Original Article

Irisin Levels and some Biochemical Parameters in Patients with Thyroid Cancer in Mosul City

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Abstract

Background: Thyroid cancer (TC) is a common endocrine disease that has become more prevalent in recent times, affecting multiple medical specialties. **Objective:** Due to the rise in instances of TC in Mosul city, the primary objective of the study was to examine levels of irisin, antithyroid peroxidase antibody (TPOAb) and some biochemical parameters in the blood of patients and compare their levels in healthy people, and to investigate the relationship of serum irisin with the development of the disease. **Materials and Methods:** From September 2021 until December 2022, 90 TC patients were chosen as the case group. The present study was created at the Oncology and Nuclear Medicine Hospital in Mosul and consisted of adults aged 25–55 years. Ninety healthy volunteers of the same age and gender constitute the control group. The subjects' medical histories, BMI, irisin, TPOAb, TSH, T3, T4 hemoglobin A1c (HbA1c), AST, ALT, and blood lipid levels had all been determined by gathering clinical data on each. The level of irisin in the blood was determined using an enzyme-linked immunosorbent assay. A ready-made analysis kit was used to estimate the above variables in the laboratories of the Department of Chemistry at the College of Science/University of Mosul and, for the most part, in the private laboratories in Mosul. Using SPSS 23.0, the data was analyzed. **Results:** The patient group's irisin concentration was significantly greater than that of the control group (14.797 ± 1 ng/mL and 4.428 ± 0.231 ng/mL, respectively), according to statistical analysis. TPOAb, HbA1c, TSH, ALT, total cholesterol, triglyceride, LDL-C and VLDL-C levels were higher, while T3 and T4 were significantly lower in the patient group compared to the control group. **Conclusion:** Since the current study shows that the serum irisin and TPOAb levels of TC patients are significantly higher than those of controls of the same age, irisin and TPOAb could be used as diagnostic markers for TC.

Keywords: TPOAb, cholesterol, irisin, thyroid cancer, TSH

Introduction

Thyroid tumors, one of the most common endocrine disorders, have been appearing more frequently lately.[1,2] About 1% of all malignancies are thyroid cancers (TCs), which result in 6–8 cancer-related deaths for every million people.[2] When compared to other cancers, they are ranked 25th out of the most frequent cancers in the US.[3] Similar to other types of cancer, there is relatively little knowledge about the root causes of TC. Firmly recognized risk variables for TC involve aging, female gender, exposure to ionizing radiation, a history of benign thyroid illness, and radiation exposure.[4] TC diagnosis is debatable,[5] and no reliable diagnostic guidelines have been established right now yet.

Thyroid peroxidase (TPO), a crucial enzyme for the synthesis of (T4) and (T3), is believed to be the microsomal antigen. Tyrosine residues are iodinated by TPO to produce monoiodotyrosine and diiodotyrosine. Proinflammatory cytokine responses are enhanced by the auto-antibodies, such as antithyroid peroxidase antibody (TPOAb), which is known to cause autoimmune thyroid illness.[6] Low quantities of TPOAb have been detected in the blood of healthy people.[7,8] TPOAb directly damages thyroid cells by fixing complement. Even in the preclinical stage of thyroid

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disorder, anti-TPOAb indicates lymphatic infiltration.[7,9] Irisin is one of the most recently discovered and isolated hormones, derived from mouse skeletal muscle in 2012. Irisin is secreted from muscles in response to exercise and may mediate some beneficial effects of exercise in humans, such as weight loss and thermoregulation. Irisin consists of 112 a.a residues.[10]

Results from the latest studies suggest that irisin may be a viable target for the diagnosis and therapy of many malignancies, as it plays a significant role in the incidence, growth, and metastasis of various tumors. Irisin is expected to have an impact on both thermogenesis and the basal metabolic rate, which are both significantly regulated by thyroid hormones. Irisin encourages the transformation of white adipose tissue into brown adipose tissue. Uncoupling protein-1 (UCP-1) is highly concentrated in brown adipose tissue. UCP-1 can enhance energy consumption and dissipate thermal energy produced by oxidation in the mitochondria, thus contributing to the maintenance of energy metabolism.[11] Numerous organs and tissues, such as the skeleton, liver, heart muscles, blood cells, spleen, connective tissue, and pancreas, produce irisin.[12,13] Cancer cells are susceptible to heat, and the thyroid is a thermogenic tissue.[14,15] Thyroid function may potentially be impacted by irisin. Energy consumption is impacted by thyroid hormone communication through both central and peripheral channels.[16] The human body's metabolic state is greatly influenced by thyroid hormones, which also affect the cardiovascular system and can control both facultative and required thermogenesis. It is reasonable to assume that serum irisin levels may be affected in thyroid dysfunction situations due to the physiological activities and probable relationships between irisin and thyroid hormones.[17]

