental groups (p>0.05).

Table 4 shows the results of the analysis of variance (ANOVA) for comparing the relationship between adhesion levels and the variables TSH, free T4, anti-TPO, T3, D3, AMH, CA125, and nodule in the experimental group. Based on the results, a significant relationship was found only between adhesion levels and the nodule presence (p<0.05).

Table 5 shows the results of the Chi-square test for examining the relationship between adhesion pain levels and the variables uterosacral ligaments (USLs) thickness, dysmenorrhea, dyspareunia, Dyschezia, right ovarian endometriomas, and left ovarian endometriomas in the experimental group. Based on the results, a significant relationship was found only between adhesion pain levels and USL thickness (p<0.05).

DISCUSSION

This cross-sectional study evaluated the relationship between thyroid hormone levels and endometriosis severity among female adults, all diagnosed with endometriosis confirmed by laparoscopy and histopathology. Our findings reveal several noteworthy associations that contribute to understanding endometriosis pathophysiology and its potential interplay with thyroid function.

Firstly, we observed that anti-TPO antibody levels were significantly higher in patients with UBESS stage 2 compared to those with UBESS stage 3 (p=0.013). Specifically, the mean anti-TPO level in stage 2 was 64.66 ± 120.64 IU/ml, whereas in stage 3 it was 15.3±19.07 IU/ml. Anti-TPO antibodies are markers of autoimmune thyroid disorders, indicating an autoimmune response against thyroid tissue. The elevated anti-TPO levels in stage 2 patients suggest that autoimmune mechanisms may play a more prominent role in the earlier stages of endometriosis. This aligns with previous studies reporting increased prevalence of autoimmune thyroid diseases in women with endometriosis, supporting the hypothesis of a shared autoimmune etiology between the two conditions.[14,21,22] In contrast, the decline in anti-TPO levels in severe endometriosis might be attributed to immune exhaustion or regulatory mechanisms that suppress autoimmune activity as the disease progresses.[15] Nevertheless, this hypothesis is based on general immunopathological pathways that are associated with the development of endometriosis, and could not be directly addressed within the context of this study.

Contrary to expectations, patients with moderate endometriosis reported significantly higher levels of dysmenorrhea compared to those with severe disease (p<0.0001). Specifically, 58.3% of Stage 2 patients experienced severe pain, while only 4.5% of Stage 3 patients reported severe pain, with 50% indicating no pain. This counterintuitive finding suggests that pain severity in endometriosis does not always correlate positively with disease stage, as found in similar previous reports.[23,24] One possible explanation is that in advanced stages, extensive fibrosis and nerve damage might lead to decreased nerve sensitivity, resulting in lower pain perception.[25] Additionally, patients with severe disease may have adapted to chronic pain over time or may have received more effective pain management, thus reporting less pain. Therefore, such discrepancy underscores the complex and multifactorial nature of pain in endometriosis, involving not only lesion extent but also individual pain thresholds, psychological factors, and neural remodeling.[26]

Our analysis did not find significant correlations between thyroid hormone levels and either pain severity or endometriosis stage. The mean TSH levels were 2.54±1.57 µIU/ml in stage 2 and 2.29±1.13 µIU/ml in stage 3, showing no significant difference (p=0.31). Similarly, free T4 and T3 levels did not differ significantly between the two groups. These findings suggest that while anti-TPOs antibodies are elevated in moderate endometriosis, thyroid hormone production remains within normal ranges for most patients. This is consistent with studies indicating that thyroid autoimmunity can occur unnecessarily with associations to overt thyroid dysfunction in endometriosis patients, pointing toward immune dysregulation rather than hormonal imbalance.[27,28]

Furthermore, we found a significant association between adhesion levels and the presence of nodules detected via ultrasound (p=0.001). Patients with higher adhesion levels had higher mean nodule counts (mean nodules in severe adhesions: 13.37±12.68) compared to those without adhesions (mean nodules: 0.56±1.59). Our findings suggest that nodule formation may contribute to or result from the development of adhesions in endometriosis. Adhesions and nodules are manifestations of advanced disease and are indicative of DIE, which can cause structural distortion and organ dysfunction.[29,30] This relationship highlights the importance of thorough imaging assessments, including ultrasound evaluation of nodules and adhesions, in patients with suspected severe endometriosis to guide appropriate management strategies.

