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Assessment of Adipsin and Lipid Profile in Iraqi Women with Thyroid Dysfunction

Maryam A. Hassoun, Eiman AA. Abass*

Department of Chemistry, College of Education for Pure Science Ibn Al-Haitham, University of Baghdad, Baghdad, Iraq

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***Corresponding author:**
Eiman AA. Abass,
Department of Chemistry, College of Education for Pure Science Ibn Al-Haitham, University of Baghdad, Baghdad, Iraq

Email:
eiman.aa.a@ihcoedu.uobaghdad.edu.iq

ABSTRACT

Background & Objective: Thyroid dysfunction (TD) and diabetes mellitus (DM) are among the most common endocrine disorders, both strongly linked to metabolic dysregulation. Adipsin, an adipokine, has been implicated in obesity-related cardiometabolic diseases and may serve as a potential biomarker. This study aimed to evaluate serum adipsin and lipid profile levels in Iraqi women with thyroid dysfunction, to investigate their diagnostic value, and to assess whether these patients are at increased risk of cardiovascular disease.

Materials & Methods: This case–control study included 130 women aged 25–55 years, of whom 100 had thyroid dysfunction and 30 served as healthy controls. Patients were divided into four groups: G1, hypothyroidism (n=25); G2, hypothyroidism with DM (n=25); G3, hyperthyroidism (n=25); and G4, hyperthyroidism with DM (n=25). Serum adipsin and lipid parameters, including triglycerides (TG), total cholesterol (Chol), low-density lipoprotein cholesterol (LDL-ch), and high-density lipoprotein cholesterol (HDL-ch), were measured and compared across groups.

Results: There were an important elevation in Triglycerides (TG), total Cholesterol (Chol), and Low-Density Lipoprotein Cholesterol (LDL-ch) levels in G2 and G3, also TG was significant increase in G3 and G4, in addition a significant elevation in LDL-ch in G4. While there were an important decrease in High-Density Lipoprotein Cholesterol (HDL-ch) levels in all patient groups. Adipsin levels were significantly higher in all patient groups than in the control group, according to the study.

Conclusion: The current study suggests that Adipsin may have a potential role as a diagnostic and follow-up marker of DM in patients with thyroid disease, but further studies are needed to confirm this. Compared to hyperthyroidism, patients with hypothyroidism may be at risk for cardiovascular diseases. Additionally, adipsin may be considered a diagnostic marker for thyroid dysfunction.

Keywords: Adipsin, Hyperthyroidism, Lipid Profile, Cardiovascular Diseases

1. Introduction

Thyroid dysfunction ranks among the most common endocrine disorders. The thyroid gland produces thyroxine (T4) and triiodothyronine (T3) under the stimulation of thyroid-stimulating hormone (TSH) secreted by the pituitary gland (1, 2). Thyroid hormones affect almost all body functions, including metabolic rate, as well as cardiac, gastrointestinal, musculoskeletal, reproductive, and neurologic functions. Thyroid disorders cause variable signs and symptoms that require thorough evaluation for correct diagnoses and effective management (3).

Thyroid disorders are common in women, and understanding the signs and symptoms of hypo- and hyperthyroidism is essential. The usual age of diagnosis of hyperthyroidism from Graves' disease is between 30 and 60 years, which is also the time many women may experience the onset of perimenopausal and menopausal symptoms (4).

Thyroid dysfunction affects human health in various ways, including contributing to dyslipidemia and cardiovascular diseases. Diabetes mellitus (DM) and thyroid dysfunction are closely related endocrine illnesses because they can make each other worse. Patients with

diabetes are far more likely than individuals without the disease to have thyroid problems (5).

Thyroid disorders and diabetes mellitus (DM) are among the most prevalent endocrine diseases, and their pathophysiological mechanisms frequently overlap. A strong bidirectional relationship exists between diabetes mellitus and thyroid dysfunction. Numerous studies have demonstrated that individuals with diabetes are more likely to develop thyroid abnormalities, and vice versa. To elucidate that the association between type 1 and type 2 diabetes and thyroid dysfunction is not merely coincidental, this study examines several underlying mechanisms (6).

