

Evaluation of the Adiponectin Hormone and Lipid profile in patients with Hypothyroidism in Thi-Qar Province-Iraq

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ABSTRACT

There are 2 types of Hypothyroidism, primary hypothyroidism and secondary hypothyroidism. Primary hypothyroidism arises when thyroid gland itself doesn't work properly, whereas secondary hypothyroidism is initiated when the thyroid gland doesn't get adequate stimulation from thyroid stimulating hormones. Primary hypothyroidism is triggered by iodine deficiency, autoimmune disease, radiation therapy, drugs or thyroid surgery. The study was designed to determine and compare the levels of **TSH**, **T4**, **T3**, adiponectin hormone (**ADP**), and lipid profile (**TCH**), (**TG**), (**HDL**), (**LDL**), and (**VLDL**) in patients with Hypothyroidism and apparently healthy individuals. Blood levels **TSH**, **T4**, **T3**, adiponectin hormone (**ADP**), and lipid profile (**TCH**), (**TG**), (**HDL**), (**LDL**), and (**VLDL**) were determined in 45 patients with Hypothyroidism and 40 apparently healthy subjects. The levels of serum **TSH** and biochemical markers of body lipid profile (serum **TCH**, **TG**, **LDL**, **VLDL**) were showing an enormous rise in patients having Hypothyroidism when matched with the control group. On the contrary, the levels of **T4**, **T3**, **ADP**, and **HDL** indicated a considerable decrease in patients having Hypothyroidism when matched with control subjects ($P \leq 0.05$). In patients with Hypothyroidism, we found an increase in TSH. There is a momentous elevation in the cholesterol and triglyceride levels during patients with Hypothyroidism. Hypothyroidism can affect on lipoproteins levels (high LDL, low HDL, and high VLDL, the decrease in T4, T3, and ADP can clearly occur).

Keywords: Hypothyroidism, primary Hypothyroidism, Adiponectin hormone, and lipid profile.

INTRODUCTION

Thyroid hormone runs the process of metabolism in the human body. Hypothyroidism is a dysfunction which is triggered when the thyroid gland does not make adequate thyroid hormones, so the body does not continue to function normally and the organic activity decreases or slows down. It is considered as a great challenge for the health of people in both developed and underdeveloped countries⁽¹⁾. There are 2 types of Hypothyroidism, primary hypothyroidism and secondary hypothyroidism. Primary hypothyroidism arises when thyroid gland itself doesn't work properly, whereas secondary hypothyroidism is initiated when the thyroid gland doesn't get adequate stimulation from thyroid stimulating hormones. Primary hypothyroidism is triggered by iodine deficiency, autoimmune disease, radiation therapy, drugs or thyroid surgery⁽²⁾. The decrease in the metabolic process is due to decreased flow of thyroid hormones. This triggers the catalyzation of anterior pituitary gland which releases TSH to favor the creation of more thyroid hormones^(3,4). The treatment of hyperthyroidism is based on several factors such as clinical appearances, patient's history, TSH and serum thyroid hormone levels. In the case of overt primary hypothyroidism, Serum TSH is increased whereas the levels of T4 and T3 are reduced. In the case of subclinical primary hypothyroidism, the T3 and T4 levels are normal whereas serum TSH levels are

high⁽⁵⁾. Adiponectin is a type of adipocyte hormones, it is named as adipokines and it plays a significant part in metabolic disorders by promoting inflammation of adipocytes, the metabolic disorders could be type 2 diabetes, obesity, cardiovascular diseases, and hypertension⁽⁶⁾. These thyroid hormones and adipokines have numerous physiological effects including the metabolism of lipids and glucose and regulating energy expenditure^(7, 8). Lipids are a class of nonpolar molecules, they are found in the cell membranes, in the endoplasmic reticulum, and in specialized fat storage cells⁽⁹⁾. Cholesterol is derived from dietary intake, most is synthesized by the liver and other tissues from simpler molecules. Almost 90% of synthesis occurs in the liver and gut; therefore, peripheral cells and other organs depend largely on cholesterol delivery from the circulation⁽¹⁰⁾. Triglycerides are known as fatty acid esters of glycerol. Each one of them contains dissimilar fatty acid⁽¹¹⁾. Lipoproteins are composed of a protein and a lipid. (HDL, LDL, VLDL, chylomicrons)⁽¹²⁾.