Owing to the rise in cases of thyroid carcinoma in Mosul, therefore, this study attempted to establish a connection between serum irisin levels and measured biochemical parameters in the blood of patients and compare their levels in healthy people, detect the relationship of irisin with the development of the disease, and provide a theoretical framework for treatment and avoidance of TC.

Materials and Methods

Study design

Ninety TC patients were chosen for the study from a sample of individuals aged 23–55 years. Each subject gave verbal agreement after being selected from a single population at the Oncology and Nuclear Medicine Hospital in Mosul. Ninety healthy individuals (collected from students, employees, and lecturers at the University of Mosul) of the same age and gender were selected to form the control group. The control group did not include those with obesity, diabetes, hypertension, hyperlipidemia, TC, or hypertension.

Study setting and period

From September 2021 until December 2022, blood samples were collected from patients and controls.

Study sampling and sampling methods

After 12 h of fasting, blood samples were taken to assess levels of irisin, HbA1c, TPOAb, TSH, T3, T4 AST, alanine transaminase (ALT), and lipids. Obese TC patients had a BMI exceeding 25 kg/m^2, while lean TC patients had a BMI fewer than 25 kg/m^2. It included 87 thin and three obese TC patients. Patients with TC were further classified into 45 males and 45 females, and blood samples were taken. The manufacturer's reagents and calibrators were utilized to measure the serum concentrations of TSH using a calibrated Roche Cobas e601 analyzer. Two monoclonal antibodies that were specific for sterically non-interfering epitopes of human TSH—one biotinylated, the other tagged with a ruthenium complex—captured TSH. The streptavidin-coated magnetic microparticles ensnared all of the antibodies. The ruthenium complex then used voltage to produce photon emission from the microparticles, which was magnetically caught by an electrode. The serum TSH concentrations were inversely correlated with the luminescence intensities. The control samples and their corresponding case samples were analyzed in the same batch. Irisin and T3 and T4 concentrations were measured using EASIA kits from BioSource Europe in Nivelles, Belgium. The anti-TPO antibody test was carried out using (ELISA-EuroDiagnostica), which is designed to measure IgG antibodies in human serum that are directed against TPO. Anti-TPO antibody reference ranges are positive for values greater than 16 IU/mL and negative for values <16 IU/mL. The HbA1c was determined using the Abbott Clinical Chemistry Analyzer Architect c8000. The immunoturbidimetric method was used to measure the concentration of HbA1c. Using standard kits supplied by Biolabo/France, serum ALT, AST, total cholesterol, LDL-C, HDL-C, and triglycerides were measured.

Ethical approval

The research was carried out in compliance with the Declaration of Helsinki, and all study participants had given written informed consent. Nineveh Health's Research and Development Department's Ethics Committee examined and approved the study protocol, subject information, and approval on September 21, 2021, in accordance with document number 1012 to receive this approval.

Statistical analysis

For this statistical work, SPSS (version 23.0; SPSS Inc., Chicago, IL) was used. To compute the measurement data (SD), the mean standard deviation was employed. The *t* test was applied to assess continuous variables. The level of significance used in each instance was deemed to be <0.05.

Table 1: Evaluation of specific characteristics between TC patients and matching controls

	Patients ($n = 90$) Number (%)	Controls ($n = 90$) Number (%)	*P* value
Age (year)			
25–35	22(24.44)	23 (25.55)	NS
35–45	29 (32.22)	30 (33.33)	
≥45	39 (43.33)	37 (41.11)	
Gender			
Male	45 (50)	45 (50)	NS
Female	45 (50)	45 (50)	
BMI (Kg/m^2)			
<25	87 (96.66)	80 (88.88)	NS
25–29.9	3 (3.33)	10 (11.11)	

Table 2: Comparison of Irisin level and biochemical parameters between TC patients group and controls

