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# ALTERATIONS IN LIPID AND CARBOHYDRATE METABOLISM IN PATIENTS WITH HYPOTHYROIDISM: A COMPREHENSIVE CLINICAL ANALYSIS

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**Abstract:** Hypothyroidism is associated with significant metabolic disturbances, particularly in lipid and carbohydrate metabolism. This study provides a comprehensive clinical analysis of metabolic alterations observed in patients with overt and subclinical hypothyroidism. Findings indicate that thyroid hormone deficiency leads to dyslipidemia, insulin resistance, impaired glucose utilization, and increased cardiometabolic risk. Understanding the metabolic profiles of hypothyroid patients is essential for improving diagnostic accuracy and optimizing therapeutic strategies.

**Keywords:** hypothyroidism, lipid metabolism, carbohydrate metabolism, dyslipidemia, insulin resistance, metabolic disorders.

## Introduction

Hypothyroidism is a common endocrine disorder characterized by insufficient production of thyroid hormones, which play a pivotal role in regulating basal metabolic rate, lipid homeostasis, and glucose metabolism [1]. Thyroid hormones—particularly triiodothyronine (T3)—modulate mitochondrial oxidative activity, hepatic lipid processing, and insulin-mediated glucose uptake. Consequently, even mild reductions in thyroid hormone levels may trigger broad metabolic disturbances, including dyslipidemia, insulin resistance, and altered carbohydrate utilization [2,3].

Alterations in lipid metabolism are among the most widely documented metabolic consequences of hypothyroidism. Thyroid hormone deficiency reduces the activity of LDL receptors, decreases lipoprotein lipase function, and impairs cholesterol clearance, resulting in increased total cholesterol, LDL-C, and triglyceride concentrations [4,5]. Several clinical studies have shown that patients with both overt and subclinical hypothyroidism exhibit atherogenic lipid profiles, contributing significantly to elevated cardiovascular risk [6]. Moreover, low T3 levels reduce hepatic β-oxidation and promote hepatic lipid accumulation, further exacerbating dyslipidemia [7].

Carbohydrate metabolism is similarly affected by thyroid dysfunction. Thyroid hormones facilitate glucose uptake through the regulation of GLUT-4 transporters and enhance hepatic gluconeogenesis. Their deficiency leads to decreased insulin sensitivity, impaired glycogenolysis, and increased fasting insulin levels [8]. Studies have demonstrated a strong association between elevated TSH levels and insulin resistance, even in patients with subclinical hypothyroidism, suggesting that early thyroid dysfunction may precede or worsen metabolic syndrome components [9].

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Given the interconnected nature of endocrine and metabolic pathways, the clinical significance of metabolic alterations in hypothyroidism extends beyond simple biochemical abnormalities. Untreated or inadequately treated hypothyroidism increases long-term risks of obesity, metabolic syndrome, and cardiovascular diseases [10]. Therefore, a comprehensive assessment of lipid and carbohydrate metabolism in hypothyroid patients is essential for improving diagnostic accuracy and therapeutic strategies.

This study aims to provide a detailed clinical analysis of the alterations in lipid and carbohydrate metabolism among patients with hypothyroidism and to highlight their pathophysiological importance.

**Literature Review**

Hypothyroidism is widely recognized as a disorder with extensive metabolic consequences, and numerous studies have documented its effects on lipid and carbohydrate metabolism. Early foundational research established that thyroid hormones regulate basal metabolic rate, lipid turnover, and glucose utilization through their action on nuclear receptors and mitochondrial activity (Mullur et al., 2014). These hormones stimulate lipolysis, enhance LDL receptor activity, and promote glucose transport; therefore, their deficiency inevitably leads to metabolic dysfunction.

A large body of literature demonstrates that the most consistent metabolic abnormality in hypothyroidism is dyslipidemia. Peeters (2017) reported that serum LDL cholesterol and total cholesterol levels tend to rise proportionally with TSH elevation, even before overt hypothyroidism develops. Similarly, Duntas and Brenta (2012) and Rizos et al. (2011) found that decreased thyroid hormone activity results in impaired hepatic clearance of LDL particles and reduced lipoprotein lipase activity, causing accumulation of atherogenic lipoproteins. These findings have been further supported by Pucci et al. (2000), who emphasized that thyroid hormone deficiency disrupts several biochemical pathways responsible for lipid mobilization, leading to hypercholesterolemia and increased triglyceride levels. Sinha et al. (2014) added that hypothyroidism affects mitochondrial β-oxidation in the liver, contributing to hepatic lipid accumulation and worsening lipid profiles.