Materials And Methods

This experiment was performed at the Center of Diabetes and the Endocrine Glands in Thi Qar governorate, and specialist clinics. It included (85) subjects, control (40) and patients (45) diagnosed with Hypothyroidism including: (23 female, 22 male). The

blood sample of approximately (5milliliters)was taken from the patients of Hypothyroidism. The blood waspermitted to clot at normal temperature in blank disposable tubes centrifuge for separating it in the centrifuge for 10 minutesat 3000 xg. The samples of serum were allowed to separate and were stored at the temperature of -20°Cuntil analyzed for T3, T4, Adiponectin hormone, TSH and Lipid Profile.It is a type of automated quantitative test that uses VIDAS instruments. The experiment was conducted for the determination of enzyme immunoassay of human TSH, T3, and T4 in the plasma or serum of human using the method of **enzyme-linked fluorescent assay (ELFA)**.The experiment used the kit provided by Elabsience, USA to determine Serum Adiponectin with the help of enzyme-linked immunoassay technique by ELISA Reader, USA.Serum cholesterol(TCH) was analyzed by the enzymatic colorimetric method byUV/VIS spectrophotometer, Japanusing kits supplied bySpinreact, Spain.Serum triglyceride (TG) was analyzed by the enzymatic colorimetric method byUV/VIS spectrophotometer, Japanusing kits supplied byBiolabs, France. Serum high-density lipoprotein (HDL) was analyzed by enzymatic colorimetric process byUV/VIS

spectrophotometer, Japanusing kits supplied byBiomerieux, France.Serum low-density lipoprotein (LDL) is estimated through the below equation:- $LDL = Total\ Cholesterol - (HDL + VLDL)$

Serum very low-density lipoprotein (VLDL) is estimated through the following equation:- $VLDL = Triglyceride/2.2$

The results of the experiment were presented in the form of mean ± standard deviations (mean ± SD). In order to compare parameters in various studied groups, one-way ANOVA-test was employed. P-values ($P \leq 0.05$) were taken statistically important.

Results

In this work, we determined the effect of this diseaseon the**TSH, T4, and T3**. Adiponectin hormone (**ADP**), we are concerning its effect onlipid profile (**TCH**),(**TG**), (**HDL**),(**LDL**), and (**VLDL**).The levels of serum TSH and biochemical markers of body lipid profile (serum TCH,TG,LDL VLDL) were showing a significant increase in hypothyroidism patients in comparison to control group.On the contrary, the levels of T4, T3, HDL,and ADPindicated a considerable reduction in hypothyroidism patients when compared to control subjects.

Table(1):-Serum TSH, T4, and T3 concentrations of(control) and(hypothyroidism patients) groups

Group	n	TSH (µIU/ml) mean ± SD	T4 (µg/dl) mean ± SD	T3 (ng/ml) mean ± SD
Control	40	2.65± 0.81 ^b	5.51± 0.99 ^b	1.22±0.35 ^b
hypothyroidism patients	45	4.92± 1.04 ^a	3.47±0.73 ^a	0.63 ± 0.10 ^a
LSD		0.82	1.03	0.08

Each value in this table indicate the values of the mean ± SD with non-identical superscript (a, b or c...etc.) and were taken as expressively different ($P \leq 0.05$).

Table(2):- Serum Adiponectin concentrations of (control) and (hypothyroidism patients) groups

Group	n	Adiponectin concentrations (ng/ mL) mean ± SD
Control	40	6.74±1.69 ^a
hypothyroidism patients	45	0.50±0.11 ^b
LSD		0.17

- Legend as in table (1).

