Evaluation of the lipid profile level in patients with hypothyroidism

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Abstract

The thyroid gland is considered one of the most important endocrine glands in the human body. It controls blood pressure, heart rate, temperature, weight, and other vital functions. The aim of this study is to evaluate the level of lipid in patients with hypothyroidism. The study was included 60 female patients with hypothyroidism and 40 healthy women, who were considered as control group, in the age group range from 22 to 44 years. The results of this study showed that subjects with hypothyroidism had significant increase in the level of lipids, as the levels of total cholesterol, triglycerides, and low-density lipoprotein were (248.0 ± 59.3 , 226.7 ± 52.5 , and 145.0 ± 32.6) mg/dL, respectively. , compared with the control group, (189.1 ± 30.5 , 189.6 ± 36.1 , 95.8 ± 22.9) mg/dL, respectively. While the level of high-density lipoprotein cholesterol and very low-density lipoprotein, there were high differences, as it reached (50.5 ± 11.6 , 42.8 ± 12.1) mg/dL compared to healthy subjects, which amounted to (55.1 ± 13.7 , 38.2 ± 11.2), mg/dL. We conclude from this study that hypothyroidism leads to an increase in the level of fats, due to a decreasing in metabolic processes, which is also the cause of hyperlipidemia.

KEYWORDS : Thyroid gland, hypothyroidism, Low – density lipoprotein, cholesterol.

1. INTRODUCTION

Hypothyroidism is one of the disorders that affect the thyroid gland, and it is the result of a decrease in the concentration of its hormones, T3 and T4 in the blood, so the TSH rise increases to compensate for this deficiency as a result of the negative feedback mechanism (1). Iodine is recognized as a contributing factor in developing hypothyroidism, which is essential for synthesizing thyroid hormones T3 and T4. However, a significant portion of the global population faces inadequate mineral intake from food sources due to variations in soil composition, affecting the crops' mineral content (2). The insufficiency of dietary iodine can decrease the capacity to manufacture T3 and T4, causing many diseases. When the synthesis of T3 and T4 is impaired, there is a significant increase in the secretion of thyroid-stimulating hormone (TSH)(3). Due to excessive promotion, there is an increase in the levels of thyroglobulin (Tg) within the thyroid follicles, leading to an elevation in their colloid content. The enlargement of the thyroid gland due to colloid accumulation is referred to as a goiter (4). A goiter alone serves as a visible manifestation of the deficit. Iodine deficiency disorders encompass Varity of adverse effectiveness, including impaired growth and development, reduced fertility, and increased risk of newborn mortality (5). Thyroid hormones significantly affect lipids' synthesis, mobilization, and metabolism (6, 7). According to a previous study conducted by Liberopoulos and Elisaf, 2002 (8), the association between thyroid dysfunction and total cholesterol and low-density lipoprotein levels has been observed. The observed changes in lipid profile in individuals with hypothyroidism can be primarily attributed to a reduction in the action of low-density lipoprotein (LDL) receptors (9). Therefore, hypothyroidism significantly contributes to developing secondary dyslipidemia (10). The prevalence of hypothyroidism escalates with progressive age, particularly among the female population (11).

2. SUBJECT AND METHODS

One hundred women participated in the study, 60 of women had hypothyroidism. Who visited Salah Al-Din Hospital in Salah Al-Din Governorate in Iraq, while the remaining 40 were healthy volunteers.



2.1. Exclusion criteria

People who are treated with thyroid hormone, high blood pressure, and cardiovascular disease.

2.2. Ethical consent

The research was carried out in adherence to the ethical principles derived from the Declaration of Helsinki. It was carried out with patients verbal and analytical approval before subjects were recruited in the study. The study protocol, as well as the subject information and permission form, underwent a thorough evaluation and received approval from the Central Scientific Research Ethics Committee at Tikrit University. Research approval number 4959 in date: 7/12/2022.

2.3. Sampling collection

Three milliliters of blood were obtained from individuals diagnosed with hypothyroidism. The blood samples were subsequently transferred into gel tubes and allowed to stand undisturbed for 20 minutes. The samples were centrifugated at a speed of 3000 revolutions per minute for ten minutes to separate the serum. Subsequently, the serum was transferred into pendroff tubes and preserved in a deep freezer at a temperature of -20°C. The samples were allowed to reach room temperature before conducting the tests.

2.4. Assessment of serum total cholesterol, triglycerides and HDL concentration:

A commercial kit was used to assess the level of total cholesterol, triglycerides and HDL using a spectrophotometer.

2.5. Study enrollment procedures

Detailed information including age, gender, weight, genetic factor, and others were recorded.

2.6. Statistical analysis

The data analysis was conducted as Mean± SD using the SPSS 23 software and Microsoft Office Excel 2010, with SPSS referring to the Statistical Package for Social Sciences. Categorical data was characterized using numbers and percentages, whereas numerical data was represented by mean and standard deviation. The independent samples t-test was employed to compare two numerical variables.

