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ORIGINAL RESEARCH

To determine thyroid function test and lipid profile markers in hypothyroidism patients

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ABSTRACT

Background: Thyroid function tests(TFT) and lipid profiles are crucial diagnostic tools for understanding body processes, assessing thyroid gland performance, and identifying primary thyroid disorders like hypothyroidism.**Aim:** The aim of the present study was to determine thyroid function test and lipid profilemarkers in hypothyroidism patients.**Materials & methods:** The study, conducted in the Department of Biochemistry, involved 300 participants aged 30-60 years, divided into two groups: 150 healthy individuals and 150 hypothyroidism patients. The study aimed to identify clinically diagnosed hypothyroid patients aged 30-60 years who have been on medication within the last five years. Patients with established conditions, chronic illnesses, pregnancy, recent surgical interventions, or those under 30 or over 60 years were excluded from the study.**Results:** T3, T4, fT3, fT4, and TSH levels are shown in Figure 1. All the thyroid profile tests were significant differed when compared between control group subjects and hypothyroid group patients. Significant differences were observed in the serum levels of lipid variables, apolipoproyein-A1 (Apo-a1), lipoprotein-A1 (lLpo-a1), and apolipoprotein-B (Apo-B) when hypothyroid group patients compared to control subjects.**Conclusion:**Thyroid dysfunction, including hypothyroidism, significantly impacts cardiovascular health and lipid profile markers, requiring early detection and management to reduce cardiac risk.

Key words: Hypothyroidism; Thyroid profile; Lipid profile; Apolipoprotein-A1; Lipoprotein-A1; Apolipoprotein-B.

INTRODUCTION

Hypothyroidism, a condition characterized by inadequate thyroid hormone production, induces a variety of metabolic disturbances that substantially influence lipid metabolism. The thyroid gland is essential for the regulation of metabolism, which encompasses the synthesis, mobilization, and degradation of lipids. Hypothyroid patients experience a slowed basal metabolic rate due to a decrease in thyroid hormone levels, specifically thyroxine (T4) and triiodothyronine (T3), leading to dyslipidemia[1,2]. This disruption is characterized by elevated levels of total cholesterol, low-density lipoprotein (LDL), and triglycerides, as well as reduced high-density lipoprotein (HDL). These modifications in the lipid profile in hypothyroid patients are associated with an elevated risk of cardiovascular diseases[3,4].Thyroid function tests measure free triiodothyronine (FT3), free thyroxine (FT4), and thyroid-stimulating hormone (TSH). They are a standard way to find out if your thyroid isn't working right[5,6]. However, these tests alone may not fully capture the impact of hypothyroidism on lipid metabolism. To gain a more comprehensive understanding of the metabolic repercussions of hypothyroidism, it is essential to conduct a comprehensive analysis of lipid profile markers, including total cholesterol, LDL, HDL, and triglycerides, in conjunction with thyroid function tests[7-10].Thyroid function and lipid profile markers in hypothyroid patients in order to investigate the correlation between thyroid hormone imbalances and lipid abnormalities. This research has the potential to enhance the management and prevention of cardiovascular complications in this population. Hence, the aim of the present study was to determine thyroid function test and lipid profilemarkers in hypothyroidism patients.

MATERIALS & METHODS

Ethical approval: The current study commenced following the acquisition of ethical clearance from the Institutional Ethics Committee. Study Design: Case-Control Study. Sampling: Purposeful Random Sampling. The current study is a prospective investigation conducted in the Department of Biochemistry. A total of 300 participants aged

