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Estimation of Resistin and Adiponectin Levels in Iraqi Women with Hypothyroidism

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Abstract

Hypothyroidism, the most common clinical condition associated with thyroid hormone deficiency, can pose significant health risks. Adipokines, such as resistin and adiponectin, are hormones secreted by adipose tissue. Research has shown that lipid profiles and adipokine levels are altered in individuals with thyroid dysfunction. The aim of this study was to evaluate serum levels of adiponectin and resistin in Iraqi women newly diagnosed with hypothyroidism and to investigate the relationship between such levels of thyroid hormones and lipid profile. The study included one hundred women in the age 20-50 years old. They were split into two groups: fifty patients with recently diagnosed hypothyroidism and fifty healthy women who are as controls. In contrast to the healthy group, the hypothyroidism group's concentrations of fasting blood glucose (FBG), body mass index (BMI), total cholesterol (TC), thyrotropin (TSH), very low-density lipoprotein (VLDL), triglyceride (TG), low-density lipoprotein (LDL), and resistin all significantly increased, while thyroxine (T4), triiodothyronine (T3), high-density lipoprotein (HDL), and adiponectin levels decreased. An important positive correlation was observed between adiponectin and T4 and T3. In terms of diagnostic performance, adiponectin and resistin showed high AUC values of 0.915 and 0.975, respectively, in the case of comparing hypothyroidism to the control group. In conclusion, hypothyroid patients had lower adiponectin and higher resistin levels, indicating these parameters could be a diagnostic marker for hypothyroidism patients because of excellent discrimination between hypothyroidism patients and healthy controls.

Keywords: adiponectin, resistin, lipid profile, thyroid dysfunction, hypothyroidism

تقدير مستويات الريزيستين والأديبونكيتين لدى النساء العراقيات المصابات بقصور الغدة الدرقية

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الخلاصة

قصور الغدة الدرقية، وهو الحالة السريرية الأكثر شيوعًا المرتبطة بنقص هرمونات الغدة الدرقية، يمكن أن تشكل مخاطر صحية كبيرة. الأديبوكينات، وتشمل الريزيستين والاديبونكيتين، هي هرمونات تفرزها الأنسجة الدهنية. أظهرت الأبحاث أن مستويات الدهون والأديبوكينات تتغير لدى الأفراد الذين يعانون من خلل في الغدة الدرقية. هدفت هذه الدراسة إلى تقييم مستويات الأديبونكيتين والريزيستين في مصل النساء العراقيات اللاتي تم تشخيص إصابتهن بقصور الغدة الدرقية مؤخراً، والتحقيق في العلاقة بين مستوياتهما مع هرمونات الغدة الدرقية

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ومستوى الدهون. شملت الدراسة مائة امرأة تتراوح أعمارهن بين (20−50) سنة. تم تقسيمهن إلى مجموعتين: 50 مريضة تعاني من قصور الغدة الدرقية المشخص حديثاً و50 امرأة سليمة كانت بمثابة مجموعة ضابطة. أظهرت النتائج أن تراكيز سكر الكلوكوز في الدم للصائم (FBG) ، مؤشر كتلة الجسم (BMI)، الكوليستيرول الكلي (TC)، الثيروتروبين (TSH)، البروتين الدهني منخفض الكثافة جدا (VLDL)، الدهون الثلاثية (TG)، البروتين الدهني منخفض الكثافة (LDL)، والريزيستين جميعها زادت بشكل ملحوظ في مجموعة قصور الغدة الدرقية، ولكن انخفضت مستويات الثايروكسين (T4) ، ثلاثي يودوثيرونين (T3)، البروتين الدهني عالي الكثافة (HDL) و الأديبونكيتين مقارنة بالمجموعة السليمة. كان هناك ارتباط إيجابي كبير بين الأديبونكيتين مع T3 وT4. فيما يتعلق بدقة التشخيص، عند مقارنة قصور الغدة الدرقية مع المجموعة الضابطة، اظهر الأديبونكيتين والريزيستين قيم المساحة تحت المنحني (AUC) عالية تبلغ 0.915 و0.975 على التوالي. يمكن الاستنتاج بأن لدى مرضى قصور الغدة الدرقية مستويات اقل من الأديبونكيتين و اعلى من الريزيستين، لذلك يمكن أن تكون هذه المعلمات علامة تشخيصية لمرضى قصور الغدة الدرقية بسبب وجود تمييز ممتاز بين مرضى قصور الغدة الدرقية والأشخاص الأصحاء.