3. RESULTS AND DISCUSSION

The result of this study in Figures (1, 2, and 3) and Table (1) showed a highly significant increase in the levels of total cholesterol, triglycerides, and LDL, as their levels reached 248.0 \pm 59.3, 226.7 \pm 52.5, and 145.0 \pm 32.6 mg/dL, respectively. , compared with the control group, it was 189.1 \pm 30.5, 189.6 \pm 36.1, 95.8 \pm 22.9 mg/dL, respectively.



Fig. 1:Assessment serum total cholesterol (mg/dL in Studied groups





Fig.2 : Assessment serum triglyceride (mg/dL) in Studied groups





Fig.3 :Assessment serum LDL (mg/dL) in Studied group

Thyroid dysfunction may result in Dyslipidemia (lipid abnormalities) which is correlated with endothelium disorders, hypertension and cardiovascular disease. Hyperlipidemia, reduces cholesterol removal, lacks antioxidant system and impacts on THs regulation of antioxidant enzymes. Hypothyroid is a common cause of hyperlipidemia in humans and animals. In hypothyroidism patients the greatest chronic lipid abnormality is hypercholesterolemia (12).

The body requires cholesterol to do all type of important roles, like support cell membranes, produce hormones, bile acids and fat-soluble vitamin which are required to aid digest fat. But like all good things, extreme cholesterol levels can be a bad thing even lethal. Levels of LDL receptor (LDLR) are depend to negative feedback control via cellular cholesterol by sterol regulatory element-binding protein-2 (SREBP-2) (13).

The greatest clear reason for hypercholesterolaemia is thyroid hormones reduction, translates to reduce LDL receptors on cell membrane, hence insufficient cholesterol removal and a raise in bad cholesterol LDL. Also, little thyroid hormones may cause more intestinal cholesterol absorption (intake) (14). Lack of the LDLR action leads to the accumulation of LDL particles in circulation result in development of atherosclerosis (15). A lineal association between TSH and cholesterol levels has been observed (16). As observed previously, thyroid hormones THs are capable to regulate the expression of LDLR, that results in changed cellular absorption and catabolism of LDL. T3 reduces the plasma concentration of cholesterol, via promoting its hepatic intake and conversion to bile acids and via promoting fecal bile acid secretion (17).

Triglycerides are raised because of an increased esterification of fatty acids at hepatic level. Also, low levels of THs reduce activity of lipoprotein lipase (LPL), the enzyme important for removal of TG, therefore result in rise levels of TG in serum (18). Jiang and his assistance revealed the novel role of TSH in decreasing the adipose triglyceride lipase ATGL expression in the mature adipocytes of rodents. These findings suggest that TSH affects basal lipolysis (19).

Figure (4,5) and table (1) showed that for high-density lipoprotein and very lowdensity lipoprotein, there were no statistically significant differences, as the differences amounted to $(50.5 \pm 11.6, 42.8 \pm 12.1) \text{ mg} / \text{dL}$, respectively. Compared to healthy subjects, which amounted to (55.1). $\pm 13.7, 38.2 \pm 11.2$), mg/dL, respectively.



Fig.4 : Assessment serum HDL (mg/dL) in Studied group



Fig.5: Assessment serum VLDL (mg/dL) in Studied group

HDL have many of atheroprotective roles involving facilitation of inverse cholesterol transport, perfection of endothelial function, LDL preservation from oxidation, control of hemostasis and lag of inflammatory activity linked with the vascular wall. Elevated levels of TGs are usually appear with decrease HDL. A potential mechanism is which HDL correlated with increased triglyceride concentrations may be extra easily catabolized (20).

The exchange by cholesteryl ester transfer protein CETP activity is believed responsible for the reverse relationship between levels of TG and HDL (21) .Lipoprotein lipase (LPL) action induced by evaluated levels of THs, may also participate to reduce lipoproteins circulating levels (15). THs also decrease Apo lipoprotein B (Apo B) levels in liver, that reduces the synthesis of VLDL and LDL (13).

 Table (1): Assessment levels of serum total cholesterol, Triglycerides, HDL, LDL, VLDL in women with hypothyroidism and control group

Parameters	Mean ± SD		P – Value
	Control	Patients	
Total cholesterol	189.1 <u>+</u> 30.5	248.0 <u>+</u> 59.3	0.01
(mg/dL)			
Triglycerides	189.6 <u>+</u> 36.1	226.7 <u>±</u> 52.5	0.01
(mg/dL)			
HDL(mg/dL)	55.1 <u>+</u> 13.7	50.5 <u>+</u> 11.6	0.172
LDL(mg/dL)	95.8 <u>+</u> 22.9	145.0 <u>+</u> 32.6	0.01
VLDL (mg/dL)	38.2 <u>+</u> 11.2	42.8 ±12.1	0.151
P – Value = probability at 0.05 level			

4. CONCLUSION

Hypothyroidism leads to an increase in fat levels due to the effect of the thyroid gland on metabolic processes; high levels of harmful cholesterol in the blood impact the heart, blood vessels, and arteries.

Examining the lipid profile is of great importance for patients with hypothyroidism, as it must be considered that thyroid dysfunction is linked to hyperlipidemia.

