

Circulating Levels of Adipose Tissue-Derived Visfatin and Ghrelin Hormone as Predictors of Inflammation in Patients with Thyroid Dysfunctions

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ABSTRACT

Autoimmune thyroid diseases (AITDs), specifically Hashimoto's thyroiditis (HT) and Graves' disease (GD), are characterized by complex inflammatory processes. Adipocytokines such as visfatin and ghrelin are emerging as potential mediators of the thyroid-adipose axis. This study intended to assess serum visfatin and ghrelin levels in patients with AITDs to determine their value as predictors of inflammation. This case-control study included 65 female patients (33 HT; 32 GD) and 25 healthy controls. Serum T3, T4, TSH, visfatin, and ghrelin levels were measured using ELISA kits and CLIA system. Visfatin levels were significantly higher in the HT (8.12 ± 4.70 ng/mL) and GD groups (5.02 ± 1.90 ng/mL) compared to controls (2.80 ± 0.87 ng/mL) ($P < 0.001$). Conversely, ghrelin levels were significantly lower in HT (1.06 ± 0.34 ng/mL) and GD (0.83 ± 0.24 ng/mL) compared to controls (1.61 ± 0.65 ng/mL). ROC curve analysis revealed that visfatin is a strong diagnostic marker for HT (AUC = 0.831, $P = 0.002$) with 81.8% sensitivity. However, Ghrelin demonstrated non-significant diagnostic accuracy ($P > 0.05$). This study provides strong evidence for a pathophysiological relation between thyroid status and adipose-derived hormones. Visfatin acts as a significant pro-inflammatory mediator in AITDs, particularly in HT, where it serves as a reliable diagnostic predictor. Ghrelin levels vary with thyroid status, reflecting the metabolic shifts characteristic of thyroid dysfunction, but they lack sufficient diagnostic potential to serve as an independent biomarker for these conditions.

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INTRODUCTION

Autoimmune thyroid disease (AITDs) is the most common autoimmune disease and is a probable cause of thyroid dysfunction, affecting women more than men. The main factors causing hyperthyroidism and hypothyroidism are Graves' disease (GD) and Hashimoto's thyroiditis (HT), sequentially (Ovčariček *et al.*, 2024). Both diseases are influenced by genetics, autoimmune dysfunction, and environmental factors (Maarroof *et al.*, 2021; Abed *et al.*, 2023).

Adipose tissues secrete hormone-like molecules called adipocytokines that regulate metabolic activities

and influence inflammation (Clemente-Suárez *et al.*, 2023). The role of adipokines in inflammatory and immune responses is recognized; they contribute to a wide range of physiological or pathological processes (Sabir and Ahmed, 2015). Adipokines and thyroid hormones have similar physiological effects. They influence thyroid function by modulating the hypothalamic-pituitary-thyroid (HPT) axis, which regulates thyroid hormones (Abdulmuttaleb *et al.*, 2024).

One such adipokine, visfatin, is found in large amounts in visceral fat (Omer and Mahmood, 2016). It has a cytokine-like extrinsic activity known as pre-B-cell colony-enhancing factor and an enzymatic intrinsic activity called nicotinamide phosphoribosyl transferase. It is a multifaceted protein known for its enzymatic, immunological, and metabolic properties (Zhang *et al.*, 2021). Visfatin is considered a pro-inflammatory adipocytokine biomarker, has a significant role in metabolic regulation and inflammation (Cybulska *et al.*, 2025), and is a significant mediator of inflammation. Furthermore, thyroid dysfunctions can affect the clearance of these cytokines (Cinar and Gurlek, 2013). Studies have reported controversial results demonstrating that the triiodothyronine [T3] hormone may increase adipocyte

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differentiation and visfatin levels (Cybulska *et al.*, 2025; Moschen *et al.*, 2007).