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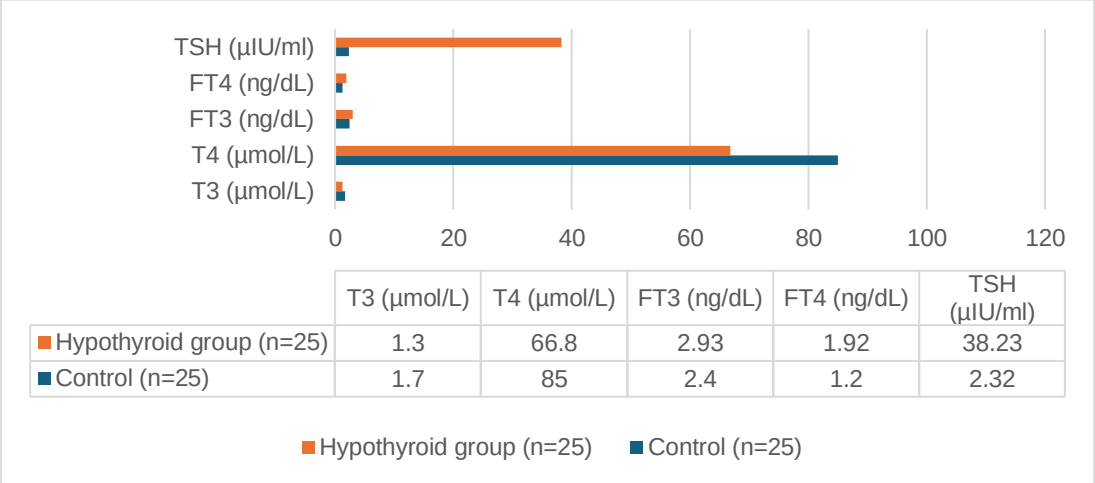
between 30 and 60 years were separated into the following two groups: Group 1: 150 healthy individuals. Group 2: 150 patients with hypothyroidism. Criteria for inclusion of Group 2 patients: The study will involve clinically diagnosed hypothyroid patients of both sexes, aged 30 to 60 years, who have been on medication within the last five years. Exclusion Criteria for Patients in Group 2: Individuals with established cardiac disease, chronic renal failure, diabetes mellitus, hepatic disorders, rheumatoid arthritis, gout, chronic illnesses, pregnancy, recent surgical interventions, any other inflammatory conditions, and those aged under 30 or over 60 years will be excluded. Methodology: Following the acquisition of written consent, subjects were enrolled. Upon obtaining consent, participants were classified according to thyroid dysfunction at the time of patient enrollment. The patient's medical history, familial history, and physical examination were recorded. Six milliliters of venous blood were collected from hypothyroid patients and euthyroid participants in a simple tube to obtain serum. Serum was employed for the assessment of the subsequent biochemical markers. Assessment of thyroid hormones (T3, T4, Free T3, Free T4, and TSH): Utilizing the chemiluminescence technique. These were acquired from Auto-bio-Laboratories, and the tests were conducted in accordance with the directions outlined in the manuals.Serum lipid profile were estimated by using the method of Cholesterol Oxidase and Peroxidase (CHOD/POD) purchased from Avantor Performance Materials India Limited, Dehradun, Uttarakhand, India.The quantification of apolipoprotein A1, lipoprotein A1, and apolipoprotein B was conducted using ELISA techniques. The kits were acquired from Thermo-Scientific Indiko Laboratories.

Statistical analysis

The study employed IBM SPSS version 29 for statistical analysis, which involved the comparison of patients with varying degrees of hypothyroidism severity, as well as the analysis of demographics, age, biochemical parameters, and the relationships between thyroid hormone levels and lipid profile markers.

RESULTS

Figure 1: At baseline, the various research populations had their thyroid functions tested.



All values expressed as Mean. Triiodothyronine (T3), Thyroxine (T4), Free-triiodothyronine (fT3), Free-thyroxine (fT4), Thyroid stimulating hormone (TSH).

T3, T4, fT3, fT4, and TSH levels are shown in Figure1. All the thyroid profile tests were significant differed when compared between control group subjects and hypothyroid group patients.As shown in the table 1, significant differences were observed in the serum levels of lipid variables, apolipoproyein-A1 (Apo-a1), lipoprotein-A1 (lLpo-a1), and apolipoprotein-B (Apo-B) when hypothyroid group patients compared to control subjects.

Table 1: Lipid profile of the different study populations at baseline.

Variable	Control group (n=150)	Hypothyroid group (n=150)	T Test
TC (mg/dL)	131.3±5.4	215.8±16.8	< **0.05**
TAG (mg/dL)	141.3±2.1	214.1±20.4	< **0.05**
HDL (mg/dL)	48.16±3.4	36.1±3.93	< **0.05**
LDL (mg/dL)	59 ± 2.1	121.7±19.2	< **0.05**
Apo-a1 (mg/dL)	98.8± 34.2	78.2 ± 21.3	< **0.05**
Lipo-a (mg/dL)	22.6 ± 2.9	42.2 ± 12.1	< **0.05**
Apo-B (mg/dL)	78 ± 21.8	142 ± 32.7	**<0.05**

All values expressed as Mean±SE. Total cholesterol (TC), triacylglycerols (TAG), low density lipoproteins (LDL), high density lipoproteins (HDL).

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DISCUSSION

In the present study, dyslipidemia might be an added insult to the antioxidant status by probably increasing the lipid peroxides, although we did not observe a correlation between oxidative stress variables and dyslipidemia along with significant changes in the values of Apo-A1, Lipo-1, and Apo-B when compared between the control individuals and the hypothyroid patients. Studies that reported an increase in Apo-A1 also reported dyslipidemia in patients in the hypothyroid group [8,9].

