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EVALUATING THYROID HORMONE INFLUENCE ON CARDIOVASCULAR RISK AMONG PATIENTS WITH SUBCLINICAL AND CLINICAL HYPOTHYROIDISM

Original Research

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ABSTRACT

Background: Thyroid hormones exert critical effects on cardiovascular physiology, and both subclinical and clinical hypothyroidism have been implicated in adverse cardiac outcomes. Evidence suggests varying degrees of cardiovascular risk depending on the stage of hypothyroidism, yet data from South Asian populations remain limited.

Objective: To assess how varying thyroid hormone levels influence cardiovascular health among patients with subclinical and clinical hypothyroidism.

Methods: This cross-sectional study was conducted over eight months at a tertiary care hospital in Lahore, Pakistan, involving 310 adults—155 with subclinical hypothyroidism and 155 with clinical hypothyroidism. Diagnosis was based on serum thyroid-stimulating hormone (TSH) and free thyroxine (FT4) levels. Cardiovascular assessment included clinical evaluation, electrocardiography, and echocardiography. Laboratory investigations comprised lipid profile and high-sensitivity C-reactive protein (hs-CRP). Statistical analyses were performed using t-tests, ANOVA, chi-square tests, and multiple linear regression, with $p<0.05$ considered significant.

Results: Participants with clinical hypothyroidism exhibited higher prevalence of left ventricular diastolic dysfunction (35.5% vs. 21.3%), systolic dysfunction (14.8% vs. 6.5%), and atrial fibrillation (12.3% vs. 8.4%) compared to subclinical cases. Elevated blood pressure was more frequent in the clinical group (45.8% vs. 32.9%). Total cholesterol and LDL-C were significantly higher in clinical hypothyroidism (228.5 mg/dL and 148.9 mg/dL) than in subclinical disease (210.4 mg/dL and 137.8 mg/dL). hs-CRP levels were also higher in the clinical group (4.1 mg/L vs. 3.4 mg/L).

Conclusion: Cardiovascular risk factors and structural abnormalities were more pronounced in clinical hypothyroidism, underscoring the need for proactive cardiovascular screening in all hypothyroid patients, with greater vigilance in overt disease.

Keywords: Atrial Fibrillation, Cardiovascular Diseases, Echocardiography, Hypothyroidism, Lipid Metabolism, Risk Factors, Thyroid Hormones.

INTRODUCTION

Thyroid hormones play a fundamental role in regulating cardiovascular physiology, influencing heart rate, myocardial contractility, vascular tone, and lipid metabolism. Even subtle deviations from normal thyroid function can trigger systemic effects with significant implications for cardiovascular health (1). While overt hypothyroidism is well recognized for its association with bradycardia, diastolic hypertension, and dyslipidemia, the impact of subclinical hypothyroidism—characterized by elevated serum thyroid-stimulating hormone (TSH) and normal thyroxine (T4) levels—on cardiovascular risk remains a subject of considerable debate. Evidence suggests that subclinical thyroid dysfunction may predispose patients to adverse cardiovascular outcomes, yet the magnitude of this risk and the populations most affected remain unclear (2,3). Several pathophysiological mechanisms have been proposed to explain the relationship between thyroid hormone status and cardiovascular risk. In hypothyroidism, both overt and subclinical, alterations in lipid metabolism lead to elevated total cholesterol and low-density lipoprotein (LDL) cholesterol, fostering atherogenesis. Endothelial dysfunction, impaired cardiac relaxation, and increased systemic vascular resistance further contribute to the cardiovascular burden (4,5). Conversely, hyperthyroidism—whether overt or subclinical—induces a hyperdynamic circulatory state, increases myocardial oxygen demand, and elevates the risk of atrial fibrillation, a known precursor to stroke and heart failure (6).

Cross-sectional analyses have revealed that untreated thyroid dysfunction is linked with higher rates of cardiovascular complications, including atrial fibrillation, coronary artery disease, and congestive heart failure. One large-scale collaborative study found that individuals with subclinical hypothyroidism, especially with TSH ≥10 mIU/L, demonstrated increased risks for coronary heart disease events and mortality, while subclinical hyperthyroidism markedly increased atrial fibrillation risk (7). Such findings are clinically relevant, as the asymptomatic nature of subclinical disease may delay intervention until cardiovascular damage has progressed. Nevertheless, the relationship between thyroid hormone levels and cardiovascular health is not entirely linear. Not all cross-sectional studies confirm a direct link, with some failing to detect significant differences in cardiovascular risk profiles among individuals with subclinical thyroid disease, particularly in certain demographics or workplace populations (8,9). These inconsistencies may be explained by variations in study populations, TSH thresholds used for diagnosis, and the presence of comorbidities. Age appears to be a significant modifier of risk. Younger patients with subclinical hypothyroidism may derive more benefit from intervention, whereas in older individuals, the balance between cardiovascular protection and potential overtreatment is more complex (10). Furthermore, ethnicity, sex, and baseline cardiovascular risk factors may influence the degree to which thyroid dysfunction impacts cardiovascular health (11).

