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A comparative study on serum lipid levels in patients with thyroid dysfunction: a single-center experience in Ethiopia

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Abstract

Background Thyroid diseases are the most common endocrine disorders worldwide. Thyroid hormones are essential for lipid synthesis, metabolism, and mobilization. Lipid levels in the blood may be altered when there is thyroid dysfunction. Lipid changes are linked to hyperthyroidism and primarily involve total and low-density lipoprotein cholesterol. The serum lipid profile is negatively impacted by hypothyroidism, which may increase the risk of atherosclerotic disease development. Thus, hypothyroidism constitutes a significant cause of secondary dyslipidemia. However, the results obtained from different studies are inconsistent, and there are few data regarding lipid profiles in thyroid dysfunction patients in the study area. Therefore, this study aimed to assess the lipid profile of thyroid dysfunction patients at Wolaita Sodo University Comprehensive Specialized Hospital from May 1 to June 15, 2021.

Methods A comparative cross-sectional study was conducted involving 200 participants (100 thyroid dysfunction patients and 100 age- and sex-matched controls). Socio-demographic and related data were collected from the study participants via a pretested structured questionnaire through face-to-face interviews. Independent sample T tests and Mann–Whitney U tests were used for data analysis. $P < 0.05$ indicated statistical significance.

Results Out of 200 study participants 40 (20%) hyperthyroid, 60 (30%) hypothyroidism, and 100 (50%) controls. In individuals with hyperthyroidism, the levels of triglycerides, total cholesterol, and low-density lipoprotein cholesterol were significantly lower than those in the control group. Compared to the control group, the hypothyroidism patients had significantly higher levels of triglycerides, low-density lipoprotein cholesterol, total cholesterol, and high-density lipoprotein cholesterol.

Conclusion The lipid profile can change significantly as a result of thyroid dysfunction. Biochemical screening of lipid profiles is essential for improving patients with thyroid dysfunction with dyslipidemia.

Keywords Dyslipidemia, Ethiopia, Hyperthyroidism, Hypothyroidism, Hyperlipidemia

Introduction

Thyroid hormones stimulate many aspects of carbohydrate metabolism, including quick uptake of glucose by cells, enhanced glycolysis, enhanced gluconeogenesis, and an accelerated rate of absorption from the gastrointestinal tract. Moreover, they enhance insulin production and consequently carbohydrate metabolism [1]. They also promote fat metabolism and stimulate protein synthesis. They cause quick mobilization of fat from fat tissues, increase plasma fatty acid concentrations and

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accelerate the oxidation of free fatty acids by cells [1, 2]. Additionally, they regulate important processes involved in thermogenesis and energy consumption [3, 4].

The majority of thyroid diseases are caused by abnormalities in the thyroid hormone production rate. Hyperthyroidism is a term for overactive tissue within the thyroid gland, resulting in overproduction and thus an excess of circulating free thyroid hormones: thyroxin (T4), triiodothyronine (T3), or both, whereas hypothyroidism is caused by insufficient production [5, 6]. Most hyperthyroid patients exhibit heat intolerance, tremor, palpitations, anxiety, weight loss despite a normal or increased appetite, increased frequency of bowel movements, and shortness of breath [4, 7, 8]. Common clinical manifestation of hypothyroidism include, fatigue, weight gain, trouble tolerating cold, joint and muscle pain, dry skin or dry, thinning hair, heavy or irregular menstrual periods or fertility problems, slowed heart rate, depression [9, 10].

Triiodothyronine (T3) and thyroxin (T4) are thyroid hormones that affect a variety of metabolic parameters. These hormones have a major impact on lipoprotein metabolism and a few cardiovascular disease (CVD) risk factors, thus influencing overall CDV risk (https://www.austincc.edu/ddingley/MLAB1331/LabManual/LabManual.htm) [11, 12]. In fact, it has been shown that rising thyroid-stimulating hormone (TSH) levels cause a linear increase in triglyceride, total cholesterol, and low-density lipoprotein cholesterol (LDL-C) levels as well as a linear decrease in high-density lipoprotein cholesterol (HDL-C) levels, even within the normal range of TSH levels [12, 13].

The findings of patients with thyroid dysfunctions clearly demonstrated the connection between thyroid hormones and lipid metabolism. Patients with overt hyperthyroidism exhibit decreased lipid levels, whereas patients with overt hypothyroidism exhibit elevated TG and cholesterol levels. These findings indicate that in addition to thyroid hormones, thyroid-stimulating hormone (TSH) has independent effects on lipid metabolism within the subclinical hypo-/hyperthyroid range [12, 14].