Ghrelin, an endogenous peptide, plays many roles in the body's physiological processes (Akalu *et al.*, 2020). It is a brain-gut peptide primarily secreted from the gastric mucosa, stimulate appetite, increase food intake, promote adipogenesis, affect endocrine function of the pancreas and regulate energy balance (Jiao *et al.*, 2020), it can also be produced in other tissues such as the hypothalamus, hypophysis, and intestines; it has different effects on the body, including decreasing thyroid hormone and testosterone, stimulating the release of many hormones, and inhibiting insulin release (Akalu *et al.*, 2020). It also has protective mechanisms, acting as a natural antimicrobial and anti-inflammatory peptide (Mathur *et al.*, 2021). Ghrelin is closely related to thyroid function and may be produced by the thyroid gland (Akalu *et al.*, 2020; Zhang *et al.*, 2021).

This study aimed to determine the relationship of visfatin and ghrelin with AITDs (HT and GD) as predictors of inflammation.

MATERIALS AND METHODS

Study design: Observational case-control study

A total of 65 female patients with thyroid dysfunction (33 with HT and 32 with GD) were collected and diagnosed in Baghdad City by the National Center for Endocrine Disease and Diabetes in Baghdad for the period from April to July 2022. A special case sheet was used to obtain information about name, age (between 25 and 60 years), family history of disease, and clinical data on patients' other diseases and treatments. The control group consisted of 25 healthy female volunteer subjects. Controls were matched to the study patients by age. The study excluded pregnant and breastfeeding women, as well as patients with chronic or systemic disorders, based on specific criteria for patient and control selection.

Sample size

G*Power program 3.1.9.7 (Franz Faul software) used with One-way ANOVA for three groups (HT, GD, and control) at alpha error (α) 0.05, power ($1-\beta$) 0.90, a total sample size of 84 participants was found to be acceptable to detect a medium-to-large effect size (f) 0.40 (Faul *et al.*, 2009).

Sample collection and serological study

About 4 mL of venous blood was drawn from each patient and control under aseptic technique to evaluate serum levels of T3, thyroxin (T4), and thyroid-stimulating hormone (TSH); to confirm diagnosis; and to determine

visfatin and ghrelin levels. The blood samples were collected in clot activator tubes and centrifuged at 3000 rpm for 5 min, and the serum was divided into small equal parts and stored in a deep freezer at -20°C until needed.

Serum levels of thyroid hormones (T3, T4, and TSH) were determined by using an automated chemiluminescence immunoassay (CLIA) analysis system; visfatin and ghrelin levels in serum were measured by using human enzyme-linked immunosorbent assay (ELISA (kits from MyBioSource Company (MBS723926; MBS2602099).

Statistical analysis

Statistical Package for Social Sciences (SPSS, version 22; Chicago, IL, USA) was used to describe and analyze the data. The variables were expressed by mean $\pm$ standard deviation (SD), and comparisons of two groups were analyzed using Student's t-tests. A unidirectional analysis of variance (ANOVA) test was used to assess differences in variable means among the three groups (patients with GD, HT, and a control group). The least significant difference (LSD) post-hoc test was used for specific group-to-group comparisons. Receiver operating characteristic (ROC) curve analysis was used to determine the predictive value of visfatin and ghrelin as inflammatory markers and diagnostic accuracy. The statistical significance was determined at a significance level of ($P<0.05$), and highly significant at ($P<0.001$).

RESULTS

This study included the analysis of three groups. Patients with thyroid dysfunction were divided into two groups: HT group of 33 patients and GD group of 32 patients. The healthy control group comprised 25 subjects. Their ages ranged between 25 and 60 years.

The results of the current study showed no significant differences in age among the three groups (HT, GD, and healthy control). The study groups consisted of 33 patients with HT with an age range between 24 and 62 years (41.6 ± 10.2 years), 32 patients with GD with an age range between 21 and 60 years (40.1 ± 10.7 years), and 25 subjects as healthy controls with age range between 20 and 55 years (41.5 ± 9.3 years), as shown in Table I.

