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Study on the link of autoimmune hypothyroidism with biochemical parameters and lipid profile in patients with autoimmune thyroid disease

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ABSTRACT

The most prevalent conditions affecting thyroid function were found to be autoimmune thyroidism, like Graves' disease (GD) and Hashimoto's disease (HT). The present study aimed to evaluate biochemical parameters and lipid profile and their correlation with TSH, FT3, and FT4 in patients with HT. The current study recruited 100 individuals (50 HT patients and 50 healthy individuals) aged 18-50 years old during the period from August 2023 to February 2024. Serum levels of TSH, FT3, and FT4 were measured. Moreover, the levels of total cholesterol (TC), triglycerides (TG), high-density lipoprotein (HDL), low-density lipoproteins (LDL), glucose, alanine aminotransferase (ALT), aspartate aminotransferase (AST), urea, and creatinine were evaluated. Additionally, the correlation of TSH, FT3, and FT4 with lipid profile, liver enzymes, glucose, urea, and creatinine was examined. The present data found that FT3 and FT4 levels were significantly elevated in Hashimoto's illness patients; however, the levels of TSH were greatly reduced in patients with Hashimoto's illness. Moreover, the statistical analysis showed no association between biochemical parameters and lipid profile in TSH, FT3, and FT4. Thus, hypothyroidism could have an indirect effect on biochemical parameters and lipid profile via an indirect path.

INTRODUCTION

Healthy growth and the smooth functioning of physiological systems depend on thyroid hormones, and the hypothalamus-pituitary-thyroid (HPT) axis mediates feedback processes that regulate thyroid hormone synthesis [1,2]. Hypothalamic thyrotropin-releasing hormone (TRH) is produced in response to decreased concentrations of thyroid hormone, thyroxine (T4), and triiodothyronine (T3). In turn, it causes the anterior pituitary to release more thyroid-stimulating hormone (TSH). TSH causes thyrocytes to produce more thyroid hormones [3]. Only a very small percentage of thyroid hormones (FT4 and FT3) remain in their free (unbound) form; the majority, more than 99.7%, are bound to plasma proteins. In their unbound state, thyroid hormones exhibit biological activity [4]. In iodine-rich regions, autoimmune disorders account for the majority of thyroid problems. Between 1 and 2% of people experience spontaneous hypothyroidism, which is ten times more frequent in women than in men and more common in older women. These conditions may include Hashimoto's thyroiditis (HT), primary atrophic hypothyroidism, and thyrotoxicosis caused by Graves' disease. Moreover, HT can also result in complicated diseases such as polycystic ovary syndrome [5]. Autoimmune thyroid disease (AITD) is more common in women of all ages. It could show up like this or be linked to a family history of autoimmune polyendocrinopathies or autoimmune thyroid disease [6].

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The incidence proportion of HT in females is about 8:1. However, based on test results of thyroid-specific autoantibodies in females, it seems that approximately 10% of the population has HT. As part of the pathophysiology of HT, dendritic cells expose T cells to thyroid antigenic molecules as foreign antigens, which leads to T cell proliferation and differentiation into thyroid-specific T cells. These T cells subsequently mediate thyroid infiltration, cytotoxicity, and antibody formation [7, 8]. HT exacerbates oxidative stress, which causes sterile inflammation to have its harmful consequences. It impairs the body's ability to control the inflammatory response, making it more vulnerable to an overabundance of inflammation, which can further result in sepsis [9, 10]. From mild dyspnea to overt respiratory failure, HT can cause a wide range of respiratory complications. This may result in systemic neutrophilia, which in turn causes lung inflammation [11, 12]. The preservation of metabolic homeostasis throughout life depends on thyroid hormone (TH). It is well known that the thyroid and liver collaborate closely, and that the TH is essential for the metabolism of glucose, fatty acid oxidation, lipogenesis, and cholesterol [13]. Because hypothyroidism and hyperthyroidism are less prevalent in the general population, researchers' attention has shifted more and more to the connection between thyroid hormones and lipid profiles in the euthyroid population. Lipid profiles in the body are linked to several hormones, such as TSH, free triiodothyronine (FT3), free thyroxine (FT4), and others in the euthyroid population, regardless of gender [14]. It is challenging to rule out the potential that FT4, FT3, and TSH are related in their relationship to lipid profile because thyroid hormones were previously thought to modulate TSH's effects on lipid profile [15].

