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UNRAVELING THE STUDY OF DYSLIPEDEMIA IN HYPOTHYRODISM

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ABSTRACT

Background: Thyroid hormone regulates a wide range of genes after its activation from the prohormone, thyroxine (T4), to the active form, triiodothyronine (T3). The signaling pathway is complex and highly regulated due to the expression of cell and tissue-specific thyroid hormone transporters, multiple thyroid hormone receptor (TR) isoforms, and interactions with corepressors and coactivators. Furthermore, in many cases, thyroid signals are involved in cross-talk with a range of other signaling pathways. **Materials & Methods:** The study conducted on a total of 20 participants in which, 10 samples were healthy and 10 samples were lies in male category and 10 samples were lies in female category. The lipid profile was assessed using standard methodologies. Statistical analysis was performed using the GraphPad Prism9 stat® Software. Analysis of data was done using one-way ANOVA employing Tukey's test, after analysis. $p<0.05$ was found to be statistically significant. **Results:** In our study population 45% belonged to Grade I obesity, while 19% Grade II obesity and the remaining 36% were found to be non- obese. A statistically significant difference was observed in Triglyceride and HDL cholesterol levels ($p <0.05$). **Conclusion:** The present study revealed that the Cholesterol level was observed significantly higher in Hypothyroid Males as compared to Hypothyroid Females. The prevalence of LDL, TG, and VLDL were seen higher in Hypothyroid Females in comparison to Hypothyroid Males. Increase in prevalence of the Metabolic Syndrome was higher in women than in men. This might be driven by the constant rise in Obesity in women. Thyroid Hormone might affect lipid metabolism and numerous studies have found that lipid levels increase as TSH levels increase

KEYWORDS: Thyroxine, Triiodothyronine, Thyroid Hormone.

INTRODUCTION

Thyroid hormone regulates a wide range of genes after its activation from the prohormone, thyroxine (T4), to the active form, triiodothyronine (T3) [1]. The signaling pathway is complex and highly regulated due to the expression of cell and tissue-specific thyroid hormone transporters, multiple thyroid hormone receptor (TR) isoforms, and interactions with corepressors and coactivators [2]. Furthermore, in many cases, thyroid signals are involved in cross-talk with a range of other signaling pathways [3]. Thyroid hormone is produced by the thyroid

gland, which consists of follicles in which thyroid hormone is synthesized through iodination of tyrosine residues in the glycoprotein thyroglobulin [4]. Thyroid hormones are any hormones produced and released by the thyroid gland, namely triiodothyronine (T3), thyroxine (T4) thyroid stimulating hormone (TSH). They are tyrosine-based hormones that are primarily responsible for regulation of metabolism. T3 and T4 are partially composed of iodine, derived from food. A deficiency of iodine leads to decreased production of T3 and T4, enlarges the thyroid tissue and will cause the disease known as simple goiter [5]. Thyroid stimulating hormone (TSH), secreted by the anterior pituitary in response to feedback from circulating thyroid hormone, acts directly on the TSH receptor (TSH-R) expressed on the thyroid follicular cell basolateral membrane [6]. TSH regulates iodide uptake mediated by the sodium/iodide symporter, followed by a series of steps necessary for normal thyroid hormone synthesis and secretion [7]. The thyroid hormones act on nearly every cell in the body. It acts to increase the basal metabolic rate, affect protein synthesis, help regulate long bone growth (synergy with growth hormone) and neural maturation, and increase the body's sensitivity to catecholamines (such as adrenaline) by permissiveness [8]. The thyroid hormones are essential to proper development and differentiation of all cells of the human body. These hormones also regulate protein, fat, and carbohydrate metabolism, affecting how human cells use energetic compounds. They also stimulate vitamin metabolism. Numerous physiological and pathological stimuli influence thyroid hormone synthesis [9].

PURPOSE

The purpose of this study is to assess the lipid profile and thyroid status of thyroid effected patients concerning their various health disorders.

In light of this, the study was taken up with the following objectives:

1. Measurement of weight, height, age and BMI.
2. To determine the serum of hypothyroid patients.
3. To determine the serum of T3, T4, TSH, HDL, VLDL, LDL, triglycerides and cholesterol levels of control and hypothyroid patients.
4. To evaluate the relationship between thyroid level with lipid profile in thyroid patients and to explore the differential effect of thyroid on lipid profile parameters.