The overall incidence of hyperthyroidism is estimated to be about 0.05% to 1.3% with a predominant number being subclinical, this figure rises to between 4 and 5% among older women [15, 16]. In addition to other problems, liver biochemical dysfunctions affect 15–79% of patients with untreated hyperthyroidism; some of these patients experience substantial liver failure and impaired synthetic function [15]. The composition and transport of lipoproteins are strongly disrupted in thyroid diseases. The magnitude of change in plasma lipids varies from patient to patient [17]. The extent of these changes depends both on the severity and duration of thyroid dysfunction [2].

Along with several other cardiovascular risk factors, lipids are significantly impacted by thyroid dysfunction [12]. Research conducted in various nations on the lipid profile parameters of people with thyroid impairment has produced inconsistent findings. Several studies have shown that there are statistically significant differences in total cholesterol, triglyceride, high-density lipoprotein cholesterol, low-density lipoprotein cholesterol, and very low-density lipoprotein cholesterol levels between hyperthyroid and hypothyroid patients and healthy controls [18–22]. However, another study showed that the levels of total cholesterol, triglycerides, and low-density lipoprotein cholesterol remained unchanged in hyperthyroid patients compared to those in the normal group [23]. Another study indicated that there was an increase in cholesterol and LDL-L levels in hyperthyroid patients compared with those in healthy controls [24]. There was no statistically significant difference observed between hypothyroid patients and controls in terms of lipid profile parameters, specifically cholesterol, triglycerides, HDL-cholesterol, and LDL-cholesterol [25]. Moreover, information on Ethiopia, particularly in the study area, is limited.

Results obtained from clinical chemistry analyses can reveal changes in lipid profiles, such as total cholesterol, triglyceride, high-density lipoprotein-cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), and very low-density lipoprotein-cholesterol (VLDL) levels [26–28]. Analysis of these parameters will definitely help in the early detection of lipid abnormalities in patients with hypothyroidism and hyperthyroidism, hence decreasing the associated mortality and morbidity rates. Therefore, this study aimed to compare the lipid profiles of dysfunction patients and controls at Wolaita Sodo University Comprehensive Specialized Hospital from May 1 to June 15, 2021.

Methods and materials

Study design, period and area

A hospital-based comparative cross-sectional study was conducted from May 1, 2021, to June 15, 2021, at the comprehensive specialized Hospital of Wolaita Sodo University from outpatient department. The hospital also serves outpatient services such as diabetic clinics, hypertension clinics, TB clinics, ART clinics, and maternity and pediatric facilities.

Inclusion criteria

Patients with TSH levels < 0.6 (mIU/L), T4 levels are (> 1.1 ng/dl) and FT3 levels > 3.07 (nmol/L) were included as hyperthyroidism. Additionally TSH levels (> 4.1

mIU/L) and FT4 levels (< 7.0 mcg/dl) were included in the hypothyroid patient group. Hospital staff members who were age and sex matched and had normal thyroid function were included as control participants.

Exclusion criteria

Study subjects and controls receiving medications known to alter lipid metabolism as well as those with neoplasms, kidney illness, liver disease, history of smokers, diabetes mellitus, or familial hypercholesterolemia were not allowed to participate in the study.

Study participants and controls with previous history of hyperthyroidism or hypothyroidism who are taking medicine were excluded from the study.

Data collection and laboratory methods

Socio-demographic, behavioral, and related data were collected through face-to-face interviews using a pretested structured questionnaire. Following the interview, a laboratory technician used a standard procedure to collect five millilitres of venous blood in ethylenediaminetetraacetic acid (EDTA) tubes, which were then carefully transferred into a clean tube. The blood was allowed to clot at room temperature and then centrifuged at 3000 rpm for 5 min. The serum was carefully separated from the red cells using a Pasteur pipette into a sample bottle and analyzed for free thyroid hormone and lipid levels as routine practice. Thyroid function tests were analyzed using the Cobas-e-411 electrochemiluminescence (ECL) analyzer. And the total cholesterol, HDL-C, triglycerides and lipoprotein A was estimated by COBAS INTEGRA 400 random access full automated auto analyzer and LDL-C concentration was calculated by Sampson equation. Triglycerides and total cholesterol was evaluated with enzymatic colorimetric method and HDL-C was analyzed by homogenous enzymatic colorimetric method**.**