Therefore, the goal of the current study was to look into how patients with AITD changed their renal parameters (urea, creatinine), liver enzymes, and glucose-lipid metabolism. Additionally, these characteristics and thyroid levels are correlated, which is a critical indicator for diagnosing autoimmune hypothyroidism.

MATERIALS AND METHODS

Study subject

From August 2022 to June 2023, 100 participants (all female) between the ages of 15 and 50 were recruited for the current study. The participants were split into two groups: 50 patients with symptoms believed to be associated with Hashimoto thyroiditis (HT) who visited the specialized diabetes and endocrine gland disease center in Amara City, and 50 people of similar age who seemed to be in reasonably good health. To find out if they had autoimmune thyroiditis, serologic testing was performed for each patient. Each study participant read and signed the patient informed consent form, and the Amara Medical Institute's Committee on Scientific Research Ethics authorized the study. Women with hematologic abnormalities, cancer, current infections, or pregnancy were not included in the study. The Research Ethics Committee of Southern Technical University has approved the present study with certificate No. 530-SOU-TE.

Sample collection

Venous blood samples were collected from both patients with HT and healthy individuals following an overnight fast and transferred into a 5 ml CAT serum Separator clot activator tube (Greiner Bio-One, Kremsmünster, Austria). The serum was used to measure liver enzymes (AST, ALT), urea, creatinine, lipid profiles, FBS, and thyroid hormones (FT3, FT4, and TSH).

Measurement of thyroid hormone concentrations

Using the electrochemiluminescence immunoassay approach, blood levels of TSH, FT3, and FT4 were determined on the same day as blood collection (Cobas, comp. Penzberg, Germany), and in compliance with the guidelines provided by the manufacturer. Briefly, samples, controls, and calibrators were brought to 20-25 °C before the performance of the measurement. Samples were centrifuged and analyzed by Cobas immunoassay analyzers (Roche, Basel, Switzerland), and the supernatants were analyzed using electrochemiluminescence immunoassay.

Biochemical measurement

Lipid profile estimation (TC, TG, HDL, and LDL) was measured in both patients and controls using a spectrophotometer (BIOLABO, Maizy, France) and in accordance with the guidelines provided by the manufacturer. Briefly, blood samples were centrifuged, and serum was collected. Levels of TC, TG, HDL, and LDL were measured spectrophotometrically in patients' and control samples. Moreover, liver enzymes were also measured using the AST and ALT activity Assay Kit (Abcam, Waltham, USA). Briefly, serum samples were diluted in the assay buffer, and 50µl of the samples were added into each well. Then, 100µl of assay reagent was added to each well and mixed well. Absorbance of samples was incubated for 60 min at 37^{o}c in a dark place. After that, samples were read at a wavelength of 450 nm. In addition, measurement of urea was performed using an assay urea kit (BioSystems S.A., Barcelona, Spain). Briefly, 10µl of samples were added, and an equal volume of reagent A and B was added to the samples, standard, and blank. Samples were mixed well and incubated at 37^{o}c for 5 min. Sample absorbance was measured at 600nm. For creatinine measurement, the Assay Atlas Creatinine kit (Blankenfelde-Mahlow, Berlin, Germany) was used. Briefly, 100 ml of samples and standards were added, and an equal volume of reagent R1 and R2 was added to the samples, standard, and blank. Samples were mixed well and incubated at 37^{o}c. Sample absorbance was measured at 490nm. Moreover, fasting blood sugar (FBS) was also measured. The kinetic FBS monitoring system (BG-710) (Kinetik, RH1 5DZ, Redhill, England) was used to measure the FBS in patients and control groups.

Statistical analysis

Data from the current study were analyzed using IBM Co. USA's SPSS software package, version 23. The significance of the difference in means was examined using independent t-tests. In order to determine the correlation between thyroid hormones and biochemical parameters. A p-value less than 0.05 is considered statistically significant. The Pearson's test was used to perform parametric correlations.

RESULTS

Estimation of serum TSH, FT3, and FT4 in HT patients

To evaluate the HT patients, the levels of serum FT3, FT4, and TSH were measured. The present results found that the levels of TSH were greatly elevated in patients with HT as compared to the control group (Figure 1A). However, the present data analysis found that the levels of FT3 and FT4 were greatly decreased (P value < 0.05) in HT patients compared to control groups (Figure 1B and C).