The ANOVA test showed a significant decrease in mean serum T3 levels in HT patients (0.58 ± 0.15 ng/mL) compared with the GD group (3.37 ± 1.39 ng/mL) and the healthy control group (1.67 ± 0.53 ng/mL) ($F=81.89$, $P<0.000$). The level of T4 in the HT group (60.52 ± 22.87 ng/mL) decreased significantly ($F=62.305$, $P<0.000$) compared with those in the GD (127.10 ± 30.07 ng/mL) and healthy control groups (86.10 ± 16.01 ng/mL). The results showed that the serum level of TSH increased significantly

Table I. Age distribution of study groups.

Age group (years)	Hashimoto disease group (HT) N (%)	Graves' disease group (GD) N (%)	Healthy control group N (%)	P value (t- test)
<40	16(48.5)	18(56.2)	9(36.0)	0.218 ^{NS}
40-45	12(36.4)	8(25.0)	12(48.0)	
50+	5(15.1)	6(18.8)	4(16.0)	
Total	33(100.0)	32(100.0)	25(100.0)	
Range	24 - 62	21-60	20 - 55	
Mean $\pm$ SD	41.6 $\pm$ 10.2	40.1 $\pm$ 10.7	41.5 $\pm$ 9.3	

Table II. Serum biomarker levels in the study groups.

Measured variable	Hashimoto's group (HG) (n=33)	Graves' Disease (GD) group (n=32)	Healthy control group (n=25)	ANOVA (F-value)	Significance (P-value)
T3 (ng/mL)	0.58 $\pm$ 0.15	3.37 $\pm$ 1.39	1.67 $\pm$ 0.53	81.890	0.000**
T4 (ng/mL)	60.52 $\pm$ 22.87	127.10 $\pm$ 30.07	86.10 $\pm$ 16.01	62.305	0.000**
TSH (μ IU/mL)	9.79 $\pm$ 3.03	0.97 $\pm$ 1.30	2.92 $\pm$ 1.23	156.399	0.000**
Visfatin (ng/mL)	8.12 $\pm$ 4.70	5.02 $\pm$ 1.90	2.80 $\pm$ 0.87	21.624	0.000**
Ghrelin (ng/mL)	1.06 $\pm$ 0.34	0.83 $\pm$ 0.24	1.61 $\pm$ 0.65	24.481	0.000**

The data expressed in mean $\pm$ standard deviation (Mean $\pm$ S.D); significant p-value $< 0.001^{**}$, highly significant (HS).

(F= 156.399, P<0.000) in the HT group (9.79 $\pm$ 3.03 μ IU/mL) compared with the GD (0.97 $\pm$ 1.30 μ IU/mL) and healthy control groups (2.92 $\pm$ 1.23 μ IU/mL). However, visfatin level in HT (8.12 $\pm$ 4.70 ng/mL) increased significantly (F= 21.62, P<0.000) compared with the level in GD and the control group (5.02 $\pm$ 1.90 ng/mL) and (2.80 $\pm$ 0.87 ng/mL), respectively. Ghrelin level in HT (1.06 $\pm$ 0.34 ng/mL) increased significantly (F= 24.48, P<0.000) than the level in GD (0.83 $\pm$ 0.24 ng/mL) and decreased significantly from the control level (1.61 $\pm$ 0.65 ng/mL).

By contrast, in patients with GD group, the mean T3 level (3.37 $\pm$ 1.39 ng/mL) increased significantly (F= 81.89, P<0.000) compared with HT (0.58 $\pm$ 0.15 ng/mL) and control groups (1.67 $\pm$ 0.53 ng/mL). In addition, the mean T4 level in GD patients (127.10 $\pm$ 30.07 ng/mL) increased significantly (F=62.305, P<0.000) compared with that in the HT (60.52 $\pm$ 22.87 ng/mL) and control groups (86.10 $\pm$ 16.01 ng/mL). Conversely, TSH level decreased significantly (F= 156.399, P<0.000) in the GD group (0.97 $\pm$ 1.30 μ IU/mL) in comparison with that in the HT (9.79 $\pm$ 3.03 μ IU/mL) and control groups (2.92 $\pm$ 1.23 μ IU/mL). While visfatin level in GD (5.02 $\pm$ 1.90 ng/mL) decreased significantly (F= 21.62, P<0.000) compared with the level in HT (8.12 $\pm$ 4.70 ng/mL) and increased significantly than control level (2.80 $\pm$ 0.87 ng/mL). Ghrelin level in GD (0.83 $\pm$ 0.24 ng/mL) decreased significantly (F= 24.48, P<0.000) in comparison with the level in HT and control groups (1.06 $\pm$ 0.34 ng/mL) and

(1.61 $\pm$ 0.65 ng/mL), respectively, as in [Table II](#).