Thyroid hormones play a central role in regulating numerous metabolic processes, including lipid synthesis, mobilization, and degradation. They act as major regulators of lipid metabolism by stimulating hepatic de novo fatty acid synthesis, as well as lipid mobilization and breakdown. In addition to their effects on glucose and lipid metabolism, thyroid hormones and adipokines influence physiological processes such as energy expenditure. Thyroid dysfunction is increasingly prevalent and is often associated with adverse alterations in lipid profiles. Among individuals with thyroid disease, dyslipidemia represents a significant risk factor for cardiovascular disease (7, 8).

Adipsin, also known as complement factor D, is an adipokine involved in the alternative complement pathway and plays a key role in lipid metabolism and insulin secretion. Alterations in adipsin levels have been linked to various metabolic disorders, prompting investigation into its relationship with thyroid hormones to better understand the metabolic consequences of thyroid dysfunction (9, 10). Changes in adipsin concentrations have been associated with a range of metabolic and endocrine abnormalities, suggesting its potential utility as a biomarker for hormonal imbalance. Given that thyroid hormones exert strong regulatory effects on adipose tissue metabolism, adipsin may serve as a novel link between thyroid dysfunction and metabolic disturbances (11).

Adipsin is secreted predominantly by white adipose tissue through both paracrine and endocrine mechanisms. It influences systemic energy homeostasis and contributes to maintaining β-cell function and triglyceride synthesis (12). Emerging evidence indicates that elevated circulating adipsin levels are associated with a significantly reduced risk of developing diabetes among middle-aged adults, independent of body mass index (BMI). Collectively, these findings highlight the potential of adipsin/C3a and DUSP26-targeted therapeutic strategies as innovative approaches to preserve β-cell health and to manage or prevent type 2 diabetes (8, 13).

The high prevalence of thyroid disorders and their related complications, including diabetes and cardiovascular diseases, underscores the importance of investigating these interconnected endocrine conditions, which forms the rationale for the present study.

2. Materials and Methods

2.1 Study Design

This analytical case-control study was conducted at the Specialized Center for Endocrinology and Diabetes, Baghdad, from December 2024 to April 2025. A total of 130 females aged 25–55 years were enrolled. Among them, 100 were patients with thyroid dysfunction, and 30 were healthy women who served as the control group (C). The control participants were free from chronic illnesses and were not on any medication. They were matched with the patients in terms of age range.

All patients were classified into four groups: 25 women with hypothyroidism as G1, 25 women with hypothyroidism and DM as G2, 25 women with hyperthyroidism as G3, and 25 women with hyperthyroidism and DM as G4.

2.2 Sample Collection

Venous blood samples were obtained from both patients and healthy controls. Serum was separated and used for laboratory analyses, including measurements of thyroid-stimulating hormone (TSH), triiodothyronine (T3), thyroxine (T4), adipsin, triglycerides (TG), total cholesterol (Chol), high-density lipoprotein cholesterol (HDL-C), and low-density lipoprotein cholesterol (LDL-C). Thyroid hormones (T3 and T4) were quantified using a competitive ELISA method, while TSH levels were determined using a sandwich ELISA technique. Adipsin concentrations were measured using a sandwich ELISA-based commercial kit. Triglycerides, total cholesterol, and HDL-C were measured using enzymatic colorimetric methods. LDL-C was calculated using the Friedewald equation: LDL-C = Chol - HDL-C - (TG/5). This calculation is invalid when TG levels are ≥ 400 mg/dL (14). Anthropometric data, including age, weight, and height, were recorded. Body mass index (BMI) was calculated as weight (kg) divided by height squared (m^2) (15).

2.3 Exclusion Criteria

All patients with other autoimmune or hematological diseases, pregnant women, and those with a history of thyroid surgery or thyroid cancer were excluded from the study.