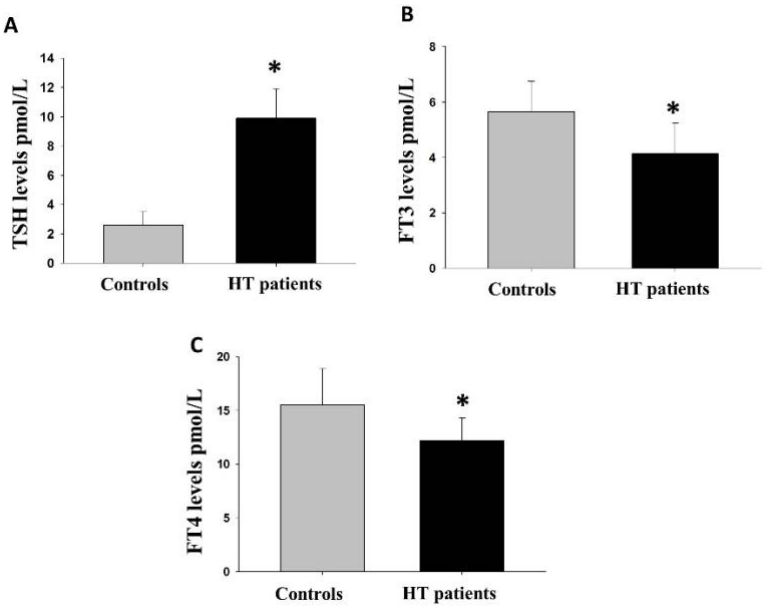

Figure 1. The levels of hormones (TSH, FT3, and FT4) in HT patients and the control group. Serum levels of A) TSH, B) FT3, and C) FT4. The black box represents HT patients. The grey box represents a healthy control group. Data introduced as means ± SEM and n=100. *P <0.05 versus control.

Estimation of lipid profile in HT patients

Next, the levels of lipid profile (TC, TG, HDL, and LDL) were evaluated in HT patients and the control group (Figure 2). The results showed that the levels of TC, TG, and LDL were substantially increased in patients with HT at p value < 0.05, as compared with the control group (Figure 2A, B, and D). However, the present findings showed that the levels of HDL were significantly reduced in patients with HT compared to the control group (Figure 2C).

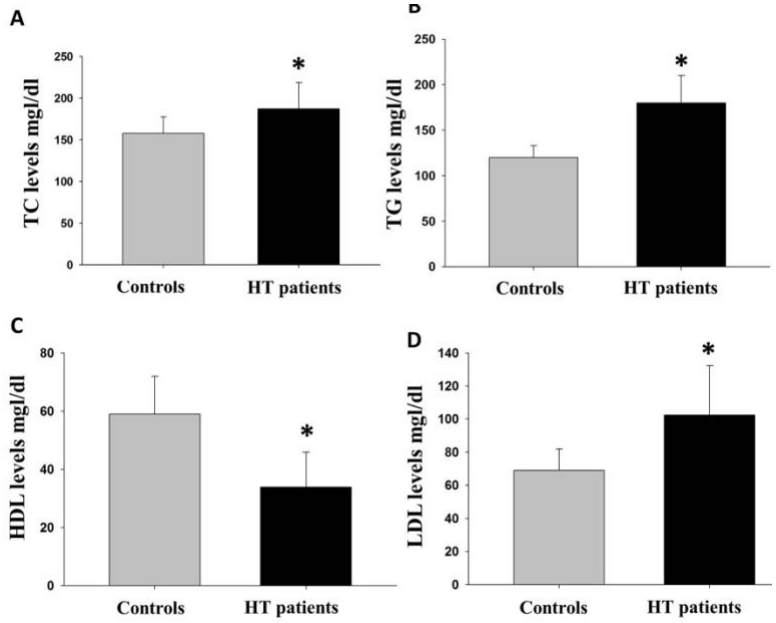

Figure 2. The levels of lipid profile (TC, TG, HDL, and LDL) in HT patients and the control group. Serum levels of A) TC, B) TG, C) HDL, and D) LDL. The black box represents HT patients. The grey box represents a healthy control group. Data introduced as means ± SEM and n=100. *P <0.05 versus control.

Estimation of liver enzyme levels in HT patients

It was important to study the effect of hypothyroidism on liver function. Therefore, the levels of AST and ALT were measured (Figure 3). The present data showed that the levels of AST and ALT were significantly increased (p value < 0.05) in the patients' group as compared to the control group (Figure 3A and B).