This study also concluded that treating the thyroid gland will be the ideal solution for people who suffer from obesity or difficulty losing weight.

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Conflict of Interest

The authors declare no conflict of interest.

Authors' Declaration

The authors hereby declare that the work presented in this article is original and that any liability for claims relating to the content of this article will be borne by them.

REFERENCES

[1] Vargas-Uricoechea H, Bonelo-Perdomo A, Sierra-Torres CH. Effects of thyroid hormones on the heart. Clínica e Investigación en Arteriosclerosis. 2014;26(6):296-309.

[2] Regev A, Teichmann SA, Lander ES, Amit I, Benoist C, Birney E, et al. The human cell atlas. elife. 2017;6:e27041.

[3] Paschou SA, Vryonidou A, Goulis DG. Thyroid nodules: A guide to assessment, treatment and follow-up. Maturitas. 2017;96:1-9.

[4] Diouf M, Salem AB, Cherif R, Saghaei H, Wague A. Super-flat coherent supercontinuum source in As 38.8 Se 61.2 chalcogenide photonic crystal fiber with all-normal dispersion engineering at a very low input energy. Applied Optics. 2017;56(2):163-9.

[5] Ward L. Thyroid and Parathyroid Diseases: New Insights into Some Old and Some New Issues: BoD–Books on Demand; 2012.

[6] Vivek, R.; Wael, T.; Shanker, K. and Mazhar, K. Atrial fibrillation and hyperthyroidism: A literature review. Indian Heart Journal. (2017) ; 69(4): 545-550

[7] Krstevska B, Bosevski M, Ch D, Serafimoski V. Dyslipidaemia and hypertension in patients with subclinical hypothyroidism. Prilozi. 2009;30(2):93-102.

[8] Liberopoulos EN, Elisaf MS. Dyslipidemia in patients with thyroid disorders. HORMONES-ATHENS-. 2002;1:218-23.

[9] Duntas LH, Brenta G. A renewed focus on the association between thyroid hormones and lipid metabolism. Frontiers in endocrinology. 2018;9:511.

[10] TSIMIHODIMOS V, BAIRAKTARI E, TZALLAS C, MILTIADUS G, LIBEROPOULOS E, ELISAF M. The incidence of thyroid function abnormalities in patients attending an outpatient lipid clinic. Thyroid. 1999;9(4):365-8.

[11] Tejomani M, Meera K, Vasudha K. Relevance of creatine kinase activity and serum creatinine levels in hypothyroidism. International Journal of Recent Trends in Science And Technology. 2013;8(3):263-9.

[12] Chen Y, Wu X, Wu R, Sun X, Yang B, Wang Y, et al. Changes in profile of lipids and adipokines in patients with newly diagnosed hypothyroidism and hyperthyroidism. Scientific reports. 2016;6(1):26174.

[13] Abdel-Gayoum AA. Dyslipidemia and serum mineral profiles in patients with thyroid disorders. Saudi medical journal. 2014;35(12):1469.

[14] Alsalmi WM, Shaglouf LHF, Azab AE. Correlation between hypothyroidism, hyperthyroidism, and lipid profile in thyroid dysfunction patients. Clin Med J. 2018;4(2):6-14.

[15] Yadav NK, Arjuman A, Chandra NC. Role of leptin on the expression of low density lipoprotein receptor. The Indian Journal of Medical Research. 2014;140(4):524.

[16] Fazaeli M, Khoshdel A, Shafiepour M, Rohban M. The influence of subclinical hypothyroidism on serum lipid profile, PCSK9 levels and CD36 expression on monocytes. Diabetes & Metabolic Syndrome: Clinical Research & Reviews. 2019;13(1):312-6.

[17] Guarnizo-Poma M, Paico-Palacios S, Pantoja-Torres B, Lazaro-Alcantara H, Urrunaga-Pastor D, Benites-Zapata VA, et al. Association between free thyroid hormones values and the lipid profile in middle-aged women with chronic symptoms. Diabetes & Metabolic Syndrome: Clinical Research & Reviews. 2018;12(4):531-5.

[18] Sinha RA, Singh BK, Yen PM. Direct effects of thyroid hormones on hepatic lipid metabolism. Nature Reviews Endocrinology. 2018;14(5):259-69.

[19] Jiang D, Ma S, Jing F, Xu C, Yan F, Wang A, et al. Thyroid-stimulating hormone inhibits adipose triglyceride lipase in 3T3-L1 adipocytes through the PKA pathway. PLoS One. 2015;10(1):e0116439.

[20] Ma S, Jing F, Xu C, Zhou L, Song Y, Yu C, et al. Thyrotropin and obesity: increased adipose triglyceride content through glycerol-3-phosphate acyltransferase 3. Scientific reports. 2015;5(1):7633.

[21] Li AA, Makris SL, Marty MS, Strauss V, Gilbert ME, Blacker A, et al. Practical considerations for developmental thyroid toxicity assessments: What's working, what's not, and how can we do better? Regulatory Toxicology and Pharmacology. 2019;106:111-36.