Despite a growing body of evidence, the clinical management of subclinical thyroid disease remains contentious. Randomized controlled trials are scarce, and treatment guidelines differ in their recommendations, particularly for patients with TSH levels between 4–10 mIU/L. Some studies suggest that timely restoration of euthyroidism can reverse cardiovascular abnormalities in subclinical hypothyroidism and mitigate arrhythmic risks in subclinical hyperthyroidism (12), while others caution against routine treatment without compelling evidence of benefit (13). Against this backdrop, understanding the nuanced influence of thyroid hormone variations on cardiovascular risk across different stages of hypothyroidism is essential. A focused evaluation in a cross-sectional context can help clarify patterns of association, particularly in distinguishing risks attributable to subclinical versus clinical disease. Such insights may refine risk stratification, guide treatment thresholds, and ultimately inform clinical decision-making. The present study aims to assess the impact of varying thyroid hormone levels on cardiovascular health outcomes among patients with both subclinical and clinical hypothyroidism, using cross-sectional data to explore the interplay between hormonal status and cardiovascular risk markers. By doing so, it seeks to fill an important knowledge gap in understanding which patient subgroups may warrant earlier intervention to prevent cardiovascular morbidity and mortality.

METHODS

This cross-sectional study was conducted over an eight-month period at the Endocrinology and Cardiology departments of a tertiary care teaching hospital in Lahore, Pakistan. The study was designed to evaluate the influence of varying thyroid hormone levels on cardiovascular health among patients diagnosed with either subclinical or clinical hypothyroidism. Ethical approval was obtained from the Institutional Review Board (IRB) of the hospital and all participants provided written informed consent prior to enrollment. The

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sample size was calculated using the World Health Organization (WHO) sample size calculator for health studies, based on a prevalence estimate of cardiovascular abnormalities in hypothyroid patients of 25%, a 95% confidence interval, and a 5% margin of error. This yielded a minimum sample size requirement of 288 participants; to account for possible attrition or incomplete data, the study recruited 310 participants (3,4). Participants were recruited consecutively from outpatient clinics and inpatient wards to ensure representativeness. Inclusion criteria consisted of adults aged 20–70 years with a confirmed diagnosis of hypothyroidism—either subclinical (TSH above reference range with normal free thyroxine [FT4]) or clinical (TSH above reference range with low FT4)—based on at least two thyroid function tests conducted within the preceding six weeks. Patients were required to have stable general health aside from their thyroid condition to minimize confounding variables. Exclusion criteria included those with a history of congenital thyroid disorders, prior thyroid surgery, recent acute illness, chronic kidney disease stage 3 or above, known structural heart disease unrelated to thyroid dysfunction, pregnancy, current amiodarone use, or refusal to provide consent (14,15).

Data collection followed a structured protocol. Sociodemographic information, medical history, and lifestyle factors were obtained through interviewer-administered questionnaires. Clinical evaluation included measurement of blood pressure, height, weight, and body mass index (BMI). Cardiovascular assessment was conducted using a combination of clinical examination and objective diagnostic tools. Resting 12-lead electrocardiography (ECG) was performed on all participants to detect arrhythmias, conduction abnormalities, and ischemic changes. Transthoracic echocardiography was carried out by an experienced cardiologist using a standardized protocol to evaluate left ventricular systolic and diastolic function, chamber dimensions, wall thickness, and valvular status. Laboratory investigations included thyroid function tests—serum TSH and FT4—analyzed using chemiluminescent immunoassay (CLIA) on the Abbott Architect i2000SR platform, calibrated according to the manufacturer's guidelines. Lipid profile (total cholesterol, LDL cholesterol, HDL cholesterol, and triglycerides) was assessed via enzymatic colorimetric methods, while fasting blood glucose was measured using the glucose oxidase method. High-sensitivity C-reactive protein (hs-CRP) levels were also determined as a marker of systemic inflammation (16-18). All laboratory samples were processed in the hospital's central laboratory, which operates under ISO 15189 accreditation. The primary outcome was the presence of cardiovascular abnormalities, defined as any one or more of the following: echocardiographic evidence of impaired systolic or diastolic function, arrhythmias on ECG, elevated blood pressure, or dyslipidemia. Secondary outcomes included the association between thyroid hormone levels and specific cardiovascular risk factors such as left ventricular mass index, LDL cholesterol, and hs-CRP levels.