Data quality assurance and management

Data quality was ensured by implementing all quality assurance programs. For accuracy and consistency, the English version of the questionnaire was translated into Wolaitisa, the native tongue, and then back into English. Again, 5% of the sample was pretested at the hospital before actual data collection, and training was given to the data collectors. The collected blood samples were kept cool in a humidified refrigerator until the time of measurement to prevent any impact from ambient temperature. During the sampling and experimentation processes, the manufacturer's instructions and normal operating procedures were closely adhered to. Background checks, automated clinical chemistry analyzers, and repeated analyses of randomly chosen samples were used to determine reproducibility; randomly chosen samples were confirmed using another clinical chemistry machine of a similar type; and hospital laboratories assessed device performance using whole-blood quality control as part of the testing procedure.

Statistical analysis and interpretation

The data were checked for completeness and consistency. Then, the data were imported into Epi-data version 3.1 (Epi-Data, Odense, Denmark) and exported into the Statistical Package for Social Science (SPSS) version 20 (SPSS, Chicago, USA) statistical software for Windows. Prior to conducting any appropriate statistical analysis, the collected data were tested for normality by the Shapiro–Wilk test. Lipid profiles were compared between dysfunction patients and controls using an independent-samples T -test for normally distributed data and the Mann–Whitney U test for non-normally distributed data. Additionally, independent-T test was used to compare lipid profiles between hyperthyroid and hypothyroid patients. The Pearson coefficient test was used to test the correlation between free thyroid hormone levels and lipid profiles. The level of statistical significance was defined as a p value ≤ 0.05 and a confidence interval of 95.

Results

Socio-demographic characteristics of the study participants

A total of 200 participants were included in the study; 60 patients had hypothyroidism (hypothyroid group), 40 patients had hyperthyroidism (hyperthyroid group), and 100 had a normal thyroid level (control group). In both the hypothyroid and hyperthyroid groups, more than 75% of the participants were female. More than 52.5% of the hyperthyroid patients were aged > 40 years. The mean ages of the hypothyroid and hyperthyroid patients were 30 ± 2.3 and 41 ± 5.2 years, respectively (Table 1).

Comparison of lipid profiles between the hyperthyroid patients and the control group

Statistically significant differences were observed in the median total cholesterol (TC) level ($P=0.001$) and mean ± SD of triglyceride level ($P=0.01$) between the hyperthyroid patients and the control group. Patients with hyperthyroidism had significantly lower mean values of low-density lipoprotein cholesterol ($P=0.039$) and median values of total cholesterol ($P=0.001$) than did the control individuals. Other lipid levels, such as VLDL-C, the LDL-C/HDL-C ratio, and HDL-C levels, did not differ substantially across the groups, as described in Table 2.

Table 1 Baseline characteristics of study participants

Variable	Thyroid status	
	Hyperthyroid	Hypothyroid
Gender		
Male	9(22.5%)	13(21.6%)
Female	31(77.5%)	47(78.33%)
Age		
< 20	4(10%)	5(8.3%)
20–40	15(37.5%)	25(41.7%)
41–60	21(52.5%)	30(50%)
Mean ± SD	41 ± 5.2	30 ± 2.3
Marital status		
Married	22(55%)	47(78.3%)
Singe	16(40%)	10(16.7%)
Other	2(5%)	3(5%)
Residence		
Urban	23(57.5%)	42(70%)
Rural	17(42.5%)	18(30%)
Body mass index	16 ± 1.6	19 ± 2.6

Other: widowed and divorced

Table 2 Comparison of lipid profile of the hyperthyroid patients with control group

Parameters	Hyperthyroid patients ($N=40$)	Control($n=100$)	*p*-value
TG(mg/dl)	84.07 ± 3.12	91.6 ± 9.14	0.01
TC (mg/dl)[α]	128.57 ± 13.54	149.76 ± 14.9	0.001
HDL-C (mg/dl)	44.02 ± 3.42	44.6 ± 4.3	0.21
LDL-C (mg/dl)	70.63 ± 13.67	81.6 ± 13.8	0.039
VLDL-C(mg/dl)[α]	17.14 ± 0.6	17.68 ± 0.93	0.4
LDL-C/HDL-C ratio	1.75 ± 0.4	2.62 ± 0.3	0.06

TG Triglycerides, *HDL* High density lipoprotein, *LDL* Low density lipoprotein. *TC* Total cholesterol, *α P*-value derived from Mann–Whitney U-test, * *p*-value < 0.05 is considered as statistically significant

Comparison of lipid profiles between hypothyroid patients and control individuals

The hypothyroid group's mean serum triglyceride level and median total serum cholesterol level were considerably greater than those of the control group. When comparing the hypothyroid group to the control group, statistically significant decreases were observed in the levels of very low density lipoprotein (VLDL-C) and high density lipoprotein (HDL-C) (Table 3).