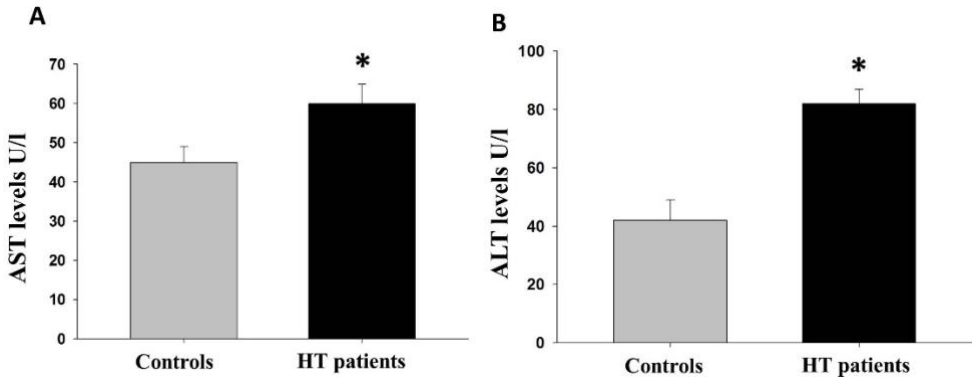

Figure 3. The levels of AST and ALT in the HT patients and the control group. Serum levels of A) AST and B) ALT. The black box represents HT patients. The grey box represents a healthy control group. Data introduced as means ± SEM and n=100. *P <0.05 versus control.

Estimation of FBS, urea, and creatinine levels in HT patients

Next, the levels of urea, creatinine, and FBS were examined in the HT patients (Figure 4). The present results found that the levels of urea and creatinine recorded a great increase in the patient group compared to the control group (Figure 4A and B). In contrast, the levels of FBS dramatically decreased (p value < 0.05) in the patient group compared to the control group (Figure 4C).

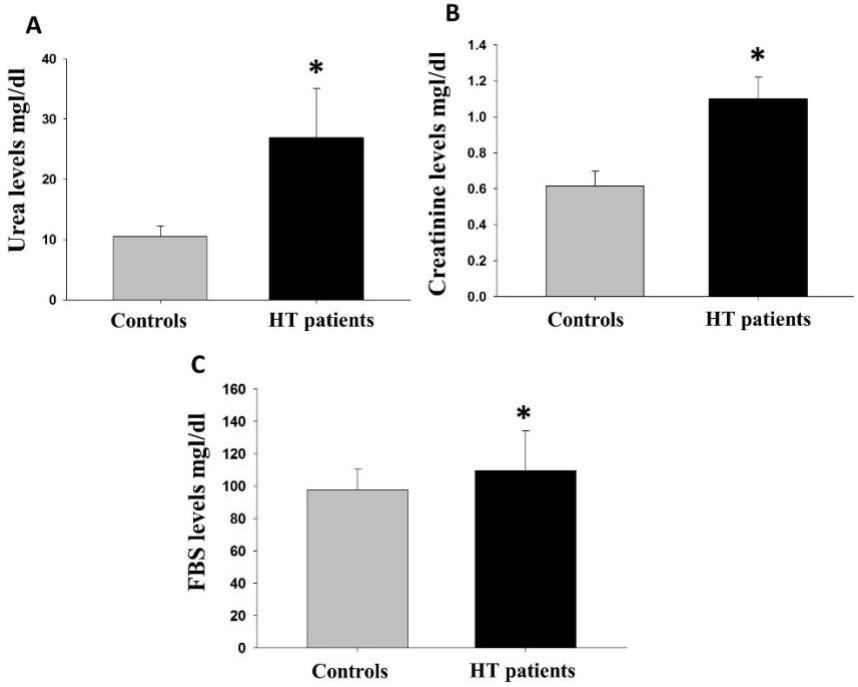

Figure 4. The levels of urea, creatinine, and FBS in the HT patients and the control group. Serum levels of A) urea, B) creatinine, and C) FBS. The black box represents HT patients. The grey box represents a healthy control group. Data introduced as means ± SEM and n=100. *P <0.05 versus control.

Association of TSH, FT3, and FT4 with lipid profile in HT patients

Next, the correlation of HT with lipid profile (TC, TG, HDL, and LDL) was sought in patients (Table 1). It was important to examine the correlation of TSH, FT3, and FT4 with lipid profile (TC, TG, HDL, LDL). The statistical analysis showed no correlation between TSH, FT3, and FT4 with lipid profile (TC, TG, HDL, LDL) at a p value of more than 0.05 (Table 1). TSH, FT3, and FT4 could affect the lipid profile indirectly.