2.4 Statistical Analysis

Data were expressed as mean ± standard deviation (SD). Statistical analyses were performed using the Student's t-test and Pearson's correlation coefficient to evaluate differences between patients and controls. A p-value ≤ 0.05 was considered statistically significant, while $p > 0.05$ was considered non-significant. Receiver operating characteristic (ROC) curve analysis was conducted to differentiate between active cases and controls and to assess the sensitivity and specificity of adipsin as a potential diagnostic biomarker for thyroid disorders.

3. Result

Table 1 summarizes the clinical and biochemical parameters for each study group, including TSH, T3, T4, BMI, and adipsin levels. These parameters were assessed in patients with hypothyroidism with and without diabetes mellitus (G1 and G2), hyperthyroidism with and without diabetes mellitus (G3 and G4), and in the control group (C). Comparing G1 and G2 with the control group, TSH levels were significantly elevated ($P \leq 0.05$). In contrast, TSH levels in G3 and G4 were significantly lower than controls, with a statistically significant difference also observed between G3 and G4. T3 levels were significantly decreased in G1 and G2 compared to controls, whereas G3 and G4 exhibited a marked increase in T3 levels. Additionally, a notable difference was observed between G3 and G4. Similarly, T4 levels were significantly higher in G3 and G4 compared to the control group. Fasting blood glucose (FBG) and HbA1c values were significantly elevated in G2 and G4 compared to G1, G3, and controls. BMI was significantly increased in G1 and G2, whereas G3 showed a significant decrease compared to controls; no significant difference in BMI was observed between G4 and the control group. Adipsin levels were significantly higher in all patient groups (G1, G2, G3, and G4) compared to controls ($P \leq 0.05$). Furthermore, adipsin levels differed significantly between G1 and G2, as well as between G3 and G4.

Triglycerides (TG), total cholesterol (Chol), high-density lipoprotein cholesterol (HDL-C), and low-density lipoprotein cholesterol (LDL-C) were measured in the control group (C) and patient groups (G1–G4). The results are summarized in Table 2. TG levels were significantly elevated ($P \leq 0.05$) in all patient groups (G1–G4) compared to controls. TG levels in G3 and G4 were lower than those in G1 and G2, yet still higher than in the control group. The highest mean TG level was observed in G1. Cholesterol levels were significantly higher ($P \leq 0.05$) in G1 and G2 compared to controls. In contrast, Chol levels in G3 and G4 were comparable to controls, with no significant differences observed between these groups. HDL-C levels were significantly reduced ($P \leq 0.05$) in G1 and G2 compared to controls, indicating early lipid metabolism disturbances in hypothyroidism. LDL-C levels were significantly elevated ($P \leq 0.05$) in G1 and G2 relative to controls. Additionally, a notable difference in LDL-C was observed between G3 and G4, with G4 showing slightly higher levels.

Overall, these findings suggest that thyroid dysfunction, particularly hypothyroidism (G1 and G2), is associated with pronounced dyslipidemia, characterized by elevated TG, Chol, and LDL-C levels and decreased HDL-C levels.

Pearson correlation analysis between adipsin and biochemical parameters revealed significant associations in the control group. Adipsin levels were inversely correlated with TSH, T4, BMI, TG, and HDL-C, suggesting that alterations in lipid metabolism and reductions in certain thyroid hormones may be linked to elevated adipsin levels. Total cholesterol exhibited a significant positive correlation, while T3 and LDL-C showed no significant associations. In patient groups, adipsin demonstrated variable correlations with metabolic and thyroid parameters. Positive correlations with BMI and significant correlations with TG were observed at specific stages, while TSH showed a positive association in one stage and T4 generally exhibited a negative correlation across most stages. No significant correlation with LDL-C was detected. These findings indicate that adipsin may contribute to the metabolic changes associated with thyroid dysfunction, potentially modulating the interplay between thyroid hormone levels and lipid metabolism, with the strength and direction of correlations dependent on the severity of thyroid disease (Table 3).