Table(3):-Serum Lipid Profile concentrations of(control) and (hypothyroidism patients) groups

Groups	n	TC mmol/L	TG mmol/L	HDL mmol/L	LDL mmol/L	VLDL mmol/L
control	40	3.76±0.98 ^b	1.42±0.63 ^b	1.35±0.42 ^a	1.55±0.84 ^b	0.35±0.11 ^b
hypothyroidism patients	45	6.12±1.31 ^a	2.36±0.72 ^a	0.79±0.16 ^b	4.05±1.22 ^a	0.45±0.12 ^a
LSD		0.82	0.34	0.31	0.91	0.15

- Legend as in table (1)

Discussion

Hypothyroidism emerges as a result of deficient excretion of thyroid hormones. This happens due to a problem in the HPT axis. Hypothyroidism is usually triggered by autoimmune thyroid and its related diseases. It is the first indicator of thyroid or pituitary disease⁽¹³⁾. In overt hypothyroidism, the fT4 levels are found to be low whereas TSH levels are noted as high. The patients having a low serum fT4 and a high serum TSH concentration indicate the symptom of hypothyroidism^(14,15). Thyroid hormones and Adiponectin have a few similar properties such as lipid oxidation and reduction in body fat by rising thermogenesis⁽¹⁶⁾. It is noted that adiponectin may affect the production of thyroid hormone by its interface with gC1q receptor located in thyroid mitochondria⁽¹⁷⁾. Dimitriadis *et al.* indicates decreasing levels of adiponectin in hypothyroidism⁽¹⁸⁾. Nagasaki *et al.*, indicated in his study the comparable levels of adiponectin hypothyroid and controls patients⁽¹⁹⁾. In the experiment performed, a considerable rise in serum levels of TG, TC, and LDL was observed in patients having hypothyroidism. This matches the results of previous reports. The movement of lipoprotein lipase (LPL) is decreased by the decreased levels of thyroid hormones. LPL is an enzyme that has a great part in the clearance of TG-rich lipoproteins^(20,21). This causes a rise in the levels of TG in the serum. Thyroid hormones like T3 are directly bound to thyroid hormone responsive elements (TREs) to control LDL receptors⁽²²⁾. They also control sterol regulatory element-binding protein⁽²³⁾. In hypothyroidism, the reduced thyroid hormones cause a decrease in the expression of LDL receptors. This weakens cellular uptake of LDL from catabolism and circulation of LDL. This eventually causes a rise in the levels of circulating TC^(24,25). The higher ratio of LDL to HDL is found in patients with hypothyroidism because of a slight decrease in HDL levels and a major increase in LDL levels⁽²⁶⁾.

Conclusion

From the experiment and the discussion, it can be concluded that there is a rise in TSH in Hypothyroidism patients. The level of triglyceride and cholesterol are also significantly high in patients with Hypothyroidism. Hypothyroidism can effect on lipoproteins levels (high LDL, low HDL, and high VLDL, the decrease in T4, T3, and ADP can clearly occur.