Parameters	TC group ($n = 90$)	Control ($n = 90$)	*P* value
TPOAb (IU/L)	(+) > 16**	(−) < 16	0.0001
Irisin (ng/mL)	14.797 ± 1.88**	4.428 ± 0.231	0.0001
HbA1c (%)	6.874 ± 1.21*	5.353 ± 0.97	0.012
TSH (μ IU/L)	12.807 ± 2.24**	2.057 ± 0.532	0.0001
T3 (ng/dL)	0.944 ± 0.17**	1.284 ± 0.14	0.0001
T4 (ng/dL)	7.232 ± 1.94*	8.514 ± 1.53	0.013
AST (mg/dL)	28.44 ± 7.68	28.35 ± 5.56	0.232 (NS)
ALT (mg/dL)	35.71 ± 3.04**	29.51 ± 2.98	0.0001
T-Cholesterol (mg/dL)	167.24 ± 19.78**	122.03 ± 11.3	0.0001
TAC (mg/dL)	131.57 ± 7.95**	116.713 ± 10.37	0.0001
HDL-C (mg/dL)	40.03 ± 7.3	40.32 ± 9.4	0.452 (NS)
LDL-C (mg/dL)	100.62 ± 15.9**	58.42 ± 7.8	0.0001
VLDL-C (mg/dL)	26.35 ± 2.01**	23.343 ± 2.17	0.0001

TPOAb = antithyroid peroxidase antibody; HbA1c (%)= hemoglobin A1C; TSH = thyroid-stimulating hormone, T3 = triiodothyronine, T4 = thyroxine, AST= aspartate transaminase; ALT= alanine transaminase; T-Cholesterol= total cholesterol; TAG = triglycerides, HDL-C = highdensity lipoprotein cholesterol, LDL-C = lowdensity lipoprotein cholesterol

Values are mean ± standard deviation

*Significant difference ($P \leq 0.05$)

**Significant difference ($P \leq 0.001$)

NS = non-significant difference ($P \geq 0.05$)

Table 3: Comparison of Irisin level and biochemical parameters between males and females TC patients

Parameters	Males group ($n = 45$)	Females ($n = 45$)	*P* value
Irisin (ng/mL)	12.931 ± 1.93**	16.14 ± 2. 31	0.0001
HbA1c (%)	6.473 ± 1.51	5.997 ± 1.87	0.012
TSH (μ IU/L)	11.08 ± 2.11*	13.65 ± 1.94	0.01
T3 (ng/dL)	2.091 ± 0.32*	1.677 ± 0.51	0.01
T4 (ng/dL)	10.24 ± 2.82*	9.745 ± 1.46	0.113 (NS)
AST (mg/dL)	29.54 ± 6.61	29.18 ± 7.1	0.222 (NS)
ALT (mg/dL)	31.13 ± 5.11	30.97 ± 3.13	0.12 (NS)
T-Cholesterol (mg/dL)	156.11 ± 17.64*	133.15 ± 14.15	0.01
TAC (mg/dL)	121.07 ± 5.55	119.65 ± 7.47	0.223 (NS)
HDL-C (mg/dL)	40.26 ± 4.27	40.17 ± 5.13	0.232 (NS)
LDL-C (mg/dL)	102.92 ± 6.45	99.75 ± 8.51	0.212 (NS)
VLDL-C (mg/dL)	22.506 ± 1.33	21.96 ± 1.54	0.242 (NS)

TPOAb = antithyroid peroxidase antibody; HbA1c (%)= hemoglobin A1C; TSH = thyroid-stimulating hormone, T3 = triiodothyronine, T4 = thyroxine, AST= aspartate transaminase; ALT= alanine transaminase; T-Cholesterol= total cholesterol; TAG = triglycerides, HDL-C = highdensity lipoprotein cholesterol, LDL-C = lowdensity lipoprotein cholesterol

Values are mean ± standard deviation.

*Significant difference ($P \leq 0.05$)

**Significant difference ($P \leq 0.001$)

NS = Non-significant difference ($P \geq 0.05$)

Results

This study included 180 participants in total. Ninety individuals within the study group had TC. Table 1 displays the attributes of the research population.

There are no significant differences between healthy people and TC patients in terms of both age and body mass index [Table 1].

The values of Irisin and TPOAb in the TC group (14.797 ± 1.88 ng/mL), [(+) > 16], increased statistically significantly ($P < 0.001$) in comparison with the control group (4.428 ± 0.231 ng/mL), [(−) < 16], respectively [Table 2].