1. Introduction

The thyroid gland is a vital organ in the body responsible for control and harmony of the body's growth and metabolism [1]. Thyroid dysfunction is considered the second most common glandular disorder regarding the endocrine system [2, 3], which are associated with either inadequate or excessive thyroid hormone production, are hypothyroidism and hyperthyroidism respectively [4]. Since thyroid hormone is necessary for all metabolically active cells, a deficiency in the hormone can have a variety of repercussions [5]. The highly sensitive negative feedback mechanism between thyroid gland failure and the hypothalamic-pituitary-thyroid axis results in elevated levels of pituitary thyrotropin (TSH) and low serum thyroid hormone levels in cases of primary hypothyroidism [6, 7].

Adipose tissue acts as an active metabolic and endocrine organ, secreting a range of hormones, cytokines, and growth factors, which have effects on both local tissue and distant organs as well as tissues throughout the body [8]. The hormones derived from adipocytes, known as adipokines or adipocytokines, include leptin, resistin and adiponectin known as adipokines or adipocytokines [9]. Thyroid hormones and these adipokines share some physiological functions, such as controlling energy expenditure and the metabolism of fats and carbohydrates. Thus, it is conceivable that there is an interaction between the action of adipose tissue and the thyroid axis. Therefore, thyroid dysfunction may influence the action of adipose tissue, and together they can alter the metabolism and energy homeostasis, which contributes to other metabolic disorders [10].

Adiponectin (~30 kDa) is the most abundant adipocytokine and possesses significant antihyperglycemic, anti-atherogenic, and anti-inflammatory properties, which could have an important implication on the development of therapies for the treatment of several diseases [11,12]. Research investigating the relationship between adiponectin to thyroid hormones have shown that shifts in serum levels of either cause fluctuations in the other, though findings have been mixed [13,14]. Experimental data suggest that adiponectin and thyroid hormones may interact with each other. Specifically, adiponectin might influence thyroid hormone production through interaction with the gC1q receptor of thyroid cell mitochondria, while alterations in the pituitary-thyroid axis may alter adiponectin levels. This interaction may occur through the PPAR pathway, adiponectin messenger RNA expression in the adipose tissue, or TSH receptors in adipose tissue [15].

Lazar and Steppan identified resistin, a cysteine-rich protein that is widely distributed in the circulatory system, in 2001 through the encoding of the RETN gene. When injected into mice,

resistin has been linked to type 2 diabetes and insulin resistance [16]. Since it's discovered, resistin has attracted a lot of research because of its multiple physiological and clinical roles in a range of metabolic illnesses. Furthermore, resistin is more effective than other adipokines in the regulation of inflammation, immunity, energy expenditure, endocrine function, bone metabolism, and energy expenditure [17]. It has long been considered a pro-inflammatory molecule which controls a number of infectious, chronic, and metabolic inflammatory illnesses in addition to human cancers [18–20]. Increased resistin mRNA levels in white adipose tissue have been linked to hypothyroidism in previous animal study [17]. In human studies examining the relationship between resistin levels and thyroid dysfunction, findings have shown a range from high to low resistin levels, with evidence being inconsistent [21–23].