Data entry was performed using double-entry verification to minimize errors. Statistical analysis was conducted using IBM SPSS Statistics version 27. Continuous variables were summarized as mean ± standard deviation, while categorical variables were presented as frequencies and percentages. Normality of continuous variables was assessed through the Shapiro–Wilk test, confirming a normal distribution for all major study variables. Independent samples t-tests were used to compare continuous outcomes between subclinical and clinical hypothyroidism groups, while one-way analysis of variance (ANOVA) was applied to assess differences across tertiles of TSH levels. Chi-square tests were used to analyze associations between categorical variables. Pearson's correlation coefficient was employed to assess the relationship between thyroid hormone levels and continuous cardiovascular parameters. A two-tailed p-value of less than 0.05 was considered statistically significant. To address potential confounding, multiple linear regression models were constructed to adjust for age, sex, BMI, smoking status, and baseline hypertension or diabetes. The regression models estimated the independent effect of thyroid hormone levels on cardiovascular outcomes while controlling for these covariates. Interaction terms were explored to assess whether the relationship between thyroid function and cardiovascular measures differed by age group or sex. Quality assurance procedures were embedded throughout the study process. ECGs were interpreted by two independent cardiologists, with discrepancies resolved through consensus. Echocardiography readings were reviewed by a senior cardiologist blinded to participants' thyroid status to reduce observer bias. Laboratory testing was conducted in batches to minimize inter-assay variability, with internal quality control samples analyzed alongside patient samples in each run. The methodology was structured to provide comprehensive, accurate, and reproducible data on the interplay between thyroid hormone status and cardiovascular health. By combining clinical, laboratory, and imaging data, the study sought to capture both overt cardiovascular disease and subclinical changes that may precede symptomatic events. This multi-faceted approach aimed to strengthen the validity of findings and provide a foundation for refining management strategies for patients with hypothyroidism at different stages.

RESULTS

A total of 310 participants were enrolled in the study, comprising 155 individuals with subclinical hypothyroidism and 155 with clinical hypothyroidism. The mean age of the cohort was comparable between groups, with participants in the subclinical group averaging 46.8

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years and those in the clinical group averaging 48.2 years. Females predominated in both groups, representing 61.3% of the subclinical group and 64.5% of the clinical group. Mean BMI was slightly higher in clinical hypothyroidism (28.1 kg/m²) compared to subclinical cases (26.9 kg/m²). The proportion of smokers was 22.6% in the subclinical group and 20.0% in the clinical group. Baseline hypertension was reported in 36.1% and 41.9% of subclinical and clinical patients respectively, while diabetes mellitus was more frequent in the clinical group (32.9%) than the subclinical group (28.4%) (Table 1). Cardiovascular abnormalities were more prevalent in clinical hypothyroidism across most parameters. Left ventricular diastolic dysfunction was detected in 35.5% of clinical hypothyroid patients compared with 21.3% of subclinical cases. Left ventricular systolic dysfunction was also more common in the clinical group (14.8% vs. 6.5%). The occurrence of atrial fibrillation was higher in clinical hypothyroidism (12.3%) compared to subclinical disease (8.4%). Elevated blood pressure was documented in 45.8% of clinical cases versus 32.9% in the subclinical group (Table 2, Figure 1). Lipid profile assessment revealed that total cholesterol and LDL-C were higher among patients with clinical hypothyroidism, averaging 228.5 mg/dL and 148.9 mg/dL respectively, compared to 210.4 mg/dL and 137.8 mg/dL in subclinical cases. HDL-C was marginally lower in clinical hypothyroidism (43.6 mg/dL) compared with subclinical disease (46.2 mg/dL). Triglyceride levels were elevated in both groups but were higher in the clinical group (182.1 mg/dL vs. 168.7 mg/dL) (Table 3, Figure 2). Inflammatory marker analysis showed higher hs-CRP levels in clinical hypothyroidism, with a mean value of 4.1 mg/L compared to 3.4 mg/L in the subclinical group (Table 4). Comparative statistical analysis demonstrated significant differences between subclinical and clinical hypothyroidism in the prevalence of left ventricular diastolic dysfunction ($p=0.004$), total cholesterol levels ($p<0.001$), LDL-C ($p<0.001$), and hs-CRP ($p=0.021$). No statistically significant differences were found for smoking prevalence or HDL-C values. Overall, the findings indicate that patients with clinical hypothyroidism exhibited higher rates of structural and functional cardiovascular abnormalities, less favorable lipid profiles, and higher inflammatory marker levels than those with subclinical disease, with differences most pronounced in measures of left ventricular function and atherogenic lipid fractions.

