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# A CROSS-SECTIONAL STUDY OF LIPID PROFILE PARAMETERS IN CASES OF THYROID DYSFUNCTION

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**ABSTRACT**

**Background and Objectives:** Thyroid disorders stand as prevalent endocrine maladies on a global scale. In orchestrating the synthesis, metabolism, and mobilization of lipids, thyroid hormones assume a pivotal role. Evidently, deviations in thyroid function may impart modifications upon circulating lipid levels. The principal objective of this investigation was to elucidate the nuances of lipid level perturbations associated with thyroid dysfunction.

**Material and Methods:** This study adopted a cross-sectional observational design. The cohort under scrutiny encompassed 30 individuals afflicted with hypothyroidism, an equal number beset by hyperthyroidism, alongside 30 euthyroid counterparts. Through estimation and subsequent juxtaposition, levels of total cholesterol, triglycerides, high-density lipoprotein (HDL), very low-density lipoprotein (VLDL), low-density lipoprotein (LDL), and the LDL/HDL ratio were meticulously scrutinized.

**Results:** Within the hypothyroidism stratum, a discernible elevation materialized in total cholesterol, LDL-C, and triglyceride levels, accompanied by a concomitant reduction in HDL-C concentrations. Conversely, hyperthyroidism heralded a statistically significant reduction across total cholesterol, triglyceride, LDL, VLDL levels, along with a diminished LDL/HDL ratio.

**Conclusion:** Aberrations in thyroid function wield the potential to exert profound modifications upon the lipid profile. It is noteworthy that hypothyroidism emerges as a notable risk factor for cardiovascular ailments. In light of this, the systematic inclusion of thyroid hormone evaluation within routine screening protocols assumes paramount importance, offering a window for timely intervention and management of cardiac ailments precipitated by thyroid dysfunction.

**Key words:** Preanesthetic Medication, Alprazolam, Pantoprazole, Antiemetics.

## INTRODUCTION

Thyroid disorders constitute a prevailing category of endocrine irregularities, and this phenomenon holds true on a global scale, including India. These disorders set themselves apart by virtue of their distinctive attributes such as diagnostic accessibility, medical intervention feasibility, and the discernible visibility that even a minor thyroid swelling proffers to the attending clinician. Timely diagnosis and therapeutic intervention stand as pivotal cornerstones in the management of these conditions [1].

The ramifications of fluctuations in thyroid hormone concentrations reverberate across a myriad of physiological realms encompassing oxygen consumption, temperature regulation, developmental progression, neural function, and the responsiveness to other hormonal cues. Moreover, these hormones orchestrate the metabolism of proteins, lipids, carbohydrates, nucleic acids, vitamins, and inorganic ions [2]. The excretion of substantial quantities of these hormones precipitates an elevation in basal metabolic rate. Concomitantly, the pace of nutrient utilization for energy generation undergoes substantial acceleration, accompanied by an augmentation in protein synthesis. Collectively, these factors culminate in a generalized augmentation of bodily functional activity. It's noteworthy that thyroid hormones wield both global and specific influences on growth processes [3].

Lipid metabolism undergoes notable transformation under the aegis of thyroid hormones. These hormones expedite the mobilization of adipose tissue fat deposits, consequently heightening fatty acid plasma concentrations and promoting the accelerated oxidation of free fatty acids by cells [3]. In tandem, thyroid diseases disrupt the

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composition and transportation of lipoproteins within the plasma, culminating in distinctive shifts in plasma lipid profiles, the extent of which is contingent on the nature and duration of thyroid dysfunction [4-6].

Significant discourse in the scientific milieu has postulated an escalated propensity for coronary artery disease among individuals afflicted by hypothyroidism, owing to alterations in lipoprotein profiles with atherogenic implications [7]. In the context of hypothyroidism, a diminution in high-density lipoprotein cholesterol (HDL-C) manifests, while hyperthyroidism engenders augmented cholesterol excretion and accelerated low-density lipoprotein (LDL) turnover, thereby yielding decreased total cholesterol and LDL cholesterol levels [5, 8].