MATERIAL AND METHODS

The blood samples were collected from Amandeep Hospital and K.D. Hospital. Analysis had been done in Khalsa Diagnostic Lab, Department of Medical Lab Science, Khalsa College of Pharmacy and Technology, Amritsar. The study included Healthy Subjects and Thyroid Patients with age groups: Which comes in divided into three categories: Group I: Healthy control (in which, the subjects were normal thyroid and lipid profile parameters), Group II: Male Hypothyroid Patients and Group III: Female Hypothyroid Patients. The study conducted on a total of 20 participants in which, 10 samples were healthy and 10 samples were lies in male category and 10 samples were lies in female category. The clinical history of the patients was obtained through an interview and a well-structured patient performa. Well informed written consents also been taken from the participants of the study. Ethical approval for the study has been obtained from the Institutional Ethical Committee.

Blood Sample Collection

For the collection of samples, a prominent vein was selected and tourniquet was applied and anterior cubital vein area was cleaned with a sterilized cotton swab dipped in spirit. Needles and syringes were properly inspected and then sampling was performed. A cotton swab was held firmly over the vein puncture site as soon as the needle is removed. After removing the needle, the collected blood was dispensed in the tubes. After obtaining the blood, the serum sample was separated for further biochemical analysis. For the preparation of serum, blood was allowed to clot and then centrifuged at 1100 to 2000 rpm for 10 minutes at 4°C. The samples were refrigerated for further use. 5 ml of blood sample was collected from the anterior cubital vein using a sterile disposable syringe and was transferred to a red top tube. Informed consent was taken from subjects. Samples were centrifuged at 1100 to 2000 rpm for 10 minutes to obtain clear serum. Serum sample was obtained from blood for the evaluation of T3, T3, Thyroid Stimulating Hormone, Total Cholesterol (TC), Triglyceride (TG), High-Density Lipoprotein Cholesterol, (HDL-C), Low-Density Lipoprotein Cholesterol (LDL-C) and Very Low-Density Lipoprotein Cholesterol (VLDL-C).

Biochemical investigation

Determination of Cholesterol in serum or plasma by CHOD/PAP method.[9]

The serum cholesterol level was estimated spectrophotometrically at 505 nm by using a commercially available kit (ERBA diagnostics Mannheim GmbH, Mannheim/Germany)

Determination of Triglycerides in serum and plasma by GPO/PAP method.[10]

The serum triglyceride level was estimated spectrophotometrically at 505 nm by using a commercially available kit (ERBA diagnostics Mannheim GmbH, Mannheim/ Germany)

Determination of HDL Direct

The serum HDL cholesterol level was estimated spectrophotometrically at 505/670 by using a commercially available kit (ERBA diagnostics Mannheim GmbH, Mannheim/Germany)

Determination of LDL Direct

LDL cholesterol was calculated from total cholesterol, triglycerides, and HDL cholesterol level, by using the following formula:

Calculation:

Cholesterol – (VLDL + HDL)

Reference Value: less than 100mg/dl

Determination of VLDL (Very low-density lipoprotein):

VLDL was calculated from TG using the following formula-

Calculation

Conc. of VLDL (mg/dl) = Triglycerides/5 Reference Value: 2-30 mg/dl

Table 1: Data of subjects

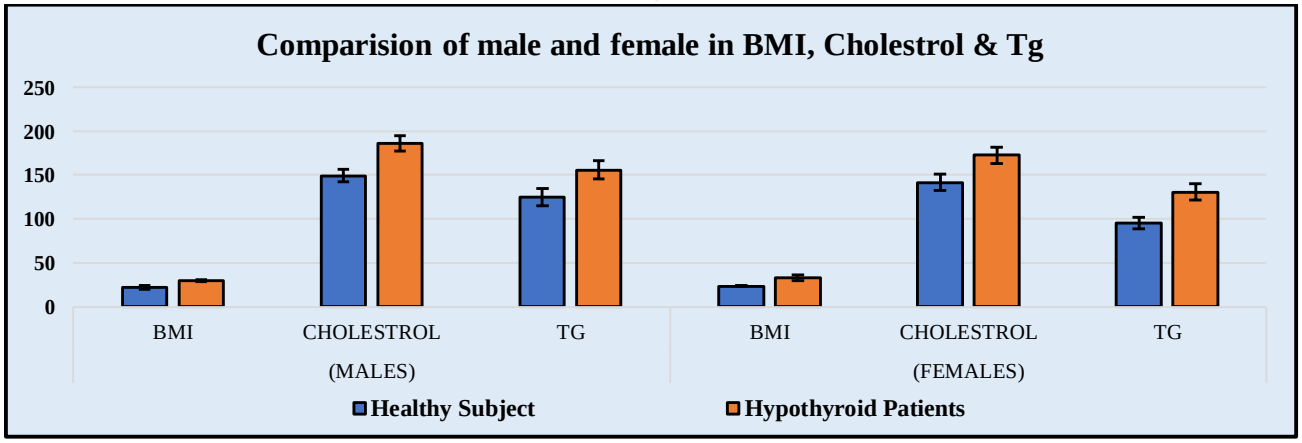
Total Population (n=100)		
Controls	**Perimenopausal women**	**Postmenopausal women**
n=13	**n=12**	**n=75**