Hypothyroidism is typically linked to mild weight gain, reduced thermogenesis, and metabolic rate. As many of these changes are associated with alterations in the function of adipose tissue, it seems of interest to assess the relationship between adipocytokines, thyroid hormones, and thyroid dysfunction [24]. Despite the high prevalence of thyroid dysfunction in Iraq, there have been very few studies examining the interesting relationship between thyroid hormones and adipokines in hypothyroid subjects in more depth. Therefore, the aim of this study was to evaluate serum levels of adiponectin and resistin in Iraqi women with hypothyroidism and to elucidate the relationship between such levels and thyroid dysfunction (hypothyroidism). Also, utilized receiver operating characteristic curve (ROC) analysis to test whether it is possible to employ the serum levels of adiponectin and resistin for the diagnosis of hypothyroidism.

2. Materials and Methods

2.1 Study design:

One hundred Iraqi women, aged between 20-50 years, participated in this study. They were divided into two groups of fifty subjects each: one group consisted of patients with newly diagnosed hypothyroidism and one group of healthy subjects served as controls. Participants were recruited verbally following being told of the study's objectives in Baghdad Medical City in Baghdad, Iraq, between October 2023 and January 2024. The Ethical Committee of the University of Baghdad College of Science approved this work (Ref. CSEC/1023/0066 on October 28, 2023).

The exclusion criteria of the study included women with a history of using corticosteroids, thyroidectomy, medications for thyroid disease, hyperthyroidism, pregnancy, thyroid cancer, diabetes mellitus, smoking, taking nutritional and antioxidant supplements, and a family history of thyroid disease, which may interfere with the study.

2.2 Blood samples preparation:

A blood sample of 5ml was collected from the subjects after they had fasted for 10 to 12 hours. The blood was drawn into a gel tube for serum separation. After that, the blood was centrifuged for ten minutes at 3000 rpm; the serum has been separated and kept at -20°C until analysis.

2.3 Laboratory assays:

The body mass index (BMI) was calculated using the basic anthropometric measurements of weight and height: weight in (kg)/height in m^2 [25]. Individuals who had a 30kg/m^2 or higher BMI were deemed obese and were not included in this study. Using an enzyme-linked immunoassay sandwich technique (DRG, Germany), serum TSH, total T3, and total T4 have been assessed as part of the thyroid profile function. Furthermore, the levels of serum

adiponectin and resistin were quantitatively determined using the sandwich ELISA technique (Cloud-clone Corp, USA), which provided a precise measurement of these biomarkers. Additionally, total cholesterol (TC), fasting blood glucose (FBG), triglyceride (TG), and high-density lipoprotein (HDL) have been measured with the use of an enzymatic colorimetric approach utilizing the available kit that has been manufactured by Linear, Spain. In parallel, the Fried Ewald and Levy formula was used to determine the very low-density lipoprotein (VLDL) and low-density lipoprotein (LDL) [26].

2.4 Statistical Analysis:

Statistical analysis was conducted using SPSS version 26. The data are presented as means ± SD. The differences between variables have been ascertained with the use of the unpaired student T-test. At $p<0.05$, the difference has been deemed statistically significant; at $p<0.01$, it was deemed very significant; and at $p>0.05$, there has not been any significant difference. Pearson's correlation was used to investigate the relationships between variables in the two groups. Receiver operating characteristic (ROC) was performed to evaluate the possibility of using adiponectin and resistin in the prognostic of hypothyroidism by measuring the area under the curve (AUC) with the cut-off values and their sensitivity and specificity.

3. Results

Table 1 presents a comparison of the studied parameters between hypothyroid group and healthy control group. The mean ± SD ages for the control and patient groups were 33.00 ± 9.50 and 34.44 ± 9.56 years, respectively, indicating no statistically significant difference in age between groups ($p>0.05$). Also, Table 1 illustrates that the patient's BMI (26.41 ± 2.73 Kg/m^2) was significantly higher than the control's (23.75 ± 1.40 Kg/m^2). The findings demonstrated that, in comparison with the controls, the patient group's level of FBG had significantly increased ($p=0.010$).