The least significant difference (LSD) post-hoc test follows ANOVA to recognize exactly which study groups (HT, GD, and Control) differ from one another concerning Ghrelin and Visfatin levels, in [Table III](#).

Visfatin levels show highly significant elevations in HT vs GD groups (Mean difference = 3.097, P=0.000), and in HT and GD compared to controls (Mean difference = 5.318, P=0.000), (Mean difference = 2.221, P=0.009), respectively. While GD shows a significant decrease from HT (Mean difference = -3.097, P=0.000).

However, ghrelin analysis showed in both disease groups (HT and GD) a significant decrease (P=0.000) in ghrelin levels relative to controls (Mean difference = -0.548, -0.780), correspondingly, and there is a lower level in the GD group than in the HT group (Mean difference = 0.232, P=0.030). While there is a significant difference (P=0.030) between HT and GD, ghrelin levels are slightly higher in HT patients than in GD (Mean Difference = 0.232).

In [Table IV](#) and [Figure 1](#), the area under the curve (AUC) quantifies a biomarker's ability to discriminate between patients and controls, indicating the diagnostic accuracy of visfatin and ghrelin in recognizing HT and GD based on the ROC data provided. Visfatin in HT (AUC = 0.831, P= 0.002), with a cut-off of 3.695 ng/mL, indicates that people below this level are considered patients, and (81.8% sensitivity, 84.0% specificity), ensures a low rate of

Table III. Multiple Comparison (LSD) among all pairs of serum Ghrelin and Visfatin levels in patient and control groups.

Dependent variable	Key pairwise comparison (LSD)		Mean difference $\pm$ SEM (I-J)	95% Confidence interval	
	(I) VAR00001	(J) VAR00001		Lower bound	Upper bound
Visfatin	HT	GD	3.097 $\pm$ 0.769**	1.568	4.626
		Control	5.318 $\pm$ 0.822**	3.684	6.952
	GD	HT	-3.097 $\pm$ 0.769**	-4.626	-1.568
		Control	2.221 $\pm$ 0.822**	0.576	3.866
Ghrelin	HT	GD	0.232 $\pm$ 0.105*	0.023	0.441
		Control	-0.548 $\pm$ 0.112**	-0.771	-0.324
	GD'	HT	-0.232 $\pm$ 0.105	-0.441	-0.023
		Control	-0.780 $\pm$ 0.113	-1.005	-0.555

GD, Graves' disease; HT, Hashimoto's thyroiditis.

Table IV. Serum ghrelin and visfatin levels in patient groups with area under the curve in the ROC curve.

Target condition	Test marker	Area Under curve (AUC)	Std. Error- a	Asymptotic P-value -b	Calculated cut-off value (ng/mL)	Sensitivity	Specificity	95% confidence interval	
								Lower bound	Upper bound
HT	Visfatin	0.831	0.071	0.002**	3.695	81.8%	84.0%	0.691	0.971
	Ghrelin	0.669	0.096	0.105	2.685	65.6%	72.0%	0.480	0.858
GD	Visfatin	0.708	0.109	0.05*	0.875	60.6%	64.0%	0.494	0.921
	Ghrelin	0.636	0.108	0.211	0.905	59.4%	60.0%	0.424	0.849

significant p-value < 0.05* and < 0.001**; HS, highly significant. Not: Cut-off values were determined using the Youden Index ($J = \text{Sensitivity} + \text{Specificity} - 1$) as described by Hajian-Tilaki, 2018.

false positives in healthy subjects. This highly significant value indicates that visfatin has the best diagnostic accuracy for the disease. This imagines its high sensitivity and specificity for HT. In GD, a significant value (AUC = 0.708, $P = 0.05$) with a cut-off of 0.875 ng/mL indicates that people above this level are considered patients, and (60.6% sensitivity, 64.0% specificity), indicating it's less effective in diagnosing GD. However, ghrelin shows non-significant values in HT and GD ($P > 0.05$). The lower AUC values and lack of statistical significance indicate that ghrelin is a less consistent marker for ADTI.