It is possible to Hypothyroid patients may experience a decrease in Apo-A1 levels, a condition that may worsen as they age [4-7] induces hypercholesteremia and hampers the uptake of cholesterol by HDL molecules. Previous studies [11–15] have reported lower HDL levels causing deposition of cholesterol, and the current study results showed reduced HDL. This could potentially be associated with an increase in oxidative stress and hypercholesteremia. On the other hand, some studies found improvements in TC and Apo-A1 in hypothyroidindividuals after thyroxine supplementation [15-17]. Consequently, research now confirms that T4 boosts the production of antioxidants [18]. Therefore, the increase in Apo-B reduced the HDL and the resulting hypercholesterolemia, potentially leading to a decrease in the formation of oxidized molecules. This may be due to an increase in the expression of genes responsible for increased antioxidant generation [9]. T4 supplementation improves dyslipidemia levels. Some studies [3-9] observed a similar improvement in these variables upon individual T4 supplementation.

The present study observed dyslipidemia in hypothyroid group at baseline. In addition, we observed positive correlation between TSH and Lipo-A protein (r^2= 0.521; P< 0.05), on the contrary negative correlation between TSH and Apo-a1 (r^2=-0.377, P=0.032) was observed. Previous studies have demonstrated that several models of lipoatrophic mice overexpress SREBP-1c in their adipose tissue, which ultimately leads to an increase in cholesterol levels. Also, T4's resistance to cells that make TSH has an effect on lipids by increasing the genes of important enzymes that break down lipids [1, 2]. for lipid metabolism [1, 2]. It is well known that HDL together with Apo-a1 are a lipoprotein that carries cholesterol to the liver and transfers TAGs to other lipoproteins, and insulin is known to promote liver apolipoprotein A and HDL biosynthesis [5,6]. Increased T4 resistance hinders this process, resulting in impaired HDL secretion [7]. Therefore, these data suggest that T4 and T4 resistance-derived factors decrease HDL and Apo-a1 levels, while also increasing the formation of TC and TAG levels in the current study. Studies [11-14] demonstrated that hypothyroid group patients are common metabolic disorders associated with profound alteration in lipid and lipoprotein profiles. Therefore, hypothyroid group patients exhibit derangement in the anabolic process, leading to increased synthesis of cholesterol and decreased utilization of TAGs.

Recommendations

The study on thyroid dysfunction's impact on cardiac health and oxidative stress markers in India suggests several recommendations for improving diagnosis, management, and research. These include nationwide screening programs, revising medical education curricula, developing personalized treatment approaches, promoting interdisciplinary research, and launching public health initiatives. National guidelines and protocols should be established to manage thyroid dysfunction effectively, addressing limitations in future research.

Limitations

The study investigates the influence of lipid profile markers and thyroid dysfunction; however, it is constrained by a cross-sectional design, the inability to monitor long-term progression, and the potential for inadequately documenting regional variations in genetics, diet, and lifestyle in India. In order to enhance comprehension of the influence of thyroid dysfunction on cardiac health and lipid profile markers, future research could resolve these constraints.

CONCLUSION

Thyroid dysfunction, including hypothyroidism, significantly impacts cardiovascular health and lipid profile markers. The thyroid gland controls metabolism and hormones, affecting heart function and lipid metabolism. Hypothyroidism leads to alterations in cardiac function elevated cholesterol, while hypothyroidism decreases cardiac output and heart rate. Early detection and management of thyroid disorders are crucial to reduce cardiac risk.

Conflict of interest

There is no conflict of interest among the present study authors.

REFERENCES

1. Wouters HJ, Slagter SN, Muller Kobold AC, van der Klauw MM, Wolffenbuttel BH. Epidemiology of thyroid disorders in the Lifelines Cohort Study (the Netherlands). PLoS One. 2020 Nov 25;15(11):e0242795. 10.1371/journal.pone.0242795
2. Desai MP. Disorders of thyroid gland in India. The Indian Journal of Pediatrics. 2015 Jan;64:11-20. Available from: 10.1007/BF02795771.
3. Menon V, Sundaram KR, Unnikrishnan AG, Jayakumar RV, Nair V, Kumar H. High prevalence of undetected thyroid disorders in an iodine sufficient adult south Indian population. *J Indian Med Assoc.* 2015;107:72–7. Available from: 10.1046/j.1365-2265.2003.01895.x.
4. Cheserek MJ, Wu GR, Ntazinda A, Shi YH, Shen LY, Le GW. Association between thyroid hormones, lipids and oxidative stress markers in subclinical hypothyroidism. Journal of medical Biochemistry. 2015 Jul;34(3):323. doi: 10.2478/jomb-2014-0044