The effect of hypothyroidism on carbohydrate metabolism has also been well documented. Maratou et al. (2009) demonstrated that thyroid hormone deficiency is associated with decreased insulin sensitivity in peripheral tissues. Their studies illustrated impaired glucose transport and reduced insulin signaling as key contributors to increased insulin resistance. Pearce (2012) found that even subclinical hypothyroidism is associated with higher HOMA-IR scores, indicating that early thyroid dysfunction may negatively influence carbohydrate metabolism before overt symptoms appear. Jabbar et al. (2017) strengthened this observation by showing that disrupted carbohydrate homeostasis in hypothyroidism increases cardiovascular risk, independent of lipid abnormalities.

Comparative studies have also highlighted differences between overt and subclinical hypothyroidism. Chaker et al. (2017) reported that metabolic disturbances become progressively more severe as thyroid hormone levels decline, with overt hypothyroidism

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showing more pronounced dyslipidemia and insulin resistance. These findings underline that metabolic impairment occurs along a continuum and may begin during the subclinical phase.

Overall, the available literature provides strong evidence that thyroid hormone deficiency significantly alters both lipid and carbohydrate metabolism. These metabolic changes contribute to increased cardiovascular morbidity and highlight the importance of early detection and intervention. Current research continues to explore molecular mechanisms linking thyroid function to metabolic health, but existing data already emphasize the central metabolic role of thyroid hormones.

## Materials and Methods

This study was conducted to evaluate changes in lipid and carbohydrate metabolism in patients with hypothyroidism. A cross-sectional design was used, and participants were selected from adults aged 18 to 65 years who underwent thyroid function testing. The study population included two groups of patients diagnosed with overt or subclinical hypothyroidism, as well as a control group of healthy euthyroid individuals.

Patients were included if they had elevated TSH levels consistent with overt or subclinical hypothyroidism. Those with diabetes, kidney or liver diseases, pregnancy, or who were taking medications affecting lipid or glucose levels were excluded from the study. All participants provided informed consent.

Blood samples were collected after an overnight fast. Thyroid function parameters, including TSH, free T4, and free T3, were measured using standard immunoassay methods. Lipid profile tests included total cholesterol, LDL cholesterol, HDL cholesterol, and triglycerides. Fasting glucose and fasting insulin were also measured, and insulin resistance was calculated using the HOMA-IR formula.

Anthropometric measurements such as body mass index and waist circumference were recorded. Blood pressure and basic clinical data were also obtained. Statistical analysis was performed to compare metabolic indices between the groups. Mean values and standard deviations were calculated, and differences between groups were assessed. A significance level of $p < 0.05$ was used for statistical comparisons.

## Results

The study included patients with overt hypothyroidism, subclinical hypothyroidism, and healthy controls. Thyroid function tests confirmed that patients with overt hypothyroidism had significantly higher TSH levels and lower free T4 values compared with the other groups, while subclinical hypothyroidism showed elevated TSH with normal free T4.

Lipid metabolism showed noticeable alterations in both overt and subclinical hypothyroidism. Total cholesterol and LDL cholesterol levels were significantly higher in overt hypothyroid patients than in the control group. Triglyceride levels were also elevated in both hypothyroid groups, while HDL cholesterol levels were lower, especially in overt hypothyroidism. These findings indicate a clear pattern of dyslipidemia associated with reduced thyroid hormone levels.

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Carbohydrate metabolism was also affected. Fasting insulin levels were higher in both hypothyroid groups compared with controls, and HOMA-IR values showed increased insulin resistance. Fasting glucose values were slightly higher but generally remained within borderline ranges. Insulin resistance was more pronounced in overt hypothyroidism, suggesting a stronger metabolic effect in more severe thyroid dysfunction.

Correlation analysis showed positive relationships between TSH levels and several metabolic markers. Higher TSH levels were associated with higher total cholesterol, higher LDL cholesterol, and increased HOMA-IR values, indicating that worsening thyroid function contributes directly to metabolic abnormalities.

Overall, the results show that hypothyroidism leads to significant changes in lipid and carbohydrate metabolism, with more severe alterations occurring in overt cases.

**Discussion**

The results demonstrate that hypothyroidism exerts profound effects on lipid and carbohydrate metabolism. Thyroid hormones play a central role in regulating hepatic lipid processing, lipoprotein synthesis, and cholesterol transport. Their deficiency results in dyslipidemia characterized by elevated LDL-C, total cholesterol, and triglycerides. Reduced activity of hepatic lipase and LDL receptors explains the persistence of lipid abnormalities even in subclinical hypothyroidism.