Comparison between Age, Weight, and BMI of Controls, Perimenopausal and Postmenopausal women (Table 2and Figure 2).

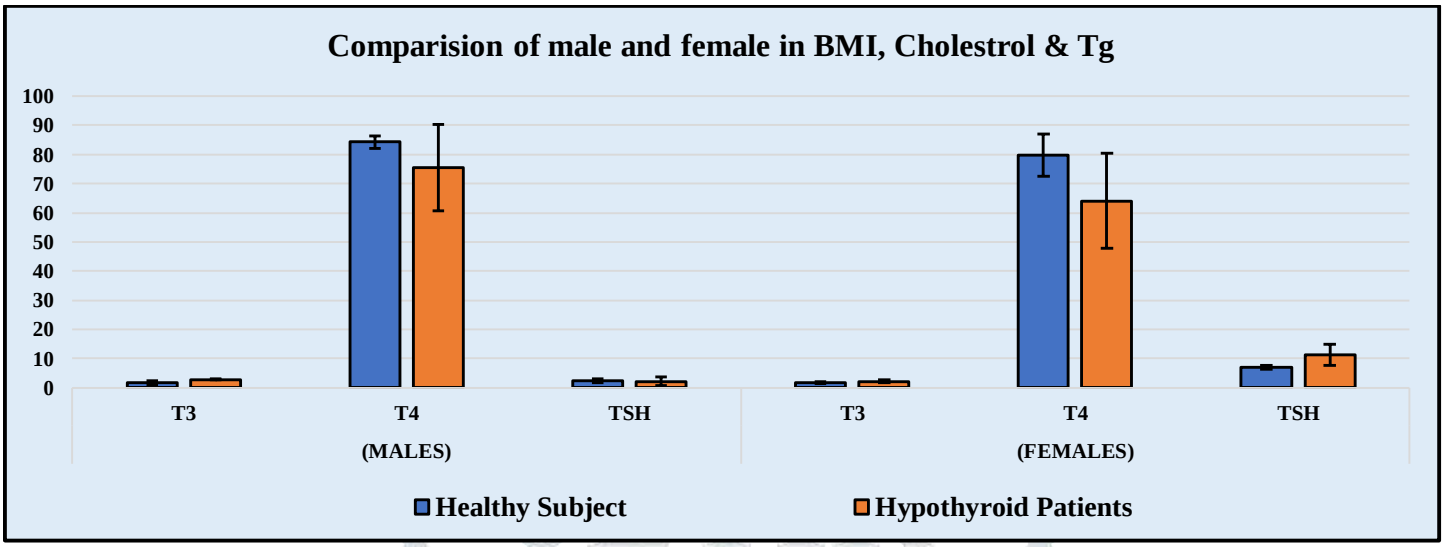
Table 2: Data of Age, Weight, and BMI of Controls, Perimenopausal and Postmenopausal women.

S. No.	Subjects	Weight (kgs)	Height (inches)	BMI (kg/m^2)
1	Controls	67.5±4.0	62.6±0.8	26.8±1.6
2	Perimenopausal women	64.8±3.2	61±0.8	26.8±1.1
3	Postmenopausal women	69.2±1.2	62.2±0.3	29.5±0.7