Correlations between serum thyroid hormone levels and lipid profiles in patients with thyroid dysfunction

Table 4 shows the relationship between serum thyroid hormone levels and lipid levels in hyperthyroid and hypothyroid patients. The results in this table show that there were negative and significant correlations between TSH and TC ($r=-0.38$, $P=0.01$), between TSH and LDL-C ($r=.-0.32$, $P=0.004$), and between T4 and LDL-C ($r=-0.12$, $P=0.0001$) in hyperthyroid patients. as described in Table 4.

Table 3 Comparison of lipid profile of the hypothyroid patients with control group

Variable	Hypothyroid patients ($N=40$)	Control ($N=100$)	*p*-value
TG(mg/dl)	153.7 ± 29.01	91.6 ± 9.14	0.01
TC (mg/dl)[α]	258.53 ± 10.7	149.76 ± 14.9	0.01
HDL-C (mg/dl)	25.4 ± 3.47	44.6 ± 4.3	0.01
LDL-C (mg/dl)	203.81 ± 38.5	81.6 ± 13.8	0.01
VLDL-C (mg/dl)[α]	19.26 ± 2.02	17.68 ± 0.93	0.21
LDL-C/HDL-C ratio	8.04 ± 3.6	2.62 ± 0.3	0.001

TG Triglycerides, *HDL* High density lipoprotein, *LDL* Low density lipoprotein. *TC* Total cholesterol, *α P*-value derived from Mann–Whitney U-test, * *P*-value < 0.05 is considered as statistically significant

Discussion

Thyroid dysfunction is more common among endocrine illnesses. The majority of thyroid illnesses are caused by abnormalities in the thyroid hormone production rate [29]. This study was conducted in three groups of subjects, that is, in the normal control group, hypothyroid group, and hyperthyroid group, with a predominance of female patients (77.5% in the hyperthyroid group and 78.3% in the hypothyroid group).

The present study showed that the mean level of serum triglycerides in hypothyroid patients was significantly greater than that in healthy controls. These findings are similar to the results of other studies [30–32]. Lipoprotein lipase enzyme activity may have decreased in the hypothyroid group, which could account for their higher triglyceride levels [33]. Conversely, in research conducted in Libya [24], these differences may be the result of racial and sex differences [27].

The hypothyroid patient median total cholesterol level was considerably greater in this study than in the control group. This result aligns with research carried out across many regions of the world [34–37]. Hormone deficiency and changes in hepatic lipase and lipoprotein lipase enzyme activity could also be causes [33, 38]. The primary factor contributing to elevated total cholesterol in hypothyroid individuals is a reduction in the expression of the low-density lipoprotein receptor gene in fibroblasts, hepatocytes, and other organs [39]. The low-density lipoprotein receptor is a protein that sits on the surface of the liver, and its main purpose is to identify and absorb lipoproteins or specific

Table 4 Correlation between hypothyroidism, hyperthyroidism, and lipid profile in thyroid dysfunction patients

	Hyperthyroid patients		**Hypothyroid patients**	
	Total cholesterol		**Total cholesterol**	
	R	*p*-value	R	*P*-value
TSH	−0.38	0.01	0.11	0.02
T3	0.229	0.06	−0.082	0.051
T4	−0.31	0.91	0.024	0.43
	High density lipoprotein		**High density lipoprotein**	
	R	*P*-value	R	*P*-value
TSH	0.23	0.31	0.52	0.72
T3	0.074	0.42	0.412	0.63
T4	0.72	0.062	0.241	0.82
	Low density lipoprotein		**Low density lipoprotein**	
	R	*p*-value	R	*P*-value
TSH	−0.32	0.004	0.92	0.001
T3	0.31	0.37	0.41	0.46
T4	−0.12	0.0001	0.13	0.11
	Triglyceride		**Triglyceride**	
	R	*P*-value	R	*P*-value
TSH	0.21	0.42	0.076	0.39
T3	0.075	0.33	0.104	0.001
T4	0.312	0.04	0.52	0.06

p-value < 0.05 is considered as statistically significant

cholesterol particles and eliminate them from the bloodstream [40]. Thyroid hormones (T3 and T4) serve to increase the expression of these receptors [41]. However, the thyroid hormone level is decreased in patients with hypothyroidism.