DISCUSSION

Thyroid autoimmunity is characterized by the development of serum autoantibodies directed against the thyroid autoantigens thyroglobulin (Tg), thyrotropin receptor (TRAb), and thyroid peroxidase (TPO) (Al-Hindawi and Al-Ghurabi, 2019; Vargas-Uricoechea *et al.*, 2023). The results of this study confirmed the diagnosis of autoimmune-induced hypothyroidism (HT) or hyperthyroidism (GD). However, the clinical characteristics of the two patient groups observed in these

results were consistent with other findings, indicating that thyroid autoimmunity can lead to a variety of thyroid diseases, including alterations in thyroid hormone production (Tywanek *et al.*, 2024).

Elevated blood levels of autoantibodies in HT are predominantly attributed to a cellular autoimmune response accompanied by significant inflammatory infiltration, resulting in damage to the thyroid gland. GD may be related to a humoral autoimmune response involving the production of autoantibodies against TRAb, which increase the growth and activity of thyroid follicular cells (TFCs). Injury occurs when autoantibodies and sensitized T lymphocytes react with thyroid cells, resulting in an inflammatory reaction (Yilmaz *et al.*, 2025).

The current visfatin results are consistent with those of Caixàs *et al.* (2009) and Han *et al.* (2012), who reported higher visfatin levels in hyperthyroidism and hypothyroidism than in controls, and that these levels further increased after treatment. According to Han *et al.* (2021), thyroid dysfunction is linked to increased visfatin levels, which may be due to elevated visfatin mRNA expression in visceral fat, and T3 causes a nonlinear regulation of visfatin mRNA expression. While Ozkaya *et*

al. (2009) reported increased visfatin levels in patients with hyperthyroidism and decreased levels in hypothyroidism, suggesting that the effects of hyperthyroidism and hypothyroidism on metabolic markers may be partially mediated by visfatin.

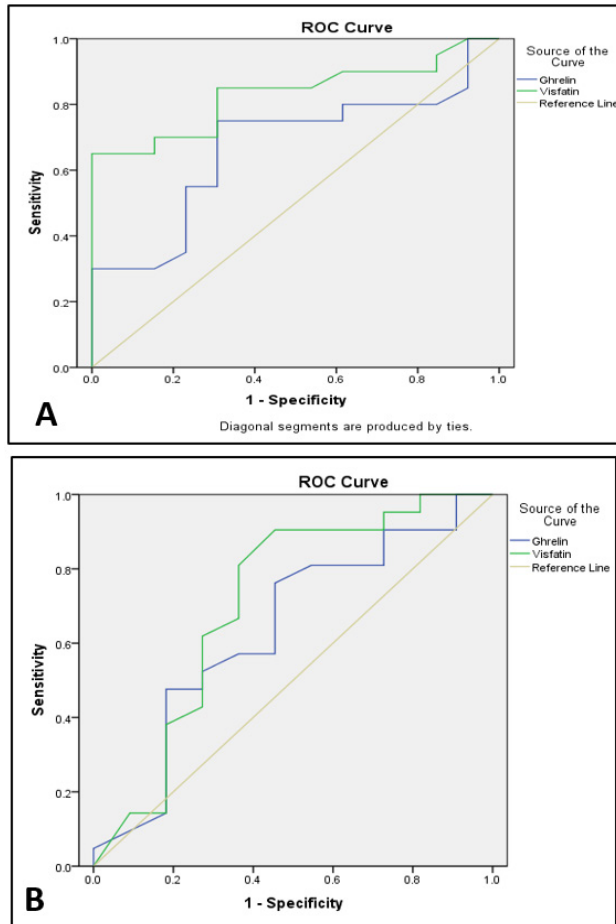


Fig. 1. ROC Curve Hashimoto vs control (A) of Graves' disease vs control (B).