The Adipsin marker's ROC analysis results for various patient groups are displayed in Table 4. Adipsin is a great diagnostic marker for thyroid dysfunction, according to the results. With an area under the curve (AUC) of 1.000, the test demonstrated flawless diagnostic accuracy for G1 and G2 patients as in Figures 1 and 2, respectively. It also demonstrated 100% sensitivity and 100% specificity. In the G3, the AUC was 0.917, while the sensitivity was 85% and the specificity was 90% as in Figure 3, indicating that Adipsin is still a useful diagnostic marker, but with somewhat less accuracy than in hypothyroid patients. For group G4, the AUC of 1.000, the test once more showed 100% sensitivity and 100% specificity for hyperthyroid patients with diabetes mellitus (Figure 4), comparable to the hypothyroid groups.

Table 1. Distribution of TSH, T3, T4, BMI and Adipsin levels in controls and patients with thyroid dysfunction.

Parameters	Control No.30	G1 No.25	G2 No.25	G3 No.25	G4 No.25
TSH (mu\L)	2.19 ± 0.89	5.95 ± 1.64*	6.08 ±2.73*	0.04 ± 0.01*	$b*$ 0.81±0.64*
T3 (nmol\L)	1.22 ± 0.02	0.77±0.21*	$a*$ 0.98 ± 0.39*	2.84 ± 0.79*	3.27±0.81*
T4 (nmol\L)	80.2±6.35	71.8±10.13*	74.45±9.35*	100.2 ±17.08*	93.7±11.67*
FBG (mg/dl)	86.9±8.05	90.10±5.93	$a*$ 186.16±17.17 *	88.8± 6.26	$b*$ 182.2± 15.72*
HbA1c%	4.965±0.30	5.88± 0.24*	$a*$ 7.75±0.80*	5.53± 0.60*	$b*$ 7.93± 0.90*
BMI (kg/m^2)	27.15±3.13	30.84±4.89*	33.59±5.70*	25.07 ± 3.01*	26.97±3.63
Adipsin (g\ml)	1.20±0.32	5.38 ± 0.57*	$a*$ 7.20 ± 0.79*	3.15 ± 1.90*	$b*$ 6.20 ± 1.19*

*t-test between all patients' groups and control; ^{a*} t-test between G1 and G2;

^{b*} t-test between G3 and G4

Table 2. Assessment of lipid panel in controls and experimental groups.

Parameters	Control No.30	G1 No.25	G2 No.25	G3 No.25	G4 No.25
TG (mg/dl)	146.85±22.95	207.25±13.66*	205.55±18.87*	167.5±2.62*	$b*$ 180.15±4.23*
Chol (mg/dl)	161.7±21.50	216.4±16.46*	210.4±25.33*	164.85±7.65	170.35±11.45
HDL-ch (mg/dl)	46.5±4.650	37±1.58*	38.35±5.87*	45.55±4.96	44.65±5.64
LDL-ch (mg/dl)	70.61±20.43	137.95±14.74*	123.77±33.99*	76.83 ± 4.54	$b*$ 80.47±5.77*

*t-test between all patients' groups and control; ^{a*} t-test between G1 and G2;

^{b*} t-test between G3 and G4

Table 3. Pearson correlation coefficient (r) and P-value between Adipsin and all studied parameters in controls and patients with thyroid dysfunction.

Parameters	Control r	G1 r	G2 r	G3 r	G4 r
TSH	-0.172*	0.085	-0.303	0.070*	0.205*
T3	-0.155	0.051*	0.020*	-0.222	-0.190*
T4	-0.440*	-0.297*	-0.449*	0.267*	-0.546*
FBG	-0.082*	-0.231*	0.034*	0.175*	-0.089*
HbA1C	-0.196*	0.503*	-0.211*	-0.067*	-0.153*
BMI	-0.108*	0.194*	0.348 *	0.076*	0.316*
TG	-0.066*	-0.122*	-0.152 *	0.369*	-0.003*
Chol	0.141*	-0.077*	-0.055*	-0.188*	0.167*

Parameters	Control r	G1 r	G2 r	G3 r	G4 r
HDL	-0.144*	-0.000*	0.213*	-0.197*	0.067*
LDL	0.028*	-0.064*	-0.071*	-0.120*	-0.138*

*t-test between Adipsin and studied parameters in control and patients' groups.