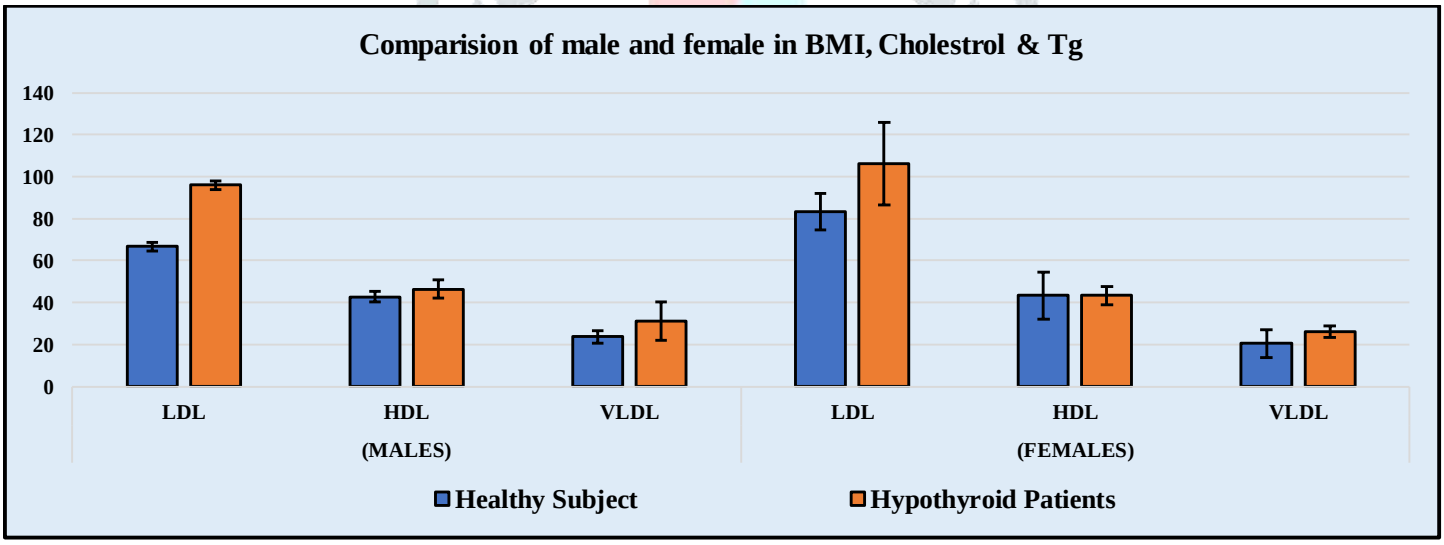
(Data represented as Mean ± SE)



The average value of BMI in Healthy subjects was (22.08±2.58 males) (23.56±1.0 females) and in hypothyroid patients was (29.74±0.77 males) (32.52±3.14 females). The average value of Cholestrol in hypothyroid patients was (149.20±7.12 males) (141.4±9.0) and in control was (185.96±8.80 males) (172.32±9.31 females) (p=0.0001) p<0.005. Furthermore, the mean value of Tg in healthy subjects was (125±9.80 males) (95.33±6.47 females) and in hypothyroid patients was (155.54±10.4 males) (130.56±9.60 females)



The average value of T3 in Healthy subjects was (16.8±0.63 males) (2.38±0.19 females) and in hypothyroid patients was (1.64±0.21 males) (2.11±0.58 females). The average value of T4 in hypothyroid patients was (79.73±14.8 males) (64.0±16.32) and in control was (84.34±2.13 males) (75.48±7.20 females) (p=0.0001) p<0.005. Furthermore, the mean value of TSH in healthy subjects was (2.17±0.67 males) (2.15±0.78 females) and in hypothyroid patients was (6.90±1.47 males) (11.14±3.60 females).



The average value of LDL in Healthy subjects was (66.7±2.20 males) (95.92±2.16 females) and in hypothyroid patients was (83.48±8.63 males) (106.38±19.63 females). The average value of HDL in hypothyroid patients was (46.32±4.37 males) (43.40±4.46) and in control was (42.80±2.59 males) (43.23.48±11.23 females) (p=0.0001) p<0.005. Furthermore, the mean value of VLDL in healthy subjects was (23.64±3.11 males) (20.46±6.64 females) and in hypothyroid patients was (31.08±9.15 males) (26.09±2.78 females).

CONCLUSION

It has been concluded from the study that Lipid parameters produced changes in blood constituents in hypothyroid condition. There are following parameters have been observed:

- The present study revealed that the Cholesterol level was observed significantly higher in Hypothyroid Males as compared to Hypothyroid Females.
- The prevalence of LDL, TG, and VLDL were seen higher in Hypothyroid Females in comparison to Hypothyroid Males.
- Increase in prevalence of the Metabolic Syndrome was higher in women than in men.
- This might be driven by the constant rise in Obesity in women.
- Thyroid Hormone might affect lipid metabolism and numerous studies have found that lipid levels increase as TSH levels increase.

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