Carbohydrate metabolism is similarly affected, as thyroid hormones modulate insulin-mediated glucose uptake and mitochondrial oxidative capacity. Insulin resistance observed in hypothyroid patients may be attributed to reduced glucose transport, impaired glycogenolysis, and increased peripheral adiposity.

The findings emphasize that metabolic abnormalities occur even in the early stages of thyroid dysfunction, reinforcing the need for metabolic screening in hypothyroid patients. Early detection and treatment with levothyroxine may reverse many metabolic disturbances and reduce long-term cardiovascular risk.

The findings of this study show that hypothyroidism has a clear and measurable impact on both lipid and carbohydrate metabolism. The increase in total cholesterol, LDL cholesterol, and triglycerides observed in hypothyroid patients supports the well-known idea that thyroid hormones play an important role in lipid regulation. Reduced levels of thyroid hormones slow down the breakdown and clearance of lipids, which leads to the accumulation of cholesterol and triglycerides in the bloodstream. The decrease in HDL cholesterol further strengthens the link between hypothyroidism and an atherogenic lipid profile.

Carbohydrate metabolism was also affected. The rise in fasting insulin and increased HOMA-IR values indicate that hypothyroid patients develop insulin resistance. This may be due to reduced glucose uptake in tissues and slower metabolic processes in the absence of adequate thyroid hormones. Although fasting glucose levels did not show major changes, signs of insulin resistance suggest that carbohydrate metabolism is disrupted even in early stages of thyroid dysfunction.

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The comparison between overt and subclinical hypothyroidism further highlights that metabolic abnormalities worsen as thyroid hormone levels continue to fall. Overt hypothyroidism showed stronger changes in both lipid and carbohydrate metabolism, confirming that the severity of thyroid dysfunction is directly related to the degree of metabolic impairment.

The correlation between high TSH levels and abnormal metabolic markers supports the idea that thyroid dysfunction contributes to the development of cardiovascular and metabolic diseases. Elevated TSH may be an early indicator of future metabolic complications, even before major clinical symptoms appear.

These findings are in line with previous research showing that thyroid hormones affect liver function, lipid transport, and glucose metabolism. Early identification and treatment of hypothyroidism may therefore help prevent long-term complications such as atherosclerosis, obesity, metabolic syndrome, and cardiovascular disease.

**Conclusion**

The results of this study demonstrate that hypothyroidism leads to significant and clinically important disturbances in both lipid and carbohydrate metabolism. Patients with overt and subclinical thyroid hormone deficiency show clear patterns of dyslipidemia, including increased total cholesterol, elevated LDL cholesterol, higher triglyceride levels, and reduced HDL cholesterol. These metabolic changes reflect the essential role of thyroid hormones in lipid breakdown, cholesterol clearance, and hepatic lipid regulation. The findings confirm that even mild reductions in thyroid hormone levels are sufficient to disrupt lipid homeostasis and create a metabolic environment that favors the development of atherosclerosis and cardiovascular disease.

Carbohydrate metabolism also shows noticeable impairment in hypothyroid patients. The presence of elevated fasting insulin and increased insulin resistance suggests that thyroid hormone deficiency reduces the body's ability to utilize glucose effectively. Although fasting glucose levels remained near normal limits, the rise in insulin resistance highlights that metabolic imbalance develops earlier than overt hyperglycemia. This indicates that hypothyroidism contributes to the early stages of metabolic dysfunction and may increase the risk of future metabolic syndrome.

A comparison between overt and subclinical hypothyroidism reveals that metabolic abnormalities intensify as the severity of thyroid dysfunction increases. Patients with overt hypothyroidism displayed more pronounced lipid and carbohydrate disturbances, supporting the conclusion that impaired thyroid function and metabolic alteration are closely linked. The positive correlations between TSH levels and abnormal metabolic parameters further suggest that thyroid dysfunction may serve as a predictor of cardiometabolic risk.

These findings emphasize the importance of evaluating metabolic parameters in patients with thyroid disorders. Early detection of dyslipidemia and insulin resistance in hypothyroid individuals may prevent long-term complications. Appropriate thyroid hormone replacement therapy, combined with lifestyle intervention and regular monitoring of lipid and carbohydrate indices, can significantly improve metabolic outcomes.

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In conclusion, thyroid hormone deficiency should be recognized as an important factor contributing to metabolic imbalance. Addressing hypothyroidism not only corrects hormonal deficiency but may also help reduce cardiovascular risk, improve metabolic health, and prevent progression to more severe endocrine and metabolic disorders. Integrating thyroid evaluation into metabolic assessments could therefore enhance patient outcomes and support more effective clinical management strategies.

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