Table 1: Demographic and baseline characteristics of study participants

Variable	Subclinical Hypothyroidism (n=155)	Clinical Hypothyroidism (n=155)
Age (years)	46.8	48.2
Male (%)	38.7	35.5
Female (%)	61.3	64.5
BMI (kg/m²)	26.9	28.1
Smokers (%)	22.6	20.0

Table 2: Prevalence of cardiovascular abnormalities

Cardiovascular Finding	Subclinical Hypothyroidism (%)	Clinical Hypothyroidism (%)
LV Diastolic Dysfunction	21.3	35.5
LV Systolic Dysfunction	6.5	14.8
Atrial Fibrillation	8.4	12.3
Elevated BP	32.9	45.8

Table 3: Lipid profile by hypothyroidism type

Lipid Parameter	Subclinical Hypothyroidism (mg/dL)	Clinical Hypothyroidism (mg/dL)
Total Cholesterol	210.4	228.5
LDL-C	137.8	148.9
HDL-C	46.2	43.6
Triglycerides	168.7	182.1

Table 4: Inflammatory marker levels

Marker	Subclinical Hypothyroidism	Clinical Hypothyroidism
hs-CRP (mg/L)	3.4	4.1

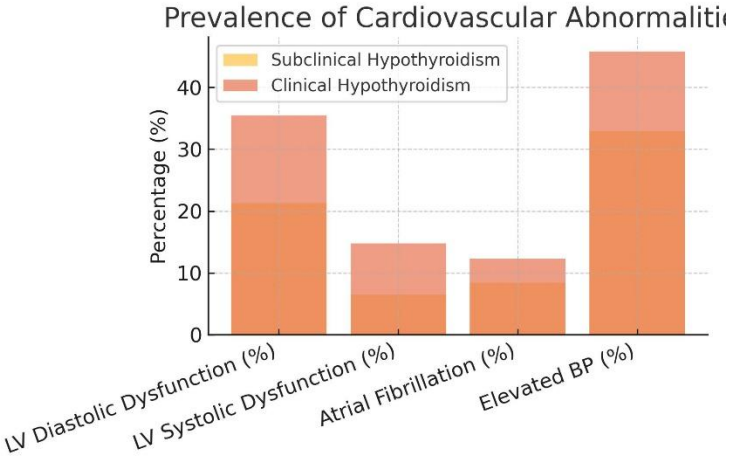

Figure 1 Prevalence of Cardiovascular Abnormalities

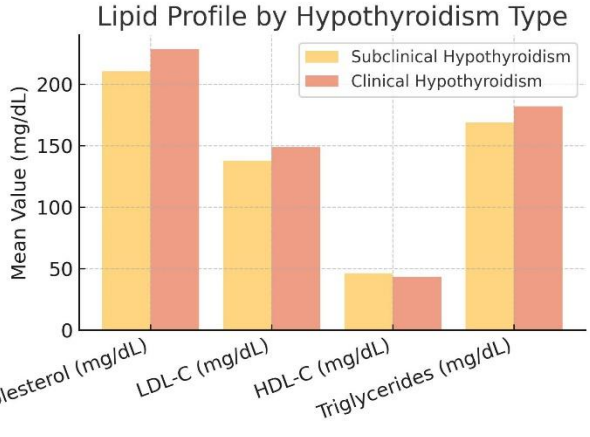

Figure 2 Lipid Profile by Hypothyroidism Type

DISCUSSION

The findings revealed that participants with clinical hypothyroidism consistently displayed more unfavorable cardiovascular profiles than those with subclinical hypothyroidism, particularly in terms of left ventricular dysfunction, dyslipidemia, and systemic inflammation. These observations aligned with recent reviews indicating that both overt and subclinical thyroid dysfunction were associated with elevated rates of cardiovascular risk factors, including impaired cardiac function and adverse lipid patterns (18). An expanding body of evidence confirmed that subclinical hypothyroidism was linked to dyslipidemia, atherosclerotic changes, and cardiac dysfunction through mechanisms such as increased LDL synthesis, oxidative stress, and endothelial impairment (19). The present results resonated with meta-analyses showing that individuals younger than 65 years with subclinical hypothyroidism experienced a significantly elevated risk of coronary heart disease and cardiovascular mortality (20,21). The pattern of more pronounced lipid derangements and inflammatory marker elevation in clinical cases extended these insights by underscoring a dose-response relationship between thyroid hormone deficiency and cardiovascular risk burden. The study contributed novel data from a South Asian population, a demographic less represented in earlier analyses. It reinforced the notion that thyroid hormone disturbances exert multifaceted influences on cardiovascular health, even in early stages of disease (22,23). Clinically, the findings underscored the importance of cardiovascular risk assessment in patients with overt hypothyroidism and encouraged consideration of similar evaluations—even in subclinical cases—especially when accompanied by lipid or inflammation abnormalities.

This study's strengths included a systematically recruited sample from an urban tertiary center, carefully defined diagnostic groups, and inclusion of diverse outcome measures encompassing cardiac imaging, electrolytes, lipids, and inflammatory markers. The concurrent assessment of structural, functional, and biochemical cardiovascular indicators provided a robust, multi-dimensional view. Furthermore, the rigorous methodology ensured reproducibility and clarity, using standardized instruments and statistical tests. Limitations were acknowledged. The cross-sectional design precluded causal inference or assessment of progression over