Table 1. Association of TSH, FT3, and FT4 with lipid profile in HT patients.

Hormones	TC		TG		HDL		LDL	
	r	P	r	P	r	P	r	P
TSH	0.050	0.755	0.015	0.927	0.149	0.352	-0.276	0.081
FT3	0.111	0.491	-0. 125	0.436	-0.018	0.911	0.133	0.407
FT4	-0.032	0.842	-0.028	0.860	-0.049	0.761	-0.002	0.990

(TSH) thyroid stimulating hormone; (FT3) free triiodothyronine; (FT4) free thyroxine; total cholesterol (TC); (TG) triglycerides; (HDL) high-density lipoprotein; (LDL) low-density lipoproteins

Association of TSH, FT3, and FT4 with FBS, urea, and creatinine in HT patients

Next, the correlation between TSH, FT3, and FT4 with FBS, urea, and creatinine was also examined in HT patients (Table 2). The present findings did not find a correlation (p value more than 0.05) between TSH, FT3, and FT4 with FBS, urea, and creatinine (Table 2).

Table 2. Association of TSH, FT3, and FT4 with AST and ALT in HT patients.

Hormones	AST (U/L)		ALT (U/L)	
	r	P	r	P
TSH	0.226	0.155	0.169	0.291
FT3	-0.276	0.080	-0.190	0.243
FT4	-0.059	0.715	0.022	0.993

(TSH) thyroid stimulating hormone; (FT3) free triiodothyronine; (FT4); (ALT) alanine aminotransferase; (AST) aminotransferase

Association of TSH, FT3, and FT4 with AST and ALT in HT patients

It was also important to examine the link of TSH, FT3, and FT4 with AST and ALT in HT patients. Notably, the present data did not find a correlation between TSH, FT3, and FT4 with AST and ALT, as shown in Table 3.

Table 3. Association of TSH, FT3, and FT4 with FBS, urea, and creatinine in HT patients.

Hormones	FBS		Urea		Creatinin	
	r	P	r	P	r	P
TSH	-0.102	0.527	0.012	0.940	-0.073	0.651
FT3	0.052	0.745	-0.029	0.858	-0.063	0.695
FT4	0.231	0.145	-0.135	0.399	0.008	0.958

(TSH) thyroid stimulating hormone; (FT3) free triiodothyronine; (FT4); (FBS) fasting blood sugar

DISCUSSION

The present study found that autoimmune thyroidism greatly increased the levels of TSH, FT3, and FT4 in HT patients. The present findings also demonstrated that thyroidism affects the levels of lipid profile. Moreover, liver and kidney functions have also been affected by thyroidism, but indirectly. Therefore, further studies are needed to investigate the mechanism by which autoimmune thyroidism affects body organs.

Since thyroid hormones regulate the rate of tissue metabolism, changes in their actions will affect the levels of several organs and enzymes. These results can be explained by the understanding that thyroid hormones are essential for the growth, development, and function of organs, that any condition affecting the thyroid gland will affect the hormone levels, leading to an imbalance in the metabolism of various organs, and that hypothyroidism is linked to a number of metabolic problems [16, 17]. This study demonstrated that thyroid problems had significant impacts on the metabolism of various body cells, which were manifested in varying degrees by raised enzyme levels and serum lipid profiles. Women are far more likely to have thyroid problems. Subfertility, irregular menstruation, ovulatory dysfunction, and increased risk of recurrent miscarriages have all been associated with HT [18]. Hashimoto's thyroiditis has become more prevalent in recent years, likely due to the use of more accurate diagnostic tests; the prevalence of the condition depends on a number of factors, including age, gender, the environmental conditions of the study area, and the criteria used to diagnose the disorder [19]. The present results showed that Hashimoto's patients had significantly higher serum levels of TSH than typical, healthy controls. Patients also have increased thyroid hormones (FT3 and FT4), however, not to a substantial degree. These results may indicate the onset of subclinical hypothyroidism, which is characterized by elevated TSH and normal quantities of the thyroid hormones FT3 and FT4. proving that subclinical hypothyroidism was more common in those with HT. Thyroid function (FT4, FT3, TSH) was evidently diminished in children with autoimmune thyroid disease, according to a recent study [20]. The results of the present study showed that serum FT3 and FT4 levels were lower in hypothyroid people, which is consistent with the previous findings. In those with normal blood T4 levels, a slight rise in TSH is indicative of hypothyroidism, the initial stage of severe hypothyroidism. A small amount of research indicates that oxidative stress is increased in subclinical hypothyroidism (SCH). TSH levels are somewhat elevated in SCH, while the levels of the other thyroid hormones are normal. In this clinical scenario, the levels of blood TSH are elevated, but FT4 readings are normal. Between 4.3% and 9% of the general population is predicted to be impacted by SCH. Older adults and women are more prone to have SCH [21]. Hypothyroid patients may have elevated blood LDL levels due to a decrease in LDL action and the quantity of receptors in the liver cells, which could damage the in vivo degradative pathway of the LDL-dependent receptor. Previous studies have shown that a diet high in fat can lower the serum FT4 level in the offspring of primates, indicating that elevated TG levels are both the "cause" and the "result" of hypothyroidism in patients [22]. Thyroid hormones affect the activity of many important enzymes involved in lipid metabolism, which substantially impairs the composition and transport of lipoproteins in thyroid diseases. Clearly, hypothyroidism causes hypercholesterolemia and elevated LDL levels. Hypothyroidism induces hyperlipidemia due to decreased LDL receptor concentration and decreased liver LDL clearance. Variations in the actions of hepatic lipase (HL) and the ester of cholesterol transfer protein (CETP) are to blame for this. The microenvironment is therefore dominated by atherogenic lipids [23]. The thyroid hormone increases the amount of cholesterol produced by the liver. Bile salts are more easily converted from it. Adipocytes may become more susceptible to the lipolytic activity of adrenaline due to