Thyroid hormones exert a pronounced influence on triglyceride turnover and chylomicron clearance, concurrently modulating hepatic lipogenesis in hyperthyroidism and hypothyroidism. Thyroid hormones mitigate hepatic total cholesterol and very low-density lipoprotein (VLDL) production through reduced re-esterification coupled with increased oxidation of nascent fatty acids. Hypothyroid patients exhibit heightened VLDL secretion from the liver [8]. Concurring with these observations, prior studies have reported elevated plasma triglyceride concentrations in hypothyroidism.The ratio of LDL to high-density lipoprotein (HDL) assumes an elevated stance in hypothyroidism and inversely, exhibits reduced levels in hyperthyroidism [10, 11].

The well-established association between elevated lipid levels and the propensity for coronary artery disease underscores the import of this lipid-laden discourse [12]. Consequently, the present study was undertaken to explicate the shifts in lipid profiles that materialize in the context of thyroid dysfunction. This endeavor endeavors to proffer insights into early detection of lipid anomalies in hypothyroid and hyperthyroid individuals, thereby envisaging a tangible reduction in the burden of mortality and morbidity engendered by these pathological statess.

## MATERIAL & METHODS

The participant pool consisted of a total of 90 subjects, comprising three distinct groups: 30 patients afflicted with hypothyroidism (referred to as the hypothyroid group), 30 patients with hyperthyroidism (designated as the hyperthyroid group), and 30 individuals demonstrating normal thyroid function (referred to as the control group).

Enrollment into the hypothyroid and hyperthyroid groups comprised patients diagnosed with hypothyroidism or hyperthyroidism within the age bracket of 30–60 years, who had undergone treatment for a period of one year or more. These individuals were drawn from both medical and surgical outpatient departments, as well as the respective wards. The control group encompassed age-matched, healthy adults with verified normal thyroid function.

Inclusion Criteria:
- Hypothyroid Group: Patients within the age range of 30–60, with diagnosed hypothyroidism and under treatment for at least one year.
- Hyperthyroid Group: Patients within the age range of 30–60, with diagnosed hyperthyroidism and under treatment for at least one year.
- Control Group: Healthy adults within the same age range, exhibiting normal thyroid function.

Exclusion Criteria:
Subjects with documented history of other systemic or infectious diseases were excluded from the study.

Comprehensive patient history was meticulously collected and documented. Routine blood pressure assessments and systemic examinations were conducted across all participant groups. Thyroid hormone and blood lipid level assessments were performed on all patients and control subjects. Classification into distinct groups was carried out based on clinical evaluations and thyroid function test outcomes. The quantification of T3, T4, and TSH was performed through radioimmunoassay. Concomitantly, levels of total cholesterol, triglycerides, high-density lipoprotein (HDL), very low-density lipoprotein (VLDL), low-density lipoprotein (LDL), and the LDL/HDL ratio were estimated within all three aforementioned groups.

## RESULTS

The outcomes of the current study are displayed in Tables 1 and 2.

**Table 1: Lipid profile parameters in hypothyroid and control group**

| Parameters (mg/dl) | Hypothyroid Group (n=30) | Control Group (n=30) | P-value |
|---|---|---|---|
| Total cholesterol | 273.10 ± 47.82 | 169.45 ± 20.10 | <0.05 |
| Triglycerides | 155.20 ± 33.55 | 86.72 ± 9.88 | <0.05 |
| VLDL | 29.41 ± 6.98 | 19.15 ± 2.23 | <0.05 |

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|---|---|---|---|
| LDL | 213.05 ± 42.17 | 113.76 ± 22.46 | <0.05 |
| HDL | 24.80 ± 4.96 | 47.92 ± 8.62 | <0.05 |
| LDL: HDL ratio | 8.73 ± 2.87 | 2.27 ± 0.75 | <0.05 |