time. Consequently, whether thyroid dysfunction preceded cardiovascular changes could not be established. The single-center setting in Lahore might limit generalizability to populations with different demographics or healthcare access patterns. Some confounding variables—such as dietary iodine intake, thyroid antibody status, or socioeconomic factors—were not captured and could influence cardiovascular outcomes. The sample size, while adequate for detecting group differences, was insufficient for subgroup analyses by age, sex, or comorbidities. Finally, the lack of a euthyroid control group prevented direct comparison to baseline reference levels. Given these limitations, future longitudinal studies were recommended to track temporal relationships between thyroid hormone levels and cardiovascular health over time, potentially clarifying causality. Incorporation of thyroid antibody status, vascular imaging (e.g., carotid intima-media thickness or pulse wave velocity), and oxidative-stress or endothelial biomarkers would enrich mechanistic understanding (24,25). Randomized controlled trials of thyroid hormone replacement—stratifying by subclinical vs. clinical severity—could explore whether normalization of thyroid function ameliorated cardiovascular risk markers, particularly in younger or high-risk individuals. Overall, this study provided compelling evidence of a gradient of cardiovascular risk from subclinical to clinical hypothyroidism, with clinical cases displaying

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significantly worse profiles. The data supported a nuanced, individualized clinical approach that integrated cardiovascular evaluation into thyroid management—highlighting opportunities for earlier detection and intervention to reduce morbidity.

CONCLUSION

This study demonstrated that cardiovascular risk is more pronounced in clinical hypothyroidism than in subclinical disease, with significant differences in cardiac function, lipid profiles, and inflammatory markers. The findings highlight the importance of integrating cardiovascular assessment into the routine management of hypothyroid patients, particularly those with overt disease. Early identification and targeted intervention may help mitigate long-term cardiovascular complications, supporting a proactive approach in clinical practice.

AUTHOR CONTRIBUTION

Author	Contribution
Shabahat Arain*	Substantial Contribution to study design, analysis, acquisition of Data Manuscript Writing Has given Final Approval of the version to be published
Saad Umer Thanvi	Substantial Contribution to study design, acquisition and interpretation of Data Critical Review and Manuscript Writing Has given Final Approval of the version to be published
Naheed Shah	Substantial Contribution to acquisition and interpretation of Data Has given Final Approval of the version to be published
Muhammad Damil Farid	Contributed to Data Collection and Analysis Has given Final Approval of the version to be published
Ali Raza	Contributed to Data Collection and Analysis Has given Final Approval of the version to be published
Irfan Ishaque	Substantial Contribution to study design and Data Analysis Has given Final Approval of the version to be published
Salih Noor	Contributed to study concept and Data collection Has given Final Approval of the version to be published
Syed Muhammad Afraz Haider	Writing - Review & Editing, Assistance with Data Curation

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