The values of HbA1c (%) in the TC group (6.874 ± 1.21) were statistically significant ($P < 0.05$) relative to the group of control (5.353 ± 0.97) [Table 2].

The values of TSH in the TC group (12.807 ± 2.24 μIU/mL) increased statistically significantly ($P < 0.05$) relative to the group of control (2.057 ± 0.532 μIU/mL).

The values of T3 and T4 in the TC group (0.944 ± 0.17 ng/mL) and (7.232 ± 1.94 ng/mL) decreased statistically significantly ($P < 0.05$) relative to the group of control (1.248 ± 0.14 ng/mL) and (8.514 ± 1.53 ng/mL), respectively [Table 2].

However, HDL-C and AST levels did not vary significantly within the groups [Table 2].

Table 4: Irisin level and biochemical parameters among age groups of TC patients

Parameters	(25–35 years) (n = 22)	(35-45 years) (n = 29)	>45 years (n = 39)
Irisin (ng/mL)	14.931 ± 1.77	13.104 ± 2.631	14.174 ± 1.79
HbA1c (%)	5.73 ± 1.21	5.87 ± 1.22	5.874 ± 2.52
TSH (μ IU/L)	12.68 ± 2.31	11.68 ± 1.64	12.84 ± 2.14
T3 (ng/dL)	1.041 ± 0.42	1.107 ± 0.31	0.994 ± 0.77
T4 (ng/dL)	7.28 ± 2.62	7.85 ± 1.46	7.192 ± 1.60
AST (mg/dL)	29.54 ± 4.63	29.45 ± 7.28	28.15 ± 7.33
ALT (mg/dL)	29.66 ± 3.11	30.68 ± 3.44	36.79 ± 3.4*
T-Cholesterol (mg/dL)	159.91 ± 13.22	163.15 ± 13.15	167.14 ± 15.53
TAC (mg/dL)	111.37 ± 8.05	119.82 ± 7.17	139.27 ± 7.55*
HDL-C (mg/dL)	41.82 ± 4.27	40.17 ± 5.13	39.63 ± 6.23
LDL-C (mg/dL)	102.13 ± 6.92	99.95 ± 8.73	100.47 ± 14.19
VLDL-C (mg/dL)	22.274 ± 1.61	23.964 ± 1.434	27.85 ± 1.51*

TPOAb = antithyroid peroxidase antibody; HbA1c (%)= hemoglobin A1C; TSH = thyroid-stimulating hormone, T3 = triiodothyronine, T4 = thyroxine, AST= aspartate transaminase; ALT= alanine transaminase; T-Cholesterol= total cholesterol; TAG = triglycerides, HDL-C = highdensity lipoprotein cholesterol, LDL-C = lowdensity lipoprotein cholesterol

Values are mean ± standard deviation

*Significant difference ($P \leq 0.05$)

Table 5: Assessment of TPOAb as a proportion by gender

Gender	16 IU/mL < frequency (%)	16 IU/mL > frequency (%)	Total frequency (%)
Male	9 (10)	36 (40)	45 (50)
Female	5 (5.6)	40 (44.44)	45 (50)
Total	14 (15.6)	76 (84.44)	90 (100)

Table 6: Relation between anti TPOAbs in percentage and aging

Age (year)	16 IU/mL < frequency (%)	16 IU/mL > frequency (%)	Total frequency (%)
25–35	3 (3.34)	19 (21.11)	22 (24.4)
435–45	4 (4.46)	25 (27.77)	29 (32.3)
≥45	7 (7.8)	32 (35.55)	39 (43.33)
Total	14 (15.6)	76 (84.44)	90 (100)

The values of ALT, T-cholesterol, TAC, LDL-C, and VLDL-C in the TC group increased statistically significantly ($P < 0.001$) in comparison with controls [Table 2].

The patients were divided into subgroups according to sex [Table 3] and age [Table 4].

For the purpose of studying the effect of aging on the variables studied in TC patients, the patients were divided into three age groups [Table 4].

According to Table 4's data, AST, triglyceride, and VLDL-C levels are significantly higher in the older age group than they are in the younger age groups.

In the current study, 76 patients (84.44%) had positive results from an anti-TPO antibody test; 40 of these patients (44.44%) were female and 36 patients (40%) were male [Table 5].