5. Elnakish MT, Ahmed AA, Mohler PJ, Janssen PM. Role of oxidative stress in thyroid hormone-induced cardiomyocyte hypertrophy and associated cardiac dysfunction: an undisclosed story. Oxidative Medicine and Cellular Longevity. 2015 Oct;2015.**10.1155/2015/854265**
6. Rasool M, Malik A, Saleem S, Ashraf MA, Khan AQ, Waquar S, Zahid A, Shaheen S, Abu-Elmagd M, Gauthaman K, Pushparaj PN. Role of oxidative stress and the identification of biomarkers associated with thyroid dysfunction in schizophrenics. Frontiers in pharmacology. 2021 Apr 29;12:646287. 10.3389/fphar.2021.646287
7. Corssac GB, de Castro AL, Tavares AV, Campos C, Fernandes RO, Ortiz VD, Siqueira R, Fernandes TR, Belló-Klein A, Araujo AS. Thyroid hormones effects on oxidative stress and cardiac remodeling in the right ventricle of infarcted rats. Life sciences. 2016 Feb 1;146:109-16.10.1016/j.lfs.2015.12.052
8. Wouters HJ, Slagter SN, Muller Kobold AC, van der Klauw MM, Wolffenbuttel BH. Epidemiology of thyroid disorders in the Lifelines Cohort Study (the Netherlands). PLoS One. 2020 Nov 25;15(11):e0242795. 10.1371/journal.pone.0242795
9. Carmona Carmona C, Bedoya P, Acevedo J, Cardona Arias J. Prevalence of thyroid disorders in an institution providing health services in Medellin-Colombia. Transl Biomed. 2018;9(2.149). DOI: 10.21767/2172-0479.100149
10. Diab N, Daya NR, Juraschek SP, Martin SS, McEvoy JW, Schultheiß UT, Köttgen A, Selvin E. Prevalence and risk factors of thyroid dysfunction in older adults in the community. Scientific reports. 2019 Sep 11;9(1):13156. 10.1038/s41598-019-49540-z
11. Martin SS, Daya N, Lutsey PL, Matsushita K, Fretz A, McEvoy JW, Blumenthal RS, Coresh J, Greenland P, Kottgen A, Selvin E. Thyroid function, cardiovascular risk factors, and incident atherosclerotic cardiovascular disease: the Atherosclerosis Risk in Communities (ARIC) Study. The Journal of Clinical Endocrinology & Metabolism. 2017 Sep 1;102(9):3306-15. 10.1210/jc.2017-00986
12. Mammen JS, McGready J, Oxman R, Chia CW, Ladenson PW, Simonsick EM. Thyroid hormone therapy and risk of thyrotoxicosis in community-resident older adults: findings from the Baltimore Longitudinal Study of Aging. Thyroid. 2015 Sep 1;25(9):979-86. 10.1089/thy.2015.0180
13. Mancini A, Di Segni C, Raimondo S, Olivieri G, Silvestrini A, Meucci E, Currò D. Thyroid hormones, oxidative stress, and inflammation. Mediators of inflammation. 2016;2016(1):6757154. 2016. **10.1155/2016/6757154**
14. Larsen CB, Riis KR, Winther KH, Larsen EL, Ellervik C, Hegedüs L, Brix TH, Poulsen HE, Bonnema SJ. Treatment of hyperthyroidism reduces systemic oxidative stress, as measured by markers of RNA and DNA damage. The Journal of Clinical Endocrinology & Metabolism. 2021 Jul 1;106(7):e2512-20. 10.1210/clinem/dgab273
15. Unnikrishnan AG, Menon UV. Thyroid disorders in India: An epidemiological perspective. Indian journal of endocrinology and metabolism. 2016 Jul 1;15(Suppl2):S78-81. *DOI:* 10.4103/2230-8210.83329
16. Biondi B, Cooper DS. The clinical significance of subclinical thyroid dysfunction. Endocrine reviews. 2015 Feb 1;29(1):76-131. 10.1210/er.2006-0043
17. Kalyani RR, Golden SH, Cefalu WT. Diabetes and aging: unique considerations and goals of care. Diabetes care. 2017 Apr;40(4):440. doi: 10.2337/dci17-0005
18. Rizos CV, Elisaf MS, Liberopoulos EN. Effects of thyroid dysfunction on lipid profile. The open cardiovascular medicine journal. 2015;5:76. doi: 10.2174/1874192401105010076