the thyroid hormone T3, which improves the liver's ability to transport fatty acids and, consequently, produce triacylglycerol [24]. We examined alterations in the metabolism of lipids and glucose in the current study.

In fact, the hypothyroidism group's serum levels of TG, LDL-C, FT3, and FT4 were substantially lower than the normal thyroid group. In contrast, TSH was significantly elevated. But there was no statistically significant difference in the fasting group and 2-hour postprandial blood glucose levels ($P > 0.05$). Blood TC, TG, and LDL-C levels were clearly higher in the hypothyroidism group than in the normal thyroid group, with statistically significant differences. The present study found a negative connection between the blood TSH level and TC, TG, and LDL-C. However, the levels of FT3 and FT4 were linked with all of these variables. When comparing hypothyroidism patients to healthy controls, the mean levels of all lipids-aside from HDL, rose noticeably. It has been found that elevated levels of alkaline phosphatase and alanine aminotransferase were associated with hyperthyroidism, while elevated levels of aspartate aminotransferase were associated with hypothyroidism [25].

Previous studies showed that the serum ALP, ALT, and AST levels of hypothyroidism patients were substantially higher (p 0.001) than those of the control group. It has been demonstrated that there were substantial differences between patients with HT and without HT in terms of BMI, waist circumference, systolic blood pressure, diastolic blood pressure, TG, HDL, UA, blood glucose, ALT, AST, and TSH [26]. FT4 and TG levels in IH patients were found to be negatively correlated [27].

The frequency of HT issues may be decreased by using the lipid profile and liver enzymes as an additional diagnostic tool for early HT detection. However, there were limitations for the present study. The mechanisms of HT affecting the lipid profile, liver, and kidney functions are lacking in the present study. Therefore, future studies are required to investigate the exact mechanism of HT in affecting the lipid profile, liver, and kidney functions.

CONCLUSIONS

It is concluded that patients with hypothyroidism had substantial levels of TSH, FT3, and FT4 that were considerably greater than those in the control group. Additionally, hypothyroidism greatly affects the levels of TC, TG, and LDL. Moreover, the present findings could also show that hypothyroidism caused a clear-cut increase in the levels of liver enzymes. Furthermore, the present results also found that kidney functions were affected by hypothyroidism. The present results did not find an association of hypothyroidism with lipid profile, liver, and kidney functions. Hypothyroidism could affect lipid profiles, both liver and kidney activity, via an indirect path. Therefore, future studies are required to investigate the exact mechanism by which hypothyroidism can affect lipid profiles, liver, and kidney functions.

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AUTHOR CONTRIBUTIONS

NAH, RM, and AAS participated equally in the interpretation of the results and manuscript writing. The experiments of the present study were carried out by NAH. The statistical analysis and display of the data were completed by NAH, RM, and AAS. Each author provided their consent after reading the finished manuscript.

CONFLICTS OF INTEREST

There is no conflict of interest among the authors.

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