However, there is agreement with the results of Sawicka-Gutaj *et al.* (2021), who demonstrated that mean serum visfatin levels were significantly elevated in patients with autoimmune hypothyroidism compared with healthy controls. They reported that in hypothyroid patients, visfatin concentration is related to both autoimmunity and thyroid hormone levels. Uçan *et al.* (2018) reported similar visfatin concentrations in the autoimmune hypothyroid and control groups; they concluded that no correlation exists between visfatin and thyroid autoimmunity.

Visfatin exhibits various immunological and inflammatory actions, including the induction of inflammatory cytokines and the regulation of macrophage

and lymphocyte proliferation; these actions are believed to underlie the association between visfatin and thyroid diseases (Luk *et al.*, 2008). Thus, visfatin may be influenced by thyroid dysfunction, either directly by release from adipose tissue or indirectly by proinflammatory cytokines, implicating its involvement in the development of thyroid dysfunction (Caixas *et al.*, 2009; Sawicka-Gutaj *et al.*, 2016). Another study showed that visfatin, in individuals with hypothyroidism depends on thyroid hormone levels and coexisting autoimmunity; conversely, in hyperthyroidism, visfatin is correlated with fat content, with minor effects of thyroid autoimmunity. Thus, thyroid hormones and thyroid autoimmunity are the two factors to consider when evaluating visfatin levels in patients with thyroid dysfunction (Mele *et al.*, 2019; Sawicka-Gutaj *et al.*, 2021).

Regarding serum ghrelin, the present results confirmed that most studies reported lower ghrelin levels in hyperthyroidism than in controls, but were inconsistent with stated higher ghrelin levels in hypothyroidism than in controls (Ruchala *et al.*, 2014; Mele *et al.*, 2019; Kaushik and Sharma, 2021). Xin *et al.* (2025) observed a significant decrease in ghrelin levels in hyperthyroid patients compared with controls, followed by a significant increase after treatment. While the levels were increased non-significantly in hypothyroid patients compared with controls.

Riis *et al.* (2003) reported significantly lower serum ghrelin in patients with GD than in controls. Biyikli *et al.* (2014) found that ghrelin levels were lower in HT patients than in controls, and were similar in patients with low and high anti-TPO concentrations. They found a correlation between low ghrelin levels and HT disease. The findings of this analysis indicated that HT was a significant predictor of ghrelin levels. The combination of atrophic gastritis and AITD has been proposed as a possible explanation for decreased ghrelin release from gastric cells in those patients (Cecchi *et al.*, 2007). By contrast, several studies disagreed with the current findings, Gimenez-Palop *et al.* (2005) reported normal serum ghrelin levels in patients with hypothyroidism. Kim *et al.* (2015) observed no difference in mean ghrelin levels among the three groups: euthyroid, hyperthyroid, and hypothyroid.

There are various theories regarding the fluctuations in ghrelin levels in subjects with thyroid dysfunction. Hyperinsulinemia with hyperthyroidism may play a role (Xin *et al.*, 2025). Ghrelin impacts the hypothalamus–pituitary–thyroid (HPT) axis in humans. Thus, the increased fT4 levels may be due to direct action of ghrelin on the thyroid gland, where ghrelin receptors are present (Kluge *et al.*, 2010). Another possible explanation is that hyperthyroidism may increase the metabolic clearance

rate, as with other hormones (Tanda *et al.*, 2009). In a previous study, Kluge *et al.* (2010) demonstrated that exogenous ghrelin in humans leads to an elevation of fT4 levels and a reduction in TSH levels. These data provide further evidence for the hypothesis that ghrelin may serve a compensatory function in thyroid dysfunction (Kluge *et al.*, 2010).