Table 4. Sensitivity, specificity & cut-off value of Adipsin in patients groups.

Roc of Adipsin	Sensitivity	Specificity	Area under curve	Accuracy	Asymptotic Sig.	Cut off value
G1	100%	100%	1.000	1.000	0.000	1.676
G2	100%	100%	1.000	1.000	0.000	1.676
G3	85.00%	90.00%	0.917	0.86	0.0453	1.579
G4	100%	100%	1.000	1.000	0.000	1.676

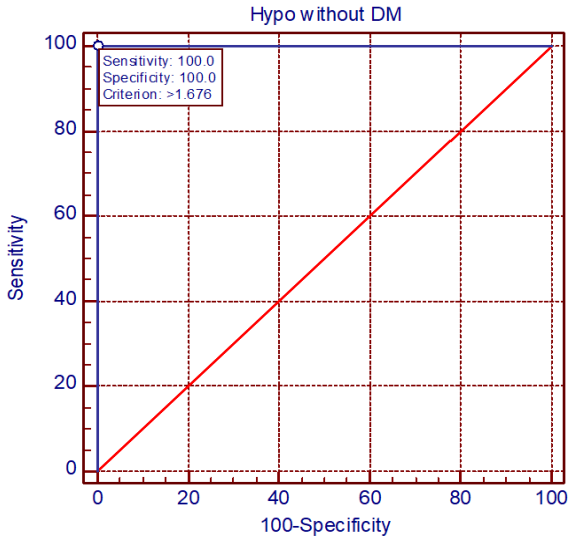

Figure 1. The ROC curve for adipsin in hypothyroidism patients and controls (**Designed by Authors, 2025**).

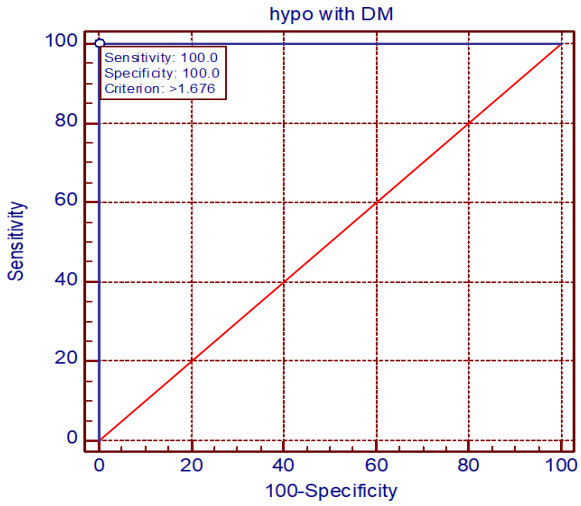

Figure 2. The ROC curve for adipsin in hypothyroidism patients with DM and controls (**Designed by Authors, 2025**).

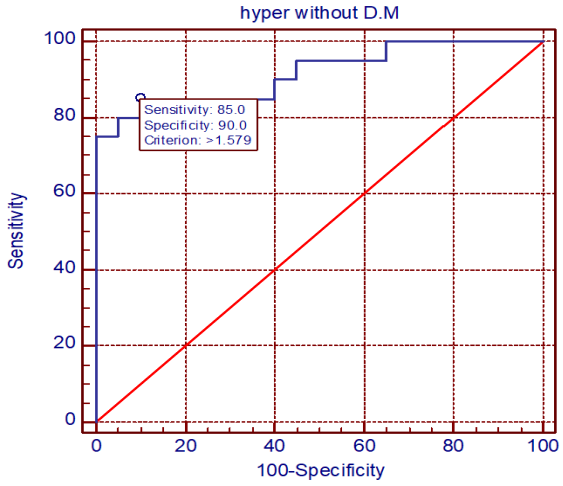

Figure 3. The ROC curve for adipsin in hyperthyroidism patients and controls (**Designed by Authors, 2025**).