Table 1: The studied parameters comparisons between the hypothyroid group and the healthy subject group

Parameters	*Groups*		*p-value*
	Control n=50	*Hypothyroidism n=50*	
Age (years)	33.00 ± 9.50	34.44 ± 9.56	0.428
BMI (Kg/m^2)	23.75 ± 1.40	26.41 ± 1.81	<0.001
FBG (mg/dl)	93.21 ± 6.98	102.11 ± 19.08	0.010
TSH (mlU/ml)	1.713 ± 0.664	9.442 ± 2.825	<0.001
Total T3 (nmol/L)	1.226 ± 0.386	0.502 ± 0.195	<0.001
Total T4 (nmol/L)	97.53 ± 19.28	20.96 ± 2.93	<0.001
TC (mg/dl)	139.66 ± 27.07	201.56 ± 40.51	<0.001
TG (mg/dl)	121.61 ± 14.44	184.32 ± 21.05	<0.001
HDL (mg/dl)	46.00 ± 4.45	39.86 ± 4.51	<0.001
LDL (mg/dl)	69.34 ± 28.66	124.84 ± 41.87	<0.001
VLDL (mg/dl)	24.32 ± 2.88	36.86 ± 4.21	<0.001
Adeponectin (ng/ml)	3.716 ± 0.584	3.166 ± 1.173	0.040
Resistin (ng/ml)	31.91 ± 6.40	38.20 ± 18.10	0.027

The difference is significant at $p<0.05$ and highly significant at $p<0.01$

The TSH level in hypothyroid patients was significantly elevated at 9.442 ± 2.825 mIU/ml compared to the control, which had a TSH level of 1.713 ± 0.664 mIU/ml. While the levels of total T3 (1.226 ± 0.386 nmol/L) & total T4 (97.53 ± 19.28 nmol/L) in control group were high in comparison with the hypothyroid group's total T3 (0.502 ± 0.195 nmol/L) & total T4 (20.96 ± 2.93 nmol/L) and this decrease was statistically significant.
Hypothyroid individuals exhibited significantly higher ($P<0.01$) levels of TG, TC, LDL, and VLDL, while their HDL levels were significantly lower compared to the controls.
Table 1 demonstrates a significant drop in serum adiponectin levels (3.166 ± 1.173 and 3.716 ± 0.584 ng/ml, respectively) between hypothyroid patients and controls. On the other hand, resistin levels in the patient group (38.20 ± 18.10 ng/ml) have been significantly higher than those in the control group (31.91 ± 6.40 ng/ml).
The Pearson correlation coefficient for serum adiponectin and resistin across all investigated factors, as displayed in Table 2. Adiponectin and (T4 and T3) showed a strong positive correlation, while no significant correlation with the other variables. However, serum resistin did not significantly correlate with any of the other variables under investigation.

Table 2: The Pearson correlation between Adiponectin and Resistin with all parameters in the patient group

Parameters	Adiponectin (ng/ml)		Resistin (ng/ml)	
	r	P	r	p
BMI (Kg/m^2)	0.019	0.893	-0.101	0.486
FBG (mg/dl)	0.014	0.922	0.035	0.807
TSH (mlU/ml)	-0.074	0.607	0.009	0.949
T3 (nmol/L)	0.280*	0.049	-0.196	0.172
T4 (nmol/L)	0.260*	0.038	0.127	0.378
TC (mg/dl)	0.139	0.336	-0.080	0.579
TG (mg/dl)	-0.167	0.246	-0.014	0.921
HDL (mg/dl)	-0.028	0.849	-0.144	0.318
LDL (mg/dl)	0.157	0.275	-0.072	0.619
VLDL (mg/dl)	-0.167	0.246	-0.014	0.921
Adiponectin (ng/ml)	-	-	-0.077	0.596
Resistin (ng/ml)	-0.077	0.596	-	-

*Correlation is significant at the 0.05

The ROC analysis was utilized to evaluate the area under curve (AUC) that assess the overall diagnostic performance of adiponectin and resistin in hypothyroid patients, as presented in Fig 1 and Table 3. Additionally, the best cut-off value, sensitivity, and specificity were calculated. The AUC for adiponectin and resistin were found to be 0.915 and 0.975, respectively; suggesting excellent discrimination between hypothyroidism and control groups, with a cutoff value of 3.401 and 34.277 ng/ml, it had a sensitivity of 94% and 96% with a specificity of 96% and 100%, respectively, as listed in Table (3).