Regarding the ROC curve results for serum ghrelin and visfatin levels in patient groups, evidence supports that TSH receptors (TSHR) are not restricted to the thyroid gland; they are present on the surface of preadipocytes, and elevated TSH may activate TSHR in adipocytes (Zhou *et al.*, 2024). These receptors are activated in HT by high levels of TSH itself and in GD by TSHR antibodies (TRAb) that mimic TSH and bind to the adipocyte TSHR, thereby triggering the release of visfatin as a pro-inflammatory mediator (Walczak and Sieminska, 2021). In Graves' Ophthalmopathy, the orbital fat behind the eyes contains high levels of TSHR, and TRAb bind to these receptors, causing the fat cells to proliferate and trigger inflammation, leading to the bulging eyes characteristic of the disease (Lee and Kahaly, 2025).

Thus, the current results reported the higher diagnostic accuracy of visfatin in HT than in GD, suggesting that visfatin secretion is more sensitive to increased TSH than to TRAb stimulation. This is consistent with the results of Walczak and Sieminska (2021), who identified a direct TSH-visfatin regulatory axis. Elevated TSH may contribute to weight gain and changes in fat distribution in hypothyroid patients by promoting the maturation of preadipocytes into white adipose tissue. However, the non-significant AUC for ghrelin is consistent with Xin *et al.* (2025), who reported that ghrelin loses the specificity required to act as an independent biomarker for autoimmune thyroid pathology (Xin *et al.*, 2025).

The findings of Ahmed and Hussain (2025) showed that adipokine (visfatin) levels change in response to the thyroid status and autoimmune activity, which observed the role of visfatin biomarker for diagnosing or monitoring the evolution of thyroid-related metabolic dysfunction. While visfatin is a direct product of the adipose-thyroid axis, ghrelin is primarily regulated by gastrointestinal signals and energy balance and is influenced by appetite, which helps explain why they often show lower diagnostic accuracy in thyroid-specific ROC analyses (Walczak and Sieminska, 2021).

Therefore, our opinion is that visfatin may play a role in the pathogenesis of AITD and can be considered a new inflammatory biomarker for diagnosing AITD, especially HT, as shown by the ROC curve.

It is worth noting that the present study provides strong evidence that visfatin may play a vital role in

the pathogenesis of AITD, specifically HT in Iraq and can be used to investigate autoimmune thyroid diseases by highlighting visfatin as a sensitive inflammatory biomarker. This needs to be confirmed by measuring immunological responses in those patients, given the role of these adipokines as important mediators of inflammation, in addition to their relationships with the HPT axis. While ghrelin does not have the potential to serve as an independent biomarker for autoimmune thyroid diseases.

CONCLUSION

This study provides strong evidence for a pathophysiological relation between thyroid status and adipose-derived hormones. Visfatin is a validated inflammatory biomarker for diagnosing AITDs, with major diagnostic accuracy for Hashimoto's thyroiditis compared with Graves' disease. Furthermore, changes in ghrelin levels in relation to thyroid status reflect the metabolic shifts characteristic of thyroid dysfunction, but they lack sufficient diagnostic potential to serve as an independent biomarker for these conditions. Collectively, these findings highlight the thyroid-adipose axis as a critical pathway in the pathogenesis of thyroid autoimmunity and suggest that serum visfatin may be a valuable tool for monitoring inflammatory activity in these patients.

Limitation of the study

Additional subjects are needed to determine the effects of thyroid diseases on adipocytokines and of adipocytokines on autoimmune processes. In addition, the thyroid autoantibody titers must be measured to clarify the results.

DECLARATIONS

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IRB approval

The Institutional Review Board (IRB)/Research Ethics Committee of the College of Dentistry at the University of Baghdad approved the research before it began (Approval No. 363 on July 6, 2021).

Ethical statement

The Ethics Committee of the College of Dentistry at

the University of Baghdad approved the research under reference number 363 on July 6, 2021. All participants received complete information about the current study's objectives and procedures.

Generative AI and AI assisted technology statement

The authors declare that no generative AI and AI assisted technology was used in the creation of this manuscript.

Statement of conflict of interest

The authors have declared no conflict of interest.

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