In our study, the high-density lipoprotein cholesterol level was significantly lower in hypothyroid patients than in the controls. These findings are similar to those of a study conducted in India [32]. The mean LDL-C level in hypothyroid patients was significantly greater than that in control individuals. The increased levels of low-density lipoprotein cholesterol could be due to a decreased fractional clearance of low-density lipoprotein. Consequently, a decreased quantity of low-density lipoprotein cholesterol receptors in the liver may be the cause of this elevation [40]. These findings were in accordance with those of the study performed by Benny PD [32].

The findings of the current study showed a significant reduction in total cholesterol, triglycerides, and low-density lipoprotein cholesterol in hyperthyroid patients compared to those in the control group. This was in line with the results of other research projects carried out globally [42, 43]. The increased activity of the HMG-CoA reductase enzyme may be the source of the reduction, and elevated biliary excretion of cholesterol combined with higher receptor-mediated LDL particle catabolism is generally associated with elevated cholesterol levels [17].

In contrast, there were no significant differences in the total cholesterol triglyceride or low-density lipoprotein cholesterol levels between the hyperthyroid and control groups [23]. The reason might be the outcome of the treatment. According to several investigators, the lipid profile values of individuals with hyperthyroidism were found to be low and were adjusted to within normal ranges following treatment [44]. The levels of HDL-C and VLDL-C and the LDL-C/HDL-C ratio were not significantly different between the hyperthyroid group and the control group, which disagrees with the findings of another study [32]. These differences may be due to sample size variations [24].

In hyperthyroid patients, strong negative correlations were found between TSH and total cholesterol, between TSH and LDL-C, and between T3 and LDL-C with respect to the associations between lipid profile parameters and thyroid hormones. These findings disagree with those of a study conducted in Sudan [27]. Additionally, a positive and significant correlation was found between TSH and LDL-C and between TSH and TC in hypothyroid patients. These results align with those of another study carried out in Nepal, which demonstrated a positive connection between TSH and total cholesterol (TC) and between TSH and LDL hypothyroid individuals [45]. In the present study, there were no significant correlations between T3, TSH or T4 and HDL-C in either the

hyperthyroid or hypothyroid patients, in contrast to the findings of other studies [21, 27].

Conclusion and recommendation

This study showed that, compared with those in the control group, most lipid profile parameters in thyroid dysfunction patients (hyperthyroid and hypothyroid) were altered. The levels of the serum lipid profile parameters TC, TG, and LDL-C were significantly lower in the hyperthyroid patients. Finally, there was also a negative and significant correlation between the serum TSH concentration and the levels of several lipids (TC and TG) in hyperthyroid patients. Biochemical screening for thyroid dysfunction is critical in all dyslipidemia patients as well as in all patients with unexpected improvement or worsening of their lipid profile [23, 46]. Periodic medical check-ups of the lipid profile of thyroid dysfunction patients should be considered before the development of cardiac and other diseases.

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Authors' contributions
TEK is the principal investigator and is involved in the design of the study, data analysis, and interpretation of the findings, report writing, and manuscript preparation. GA, YS, and participated by advising and supervising the research process and gave constructive comments to increase the quality of the study, TEK and AT commented, edited, and approved the final manuscript of the research.

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Data availability
The data set supporting the conclusion of this article are incorporated in this manuscript.

Declarations

Ethics approval and consent to participate
Ethical clearance was obtained from the Ethical Review Committee of Wolaita Sodo University, College of Health Science and Medicine (reference number: CSHM/ER/01/21). Written informed consent was obtained from each study participant after the objective and procedures of the study were explained. Written consent to participate was obtained from the parents/guardians of the minors included in this study (minors are considered anyone under the age of 16). To ensure data confidentiality, study participants were identified by codes rather than names or other identifiers, and data collection was prevented from being accessed by unauthorized parties. Above all, the Committee on Publication Ethics and the Declaration of Helsinki ethical guidelines for medical research involving human beings were strictly followed during the entire study.

Consent for publication
Not applicable.

Competing interests
The authors declare no competing interests.

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