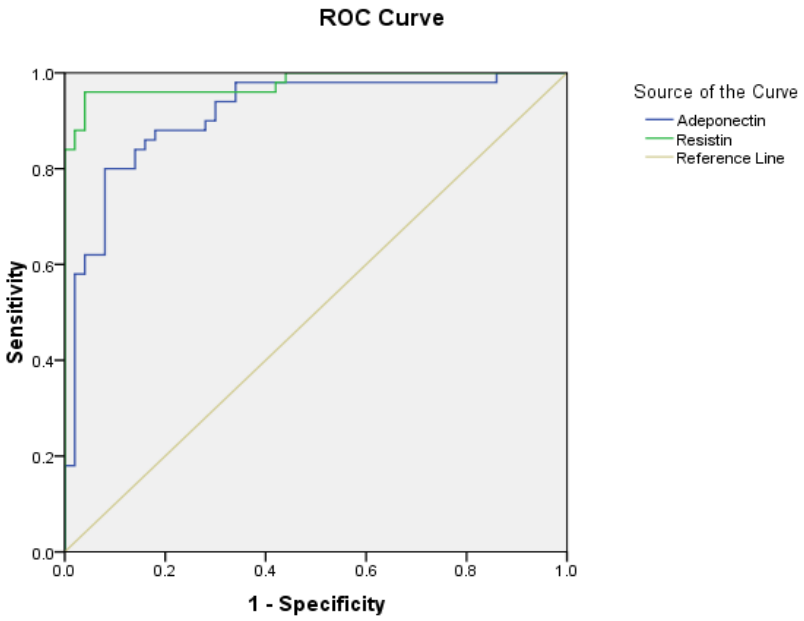

Figure The ROC curve of Adiponectin and Resistin within in hypothyroid group compared with the controls

Table 3: The ROC curve analysis for adiponectin and resistin to control hypothyroid patients

Test Result Variable(s)	*Area under the curve (AUC)*	*Asymptotic Sig.* [a]	Sensitivity %	Specificity %	Cut off value	Asymptotic 95% Confidence Interval	
						Lower Bound	Upper Bound
Adeponectin (ng/ml)	0.915	0.001	94	96	<3.401	0.858	0.972
Resistin (ng/ml)	0.975	0.001	96	100	>34.277	0.954	1.000

a. Null hypothesis: true area = 0.5

4. Discussion

The present study aimed to investigate the effects of adiponectin and resistin effects on hypothyroidism. Thyroid hormones are crucial for cellular metabolism, regulating various pathways involved in the metabolism of lipids, proteins, carbohydrates, and fat oxidation in a range of target tissues throughout life [27]. A prevalent illness in the general population is hypothyroidism, a clinical condition characterized by a deficit in thyroid hormone. TSH levels beyond the upper limit of the reference range and thyroid hormone (T4 and T3) levels below the lower limit of the reference range are its defining characteristics [28]. The findings of this study indicate that patients with hypothyroidism are overweight (BMI 25-29.90), yet not obese (BMI > 30), with their BMI 26.41 ± 2.73 Kg/m^2 considerably higher than that of the control group, which is normal 23.75 ± 1.40 Kg/m^2. As previously indicated, hypothyroidism affects how all body cells metabolize fat, especially in adipose tissue, which ultimately results in obesity. BMI is commonly used to assess metabolic syndrome and obesity. Overt thyroid dysfunction was demonstrated to have an influence on body weight in numerous researches investigating the relationship between thyroid hormones and BMI [29-31]. Additionally, there is a correlation between a greater BMI and a higher incidence of hypothyroidism and obesity. It has also been connected to decreased metabolic rate and thermogenesis. Additionally, clinical data shows that subclinical hypothyroidism, a minor form of thyroid dysfunction, is linked to notable changes in body weight and serves as an example of a risk factor for obesity

and overweight. Consequently, weight increase could be the outcome of clinical hypothyroidism [32].