Table 6 displays that 19 (21.11%), 25 (27.77%), and 32 (35.55%) patients were positive anti-TPO antibody tests (>16 IU/mL) in 25–35, 35–45, and ≥45 years, respectively. These results reveal that the age group of ≥45 years (35.55%) had a higher percentage of positive anti-TPO antibody tests, with a mean age of 53 years and a standard deviation of 2.33.

To show the relationship between irisin and other biochemical parameters measured in the TC patients group, the linear correlation coefficient [r] was found, as shown in [Table 7]. The findings illustrate that there was a direct positive and significant correlation of irisin with each of the anti-TPOAbs, HbA1c, and T-cholesterol, while anti-TPOAbs showed positive correlations with (irisin, TSH, and T3). Moreover, it has also been observed that there was an inverse relation between both Irisin and anti-TPOAbs with ALT [Table 7].

Our results shown in Table 7 have indicated that the levels of irisin and TPOAb were increased (or higher than their levels in healthy subjects) in the TC patients.

Discussion

The thyroid gland produces TPOAb. Elevated TPOAb levels may contribute to the development and occurrence of TC.[18] TC may occur as a result of elevated TPOAb levels, according to earlier research.[19] TPOAb(+) in this study exhibits noticeably higher TSH values than TPOAb(−); yet, free T3 and free T4 values were virtually the same in both of them (TPOAb (+) and (−)) [Table 2].

According to a prior study, TPOAb stimulates T-cell responses to thyroid peroxidase and causes proinflammatory cytokines to be released from phagocytic cells.[9]

TPOAb inhibits thyroid peroxidase, which means that latent thyroid damage caused by TPOAb may lessen the thyroid's ability to produce thyroid hormone, which raises serum TSH.[20] It is still unclear how TSH and thyroid hormones—triiodothyronine (T3) and thyroxine (T4)—affect the development of TC in humans. The primary regulator of thyroid function and growth factor for thyroid cells is TSH. It regulates the mechanisms that result in elevated secretion and synthesis of thyroid hormones.[21] A key factor in the aggressiveness and onset of TC is TSH.[22] The present research revealed that TC patients had considerably higher serum TSH levels than the control group. This is consistent with[23] who concluded that thyroid hormone levels in the blood inversely control the pituitary gland's release of TSH via a negative feedback loop. In a mouse model, high TSH levels have

Table 7: Linear correlation of Irisin and TPOAb levels with another parameters among thyroid cancer patients

Biochemical parameters	Variables			
	Irisin		Anti-TPOAb	
	r value	*P* value	*r* value	*P* value
Irisin (ng/mL)			0.763*	0.001
TPOAb (IU/L)	0.763*	0.001		
HbA1c (%)	0.512**	0.001	0.425	0.192
TSH (μ IU/L)	0.437	0.002	0.524*	0.015
T3 (ng/dL)	0.452	0.198	0.537**	0.001
T4 (ng/dL)	0.337	0.172	0.251	0.301
AST (mg/dL)	0.434	0.193	0.447	0.407
ALT (mg/dL)	−0.538*	0.015	−0.540*	0.022
T-Cholesterol (mg/dL)	0.985**	0.001	0.425	0.161
TAC (mg/dL)	0.436	0.112	0.363	0.037
HDL-C (mg/dL)	0.467	0.039	0.267	0.041
LDL-C (mg/dL)	0.165	0.710	0.458	0.123
VLDL-C (mg/dL)	0.467	0.213	0.357	0.041

*Significant difference ($P \leq 0.05$)

**Significant difference ($P \leq 0.01$)

been linked to TC pathogenesis.[24] This supports our results documented in Table 2. TC patients are currently advised to have their TSH suppressed, since this has been found to improve patient survival.[25] However, the results of epidemiological analyses relating thyroid hormones and TSH to the incidence of TC have been conflicting.[26] Most studies found that raised TSH levels were linked to an increased risk of TC.[26,27] Conclusive evidence has also not been found on the relationship between thyroid hormones and TC risk.[26,28,29] A couple of studies discovered a link between a higher risk of TC and decreased thyroid hormone levels.[30,31] This supports our results documented in Table 2. In this investigation, individuals with TC had considerably higher serum irisin concentrations than controls. It was found that thyroid hormone might trigger white fat to turn brown through thyroid hormone receptors (TRs). This browning was then accompanied by an increase in (UCP-1). Irisin significantly affects metabolism by causing subcutaneous white adipocytes to brown through increased UCP-1 expression, which raises oxygen consumption and thermogenesis.[32] As a result, irisin levels vary depending on the thyrometabolic state and the interaction between irisin and thyroid hormones.