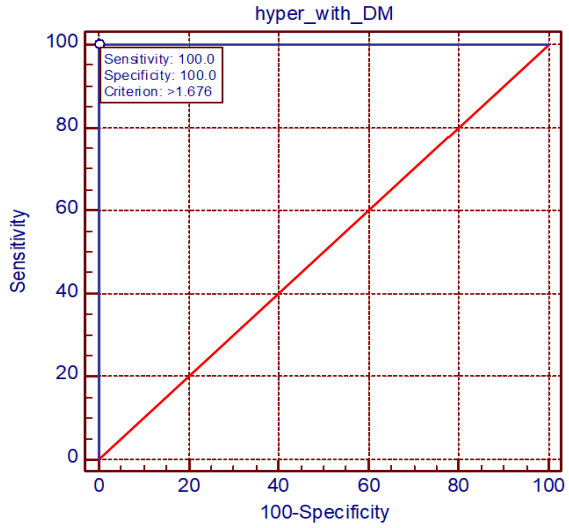

Figure 4. The ROC curve for adipsin in hypothyroidism patients with DM and controls (**Designed by Authors, 2025**).

4. Discussions

The present study aimed to evaluate adipsin and lipid levels in Iraqi women with thyroid dysfunction, to determine whether adipsin could serve as a diagnostic marker for thyroid disorders, and to monitor the progression of diabetes mellitus in these patients. Additionally, the study sought to assess whether these patients are at increased risk of developing cardiovascular diseases (CVDs). The current findings regarding TSH, T3, and T4 levels are consistent with those reported by Dawood et al (16) and with study Abass et al (17). Thyrotropin-releasing hormone (TRH) stimulates the anterior pituitary gland to secrete thyroid-stimulating hormone (TSH), which in turn acts on the thyroid gland to promote the production and release of thyroxine (T4) and triiodothyronine (T3). These hormones exert negative feedback on both TRH and TSH secretion, maintaining homeostatic regulation of thyroid function. Disruption of this feedback loop results in hypothyroidism or hyperthyroidism, depending on the direction of the imbalance (18). Thyroid hormones play an essential role in regulating glucose metabolism. Both hypothyroidism and hyperthyroidism are associated with impaired glycemic control due to increased insulin resistance. Diabetes mellitus can also influence TSH function and the peripheral conversion of T4 to T3, leading to hyperinsulinemia and insulin resistance (19-21).

In the present study, there was a significant increase in fasting blood glucose (FBG) and HbA1c levels in both Group 2 and Group 4 compared with the control group. These findings agree with those of Al-Rubaei et al (22). Thyroid hormones affect glucose metabolism through multiple mechanisms. Hyperthyroidism, in particular, has long been associated with hyperglycemia due to a shortened insulin half-life, likely resulting from accelerated insulin degradation and increased release of biologically inactive insulin precursors (23).

A significant positive correlation was observed between serum adipsin levels and body mass index (BMI) in thyroid patients with type 2 diabetes mellitus (T2DM). This suggests that adipsin may serve as a biomarker for monitoring the progression of diabetes in such patients. This finding aligns with the study by Milek et al (24), which suggested that adipsin could act as both a diagnostic marker in thyroid dysfunction and a prognostic marker for diabetes mellitus, though further validation is needed. Adipsin, an adipokine, is believed to link increased fat mass and adipose tissue dysfunction with cardiometabolic disorders related to obesity. Measuring adipsin levels could be diagnostically valuable for identifying individuals at elevated risk of β-cell dysfunction and rapid progression to diabetes. In T2DM patients, stratification by adipsin level may help identify those requiring closer monitoring, even when other glycemic parameters appear normal (25).

The current study also revealed a significant increase in adipsin levels among thyroid patients with T2DM. According to Farhan et al (26), adipsin is one of the most specific and sensitive markers for diagnosing diabetic neuropathy. Elevated adipsin concentrations may result from decreased insulin sensitivity in T2DM patients with neuropathy. Furthermore, adipsin levels have been positively correlated with insulin resistance, highlighting its potential diagnostic role in T2DM (26).