In the current study, women with hypothyroidism exhibited significantly higher FBGs levels compared to control group (p=0.010). This finding aligns with previous research indicating that hypothyroidism can lead to elevated blood sugar levels. Blood sugar levels in hypothyroidism patients are frequently found to be poorly regulated. Insulin resistance (IR) is linked to the rise in blood sugar. Various pathogenesis mechanisms as well as defects in insulin receptors, such as altered muscle fiber composition, decreased glycogen synthesis, altered trans-capillary insulin delivery, aberrant phosphorylation of insulin signaling proteins, and impaired oxidative phosphorylation of mitochondria, might lead to IR status due to hypothyroidism [33, 34]. Thyroid hormone levels in the hypothyroid and control groups differed significantly, according to the data. In comparison to the control group, the hypothyroid group had considerably higher TSH levels together with decreased total T3 and T4 levels. These results align with the typical hormonal dysregulation associated with hypothyroidism. In addition, a prior study by AL-Shammaree and Jouad [1,27] discovered that hypothyroid patients had significantly lower T4 and T3 levels and much higher TSH levels in comparison to controls. Thyrotropin-releasing hormone (TRH) from the hypothalamus induces the anterior pituitary gland to release TSH. The amount of thyroid hormone in the blood affects this production. There is an inverse relationship between thyroid hormone levels and TSH; even slight variations in T4 concentration can significantly affect TSH production, making it a sensitive indicator of thyroid function in clinical diagnoses. As a result, elevated TSH levels could suggest hypothyroidism, while low TSH levels could imply hyperthyroidism [35].

Compared to the control group, the hypothyroidism group showed increased levels of TG, TC, VLDL, and LDL increase, whereas HDL decreases. Lipid synthesis, circulation, absorption, and metabolism are all altered in hypothyroidism, which is associated with hyperlipidemia [36]. The thyroid hormone regulates cholesterol synthesis in the liver, the primary organ for the production of cholesterol, through several pathways. Hepatic lipase, cholesteryl ester transfer protein (CETP), lecithin: cholesterol acyltransferase, and 3-hydroxy-3-methyl-glutaryl coenzyme A reductase activity are all boosted by thyroid hormone [37]. Furthermore, it makes sense that low HDL levels and high serum levels of TC, TG, and LDL might be associated with insufficient thyroid hormone [38]. The present results corroborate those of Hashim *et al*. [39] and Lefta *et al*. [40], who discovered significantly elevated levels of LDL, TC, VLDL, and TG in patients who have hypothyroidism, along with a reduction in HDL compared to the controls. However, the present findings were in contrast with those of Aati *et al*. [41], who observed that participants with hypothyroidism had normal HDL levels.

In the situation of thyroid dysfunction, adipocytokines, active biological molecules produced by adipose tissue, play either a protective or causal role [42]. Resistin, leptin, visfatin, and adiponectin are some of such adipocytokines [17]. With conflicting outcomes, abnormal adipocytokine levels were linked to thyroid dysfunction (hyperthyroidism and hypothyroidism) [43]. Resistin levels have been found to be significantly greater in the hypothyroidism group than in the controls in the present investigation. These findings are in line with the results of a previous study by Zhou *et al*. [17], which reported that individuals with hypothyroidism had significantly higher levels of resistin compared to healthy controls. This suggests that resistin levels and thyroid dysfunction might be significantly related. Also, Hedayati *et al*. reported that resistin serum levels in hypothyroidism patients were significantly higher than in the control group, and no significant correlation was observed between resistin levels, BMI, age, and thyroid hormones level [44], which is in the line with the current findings.