Similarly, adhesion pain levels were significantly associated with increased thickness of the USL (p=0.001). Among patients with severe adhesions, 88.9% had USL thickening compared to only 25% of those without adhesions. Thickening of the USL is a common finding in DIE and is associated with severe pain due to the high density of nerve fibers in this region.[31,32] This finding reinforces the role of USL involvement in the pathogenesis of pain in endometriosis and suggests that USL thickness could be a useful marker for predicting pain severity and guiding surgical planning.

Our study did not find significant relationships between dysmenorrhea pain levels and variables such as age, TSH, free T4, anti-TPO, T3, and D3 levels in either UBESS stage 2 or stage 3 groups. Similar findings have been previously reported when endometriosis patients with and without thyroid dysfunction had been compared in regards to dysmenorrhea.[33] This lack of association indicates that these factors may not be primary contributors to pain severity in endometriosis or that their effects are overshadowed by other influential factors such as lesion location, depth of infiltration, and neural involvement.

In terms of ultrasound findings, there were no significant associations between adhesion levels and the prevalence of right or left ovarian endometriomas. This suggests that while ovarian endometriomas are common in endometriosis, their presence may not directly correlate with adhesion severity.[34]

The clinical implications of our findings are multifaceted. The elevated anti-TPO levels in moderate-stage endometriosis patients highlight the potential role of autoimmune mechanisms in the early stages of the disease. This suggests that screening for thyroid autoimmunity might be beneficial in patients presenting with endometriosis, particularly those in the moderate stages. Additionally, the significant associations between adhesion levels, nodule presence, and USL thickening emphasize the importance of comprehensive imaging, including the assessment of USL thickness and the detection of nodules, in evaluating disease severity and planning treatment. It is possible thus, despite the lack of a single biomarker capable of predicting more serious cases of UBESS endometriosis, that a combination of biomarkers could work as a whole in the context of a non-invasive diagnostic approach.[35]

Several limitations of this study should be acknowledged. The sample size was relatively small, which may limit the generalizability of our findings. Additionally, certain data points were not fully available (e.g., specific patient numbers and mean ages), which could affect the robustness of the statistical analyses. Future studies with larger cohorts are necessary to validate our findings and further explore the relationship between thyroid autoimmunity and endometriosis.

The cross-sectional design of the study limits the ability to infer causality. Longitudinal studies are needed to determine whether thyroid autoimmunity predisposes individuals to endometriosis or if endometriosis contributes to the development of thyroid autoimmunity. Moreover, we did not assess other autoimmune markers or cytokine profiles that could provide deeper insights into the immunopathogenesis of endometriosis.

Future directions

An in-depth understanding of the mechanisms underlying thyroid autoimmunity and endometriosis is required. The role of cytokines, immune cell populations, and genetic factors could shed light on the shared pathways contributing to both conditions. Additionally, determining whether thyroid autoimmunity affects endometriosis symptoms and progression could provide valuable insights into integrated management.

CONCLUSION

As compared with patients with UBESS stage 3, patients with UBESS stage 2 had significantly higher levels of anti-TPO antibodies. Dysmenorrhea was more prevalent in UBESS stage 2 patients than those with UBESS stage 3. No significant correlations were observed between thyroid hormone levels and pain severity or endometriosis stage.

Additionally, significant associations were identified between adhesion levels and the presence of nodules detected via ultrasound, as well as increased thickness of the uterosacral ligaments.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: The study was approved by the Institutional Ethics Committee

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Cite this article as: Taheri MS, Boroujeni MV, Fouladi ZM, Ardebili SN, Chegeni S, Moharamzad Y, et al. Evaluation of the relationship between thyroid hormones and endometriosis: a clinical investigation. Int J Reprod Contracept Obstet Gynecol 2025;14:2846-52.