Researchers found that circulating ALT levels were significantly higher ($P \leq 0.05$) in TC participants than in controls during the current study. This result further corroborates a study by Zhang *et al.*[33] Furthermore, the current study employed serum ALT and AST values to evaluate liver function. Thyroid hormones play an important role in liver physiology.[34]

Many different kinds of interactions exist between thyroid hormones and the liver to preserve homeostasis; the thyroid gland alone produces both T4 and T3, while the thyroid gland as well as other tissues produce T4.[35,36] Serum proteins, which are mostly made in the liver, bind to more than 99% of the T4 and T3 in the blood. Thyroid hormone levels must be sufficient for the liver to do its metabolic tasks as best it can. This means that low thyroid hormones make the liver not work fully efficiently. The combined effects of thyrotoxicosis and liver illness may cause hepatic dysfunction in those who already have liver disease.[37]

This study found a negative correlation between irisin and ALT and a positive correlation between TPOAb, irisin, HbA1C, and total cholesterol. TPOAb, T3, and TSH showed a positive correlation. However, there was a statistically significant connection between serum irisin and total cholesterol.

In the current study, 76 patients (84.44%) had positive anti-TPO antibody tests; of these, 40 patients (44.44%) were females and 36 patients (40%) were men [Table 5]. Also, irisin levels were higher in females compared to males [Table 3]. This is consistent with a previous study that showed TSH levels over the normal range were linked to a higher risk of TC in men but not in women (OR = 1.96, 95% CI: 1.04, 3.66).[38]

Similarly, the present study observed that the age group of >45 years (35.55%) had a higher percentage of positive anti-TPO antibody tests, with a mean age of 53 years and a standard deviation of 2.33 [Table 6]. The majority of cases in the Thomas Cyriac *et al*. study with anti-TPO antibody positives were in the age range of 31–40. The study's patient population was 35.4 years old on average.[39]

Our research demonstrated a correlation between TC and increased HbA1c. Our study's results were consistent with those of other research.[40] According to a prior study,

patients with differentiated thyroid carcinomas had a higher prevalence of insulin resistance.[41] TC patients have higher levels of insulin resistance, according to a Sahin *et al*. study.[40] Additionally, Bae *et al*. showed that the development of TC in Korean women may be linked to hyperinsulinemia and/or insulin resistance.[42] According to He *et al*.,[43] blood irisin levels were greater in T2DM patients and were correlated with HbA1c levels. Our results were supported by the investigation's outcomes.

As illustrated [Table 2], HbA1c was increased in TC patients as compared to control. This study's increased HbA1c values suggest that people with TC may have trouble controlling their blood sugar. Additionally, this investigation confirmed[44] that there was a significant increase in HbA1c values between TC patients and controls. Insulin antagonists, such as thyroid hormones, have an indirect effect on insulin activity. In line with earlier research, lipid profiles were found to influence irisin secretion.[45]

This study found that the TC group had higher TG levels. This could be because the liver produces more triglyceride-rich VLDL at a faster rate.[46] Additionally, the activation of lipoprotein lipase, which extracts TG from VLDL, raises TG levels.[47]

The liver's TG-rich lipases and lipoproteins are catabolized by lipoprotein lipase, which is increased by thyroid hormones. Increased blood levels of triglycerides lead to a reduction in the retention of free fatty acids (FFA) and an increase in FFA flow from adipose tissue to other tissues, such as the liver, where they boost triglyceride synthesis and hepatic glucose production (vicious cycle),[46] HMG-CoA reductase is also in charge of the restricted stage of blood cholesterol production.[48] Consequently, dyslipidemia increased TG concentrations may be seen in hypothyroidism.[49] The TC group has higher total cholesterol and TG. The results of this study were consistent with those of,[50] who found that individuals with hypothyroidism had TG values that were considerably greater than those of the group with normal thyroid function.

As far as current research indicates, this is the first study that suggests irisin and TPOAb levels may function as TC biomarkers, determining factors in the relationship between TC progress status and these parameter concentrations. This investigation also establishes the link between TPOAb and irisin for the first time.

In Conclusion, the blood TPOAb and irisin levels in the TC patient group were higher than in the control group. Irisin is crucial for the diagnosis, management, and survival of TC, and irisin may be a TC protective factor.

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Nil.

Conflict of interest

The authors declare that there is no competing interest.

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