References

- Unnikrishnan A.G. and Menon U.V. Thyroid disorders in India: An epidemiological perspective. *Indian J Endocrinol Metab.* 2011; 15(Suppl2):S78–81.
- Díez J.J. Hypothyroidism in Patients Older Than 55 Years: An Analysis of the Etiology and Assessment of the Effectiveness of Therapy. *J Gerontol A Biol Sci Med Sci.* 2002; 1; 57(5):M315–20.
- Porth C. and Matfin G. *Pathophysiology: Concepts of Altered.* 2010.
- Ignatavicius D. and Workman L. *Medical-Surgical Nursing: patient centered collaborative care (6th ed),* 2010.
- Urden L. D., Stacy K. M., and Lough M. E. *Critical Care Nursing: Diagnosis and Management.* 6th ed. St. Louis, MO: Elsevier Mosby. 2010.
- Gulcelik N. E., Usman A. and Gurlek A. Role of adipocytokines in predicting the development of diabetes and its late complications. *Endocrine.* 2009, 36, 397–403. Health States. 8th ed. Philadelphia, PA: Lippincott Williams & Wilkins.
- Iglesias P. and Díez J. J. Influence of thyroid dysfunction on serum concentrations of adipocytokines. *Cytokine.* 2007, 40, 61–70.
- Lin S. Y., Huang S. C. and Sheu W. H. Circulating adiponectin concentrations were related to free thyroxine levels in thyroid cancer. 2010.
- Mathews Ch. K., Van Hold K.E. and Ahern K.G. "Biochemistry", 3rd edn. Eds. Addison Wesley Longman, Inc. San Francisco, 2000.
- Burtis C.A. and Ashwood E.R. "Tetiz Fundamentals of Clinical Chemistry", 4th edn. Eds. W.B. Saunders company. Philadelphia. 1996, p. 272, 274, 275, 376, 727. *Critical Thinking for Collaborative Care.* 6th ed. Philadelphia, PA: Elsevier Saunders.
- Mayne P.D. (2002). "Clinical Chemistry in diagnosis and Treatment", 6th edn. Eds. Arnold. 2002, 224–225, 317, 322.
- Rodenburg K.W. and Vander Horst D.J. "Lipoprotein-mediated lipid transport in insects: Analogy to the mammalian lipid carrier system and novel concepts for the functioning of LDL receptor family members". *Biochim. Biophys. Acta.* 2005, 1736:10–19.
- Shargel L., Mutnick A.H., Souney P.F., and Swanson L.N. *Comprehensive Pharmacy Review.* 8th ed. New Delhi: Wolters Kluwer (India) Pvt Ltd. 2013.
- Jameson and Groot D. *Endocrinology: Adult and Pediatric Volume 2.* 7th ed. India: Reed Elsevier India Pvt. Ltd. 2016.
- Wass J., Owen K., and Turner H. *Oxford handbook of endocrinology and diabetes.* 3rd ed. India: Oxford University Press. 2014.
- Ahima R. S., Qi Y., Singhal N. S., Jackson M. B., and Scherer P. E. "Brain adipocytokine action and metabolic regulation," *Diabetes.* 2006, vol. 55, no. 2, pp. S145–S154.
- Fernández-Real J. M., Lopez-Bermejo A. L., Casamitjana R., and Ricart W. "Novel interactions of adiponectin with the endocrine system and inflammatory parameters," *Journal of Clinical Endocrinology and Metabolism.* 2003, vol. 88, no. 6, pp. 2714–2718.
- Dimitriadis G., Mitrou P., Lambadiari V. et al. "Insulin action in adipose tissue and muscle in hypothyroidism," *Journal of Clinical Endocrinology and Metabolism.* 2006, vol. 91, no. 12, pp. 4930–4937.
- Nagasaki T., Inaba M., Hiura Y. et al. "Plasma levels of adiponectin and soluble thrombomodulin in

- hypothyroid patients with normal thyroid function following levothyroxine replacement therapy," *Biomedicine and Pharmacotherapy*.2005, vol. 59, no. 10, pp. 571–577.
20. Thompson G. R. et al. Defects of receptor-mediated low density lipoprotein catabolism in homozygous familial hypercholesterolemia and hypothyroidism in vivo. *Proc Natl Acad Sci USA*.1981, 78, 2591–2595.
 21. Nikkila E. A. and Kekki M. Plasma triglyceride metabolism in thyroid disease. *J Clin Invest*.1972, 51, 2103–2114.
 22. Bakker O., Hudig F., Meijssen, S. and Wiersinga W. M. Effects of triiodothyronine and amiodarone on the promoter of the human LDL receptor gene. *Biochem Biophys Res Commun*.1998, 249, 517–521.
 23. Shin D. J. and Osborne T. F. Thyroid hormone regulation and cholesterol metabolism are connected through Sterol Regulatory Element-Binding Protein-2 (SREBP-2). *J Biol Chem*.2003, 278, 34114–34118.
 24. Duntas L. H. Thyroid disease and lipids. *Thyroid*.2002, 12, 287–293.
 25. Faure P., Oziol L., Artur Y. and Chomard, P. Thyroid hormone (T3) and its acetic derivative (TA3) protect low-density lipoproteins from oxidation by different mechanisms. *Biochimie*.2004, 86, 411–418.
 26. Lagrost L. Regulation of cholesteryl ester transfer protein (CETP) activity: review of in vitro and in vivo studies. *Biochim Biophys patients after thyroid hormone withdrawal. Metabolism*.1994, 59, 195–199.