A few studies have examined the relationship between resistin and thyroid hormones; conflicting results have been documented. Another study by Krassas *et al*. showed that resistin levels in patients with hypothyroidism were similar to those in the control group [45]. A study by Kaplan *et al*. demonstrated that no significant changes in resistin, and adiponectin levels occurred in thyroidectomy-induced hypothyroidism when compared to the euthyroid state [46]. Another study showed a positive association between resistin levels and thyroid hormones [47], which is in contrast with our findings. While, Iglesias *et al*. [48] observed that resistin levels in hypothyroid patients were significantly lower than euthyroid control group, which also conflict with our results.

According to the current study, adiponectin levels are positively correlated with T4 and T3 levels, and they are significantly lower in women who have hypothyroidism when compared to healthy controls. Those findings have been consistent with those by Sharma *et al*. [49], who similarly observed a significant decrease in serum adiponectin levels and a significant correlation between the thyroid hormones T4 and T3, in female patients with hypothyroidism. Considering that adiponectin and thyroid hormones both reduce body fat through enhanced thermogenesis and lipid oxidation, among other physiological effects [50]. The lower metabolic rate and altered metabolism associated with hypothyroidism may cause alterations in adiponectin levels [51]. Another study indicated increased adiponectin levels in hypothyroid patients than in healthy control [15]. A study conducted by Mukherjee *et al*. found that the adiponectin level was decreased marginally in hypothyroid patients as compared to euthyroid control which is statistically insignificant. They also found that there was a positive association between thyroid hormones and adiponectin levels and negative association between TSH and adiponectin [52]. In contrast to our findings, other studies found no changes in serum adiponectin levels in hypothyroidism [48,53]. Furthermore, since serum T4 levels have been linked to adiponectin levels, decreased adiponectin levels in hypothyroidism might suggest that thyroid hormones regulate adiponectin in living things [54]. Brenta [55] suggests that there could be an indirect or direct connection between adiponectin and thyroid hormones. Adiponectin plays a complex role in the metabolic consequences of hypothyroidism. Its level can be elevated in hypothyroid patients, possibly as a compensatory mechanism or due to secondary resistance of adiponectin receptors [56]. Despite its anti-inflammatory and insulin-sensitizing effects, adiponectin levels are typically lower in obesity, a condition that often coexists with hypothyroidism. The relationship between adiponectin and thyroid hormones is not entirely clear, as studies have shown variable results regarding adiponectin levels in different thyroid states [57]. The association between thyroid dysfunction and adipokines such as adiponectin and resistin involves complex mechanisms. Resistin is linked to IR and inflammation, with potential roles in thyroid dysfunction through pro-inflammatory pathways [58]. Hypothyroidism is associated with elevated resistin levels, which may contribute to IR and inflammation [17]. Adiponectin, which has anti-inflammatory properties, shows variable levels in thyroid disorders, potentially due to its interaction with thyroid hormones that influence energy metabolism and body composition [14,59]. These adipokines may mediate metabolic consequences of thyroid dysfunction, including altered glucose and lipid metabolism [60].

Adiponectin has been identified as an effective diagnostic marker with a cut-off value of 3.401; people below this level are considered patients. The ROC test for adiponectin revealed a great cut-off value with 96% specificity and 94% sensitivity. Resistin can be defined as one of the good diagnostic markers with a cut-off value of 34.277; persons greater than this level are termed patients. Interestingly, the ROC test for resistin indicated a good cut-off value with 100% specificity and 96% sensitivity.

Conclusions

This study found that patients with hypothyroidism had significantly higher levels of resistin and lower levels of adiponectin compared to the healthy control group. A positive significant correlation between adiponectin and thyroid hormones (T4 and T3) in hypothyroid patients might suggest that thyroid hormones regulate adiponectin. Resistin and adiponectin show excellent specificity and sensitivity in the patient group compared with the healthy controls, making them promising indicators of hypothyroidism and useful treatment targets.

Conflict of Interest

There was no conflict of interest.

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