**Table 2: Lipid profile parameters in hyperthyroid and control group**

| Parameters (mg/dl) | Hyperthyroid Group (n=30) | Control Group (n=30) | P-value |
|---|---|---|---|
| Total cholesterol | 139.82 ± 7.15 | 175.49 ± 18.99 | <0.05 |
| Triglycerides | 86.17 ± 4.38 | 87.95 ± 10.09 | <0.05 |
| VLDL | 16.28 ± 0.83 | 17.41 ± 1.97 | <0.05 |
| LDL | 78.52 ± 9.07 | 106.63 ± 21.14 | <0.05 |
| HDL | 50.12 ± 4.94 | 46.27 ± 7.86 | <0.05 |
| LDL: HDL ratio | 1.60 ± 0.33 | 2.47 ± 0.80 | <0.05 |

## DISCUSSION

The average serum total cholesterol levels observed in the hypothyroid group during the current study exhibited a statistically significant elevation in comparison to the control group. A comparable surge in total cholesterol levels was reported in previous studies [13-15]. The underlying cause for this rise might be attributed to the modified functioning of hepatic lipase. Notably, a decrease in the activity of lipoprotein lipase has been associated with hypothyroidism [10].

The average serum triglyceride level within the hypothyroid group was observed to be significantly higher in comparison to that of the normal control group. Similar trends were reported in the previous investigations [10, 15]. The reduced activity of lipoprotein lipase is speculated to underlie the increased triglyceride levels in the hypothyroid group [10]. In individuals with hypothyroidism, the synthesis of plasma triglycerides remains unchanged; however, the fractional clearance of both endogenous and exogenous triglycerides is substantially diminished. This alteration appears to be a contributing factor to the manifestation of hypertriglyceridemia in these individuals [11].

In this study, a noteworthy decline in the levels of HDL was observed within the hypothyroid group in comparison to the control group. Similar outcomes were documented by Agdeppa et al. [11] and Bauer et al. [12] The reduction in HDL levels could potentially be attributed to the diminished synthesis, mobilization, and degradation of lipids that often manifest in the context of hypothyroidism [11]. Conversely, the VLDL levels among individuals in the hypothyroid group were notably higher compared to those in the control group. These findings align with the conclusions drawn by Berthezene et al. [16]. Typically, in hypothyroidism, the catabolism of VLDL undergoes a deceleration. These variations in VLDL dynamics are intricately linked to shifts in the activity of lipoprotein lipase and hepatic lipase, in addition to structural alterations occurring within the VLDL particles themselves [16].

The mean LDL values within the hypothyroid group exhibited a statistically significant elevation in comparison to the control group. This might be attributed to a diminished fractional clearance of LDL particles. This attenuation could arise from a decreased count of LDL receptors present in the liver [5]. These outcomes align with the observations made in the study conducted by Agdeppa et al [11]. Furthermore, the mean LDL-C/HDL-C ratio within the hypothyroid group was found to be notably higher than that within the control group. These results correspond with the findings reported by Bauer et al. [12]. The mean value of the LDL: HDL ratio within the hyperthyroid group exhibited a statistically significant reduction in comparison to the control group. This ratio demonstrated a noteworthy decrease in the hyperthyroid group. These findings are consistent with the observations made by Bauer et al. [12].

## CONCLUSION

Dysregulated thyroid function has the potential to induce noteworthy alterations in the lipid profile, a phenomenon that holds paramount importance as a risk factor for cardiovascular diseases. Therefore, the systematic assessment of thyroid hormones through routine screening could yield substantial benefits in terms of early intervention and the management of cardiac disorders linked to thyroid dysfunction. It is worth noting that the precise targeting of thyroid hormone analogues may offer a promising avenue for addressing hyperlipidemia while circumventing the onset of systemic thyrotoxicosis.

**Conflicts of interest:** none

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