Mechanistically, adipsin may contribute to the pathogenesis of obesity and T2DM through its regulation of complement system components, particularly complement factor C3. Adipsin catalyzes the generation of C3a, which can enhance insulin secretion from pancreatic β-cells during hyperglycemia (25). Adipose tissue secretes adipokines, including adipsin, into circulation, influencing cardiovascular disease (CVD) development. Although the association between adipsin and CVD is well established, the precise mechanisms remain unclear (27). Serum adipsin has been linked to insulin resistance, especially in individuals with higher BMI and elevated blood glucose levels, suggesting its involvement in abnormal glucose metabolism and the need for further research (28).

Jawzal et al (29) demonstrated that thyroid hormones significantly influence lipid profile parameters in subclinical hypothyroidism (SCH), as indicated by increased LDL-C and decreased HDL-C levels. These results suggest that patients with thyroid dysfunction are at greater risk of cardiovascular disease. Therefore, regular monitoring for dyslipidemia is critical for initiating early treatment and recommending appropriate lifestyle modifications (29). The elevation in total cholesterol (TC) levels may be attributed to the direct action of thyroid hormones on the Niemann-Pick C1-like 1 protein in the intestine, leading to enhanced cholesterol absorption (30). Moreover, thyroid hormones regulate LDL receptor expression; thus, in SCH, the number of these receptors decreases, reducing LDL-c catabolism. The observed decline in HDL-c may also be due to the influence of thyroid hormones on HDL binding sites (31). Dyslipidemia remains a major risk factor for cardiovascular disease, emphasizing the importance of lipid profile monitoring in patients with thyroid disorders to prevent cardiovascular complications (7). The increased cardiovascular risk in both hyperthyroid and hypothyroid states arises not only from lipid abnormalities but also from hemodynamic alterations, endothelial dysfunction, coagulation disorders, and metabolic imbalances, all of which increase susceptibility to atherosclerotic disease. In hypothyroidism, lipid disturbances partially explain the elevated incidence of coronary artery disease (32, 33).

Novelty of the study

This study highlights the relationship between adipsin and diabetes mellitus in Iraqi women with thyroid disorders within the geographical context of Baghdad. Additionally, it demonstrates that patients with hypothyroidism are more prone to cardiovascular diseases compared to those with hyperthyroidism.

5. Conclusion

The findings of this study show that serum adipsin levels were significantly elevated in women with both hypothyroidism and hyperthyroidism. Therefore, adipsin may serve as a diagnostic biomarker for thyroid dysfunction and for monitoring the progression of diabetes mellitus in these patients. Moreover, the significant dyslipidemia observed, particularly in hypothyroid patients, places them at higher risk of cardiovascular disease compared with hyperthyroid patients. The ROC analysis of adipsin levels among thyroid patients and control groups demonstrated excellent cutoff values, sensitivity, and specificity, indicating that adipsin may be a reliable diagnostic biomarker for thyroid dysfunction and a potential tool for tracking cardiovascular disease progression. Given the increased cardiovascular susceptibility among individuals with thyroid disorders, routine screening for dyslipidemia is crucial to enable early intervention and appropriate lifestyle modifications.

6. Declarations

6.1 Acknowledgments

Not applicable.

6.2 Ethical Considerations

The study protocol was reviewed and approved by the Ministry of Health, Baghdad Health Directorate (Rusafa), under approval number 126249, dated December 18, 2024. All participants provided informed consent prior to enrollment. Ethical clearance was also obtained from the Ethics Committee of the Specialized Center for Endocrinology and Diabetes.

6.3 Authors' Contributions

All authors participated in the preparation and review of the manuscript. Study conception & design: Eiman AA. Abass. Literature search: Maryam A. Hassoun & Eiman AA. Abass. Data acquisition & Data analysis: (Eiman AA. Abass). Manuscript preparation Maryam A. Hassoun & Eiman AA. Abass, editing & review: Eiman AA. Abass. The authors confirm that all the Figures and Tables in the manuscript belong to the current study.

6.4 Conflict of Interest

The authors have no conflict of interest.

6.5 Fund or Financial Support

This research received no specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

6.6 Using Artificial Intelligence Tools (AI Tools)

The authors did not use artificial intelligence programs in preparing this article.

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