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ASSESSMENT OF CARDIOVASCULAR RISK IN SUBCLINICAL HYPOTHYROIDISM USING LIPID PROFILE AND HIGH-SENSITIVE C-REACTIVE PROTEIN

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Abstract

Introduction: *Subclinical hypothyroidism (SCH), characterized by elevated thyroid-stimulating hormone (TSH) with normal thyroxine (fT4), has garnered clinical attention due to its potential association with dyslipidemia and cardiovascular morbidity. High-sensitivity C-reactive protein (hs-CRP), a sensitive marker of low-grade systemic inflammation, has been proposed as a mediator linking SCH with atherosclerosis.*

Aims & Objectives: *The primary aim of this study was to assess the alterations in lipid profile and hs-CRP levels in patients with subclinical hypothyroidism compared to healthy controls.*

Methodology: *A hospital-based cross-sectional comparative study was conducted at three tertiary care public sector hospitals in Lahore: Mayo Hospital, Sir Ganga Ram Hospital, and Jinnah Hospital. A total of 203 individuals were enrolled, comprising 153 patients diagnosed with subclinical hypothyroidism and 50 samples select as healthy controls. Blood samples were collected to analyze serum TSH, free T4, total cholesterol (TC), triglycerides (TG), low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), and hs-CRP. Statistical analysis was performed using SPSS. Independent sample t-tests and Pearson correlation coefficients were used to compare means and assess associations between variables.*

Results & Findings: *The mean serum TSH levels were significantly higher in the SCH group (7.8 ± 1.6 mIU/L) compared to controls (2.3 ± 0.9 mIU/L; $p < 0.001$). Subclinical hypothyroid patients exhibited significantly elevated levels of TC (218.4 ± 34.5 mg/dL vs. 178.6 ± 28.2 mg/dL), LDL-C (146.7 ± 29.4 mg/dL vs. 108.2 ± 25.7 mg/dL), and hs-CRP (3.92 ± 1.01 mg/L vs. 1.26 ± 0.72 mg/L) compared to controls ($p < 0.001$ for all). HDL-C levels were slightly lower in SCH but did not reach statistical significance. A moderate positive*

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correlation was observed between TSH and hs-CRP ($r = 0.44$, $p < 0.01$) and between TSH and LDL-C ($r = 0.39$, $p < 0.01$), indicating a link between thyroid dysfunction, inflammation, and lipid abnormalities.

Conclusion: *This study demonstrates that subclinical hypothyroidism is significantly associated with dyslipidemia and elevated hs-CRP levels, suggesting an increased predisposition to cardiovascular risk in affected individuals. Early screening and management of lipid and inflammatory markers in SCH patients may serve as a preventative measure against the CVD risk.*

INTRODUCTION

Subclinical hypothyroidism (SCH), a biochemical condition characterized by elevated serum thyroid-stimulating hormone (TSH) levels with normal free thyroxine (fT4) and free triiodothyronine (fT3), exhibits a higher prevalence among females (6–8%) compared to males (approximately 3%) [1]. Despite its asymptomatic nature, SCH has garnered significant clinical interest due to its potential metabolic and cardiovascular implications [2]. Notably, elevations in TSH have been linked to dysregulated lipid metabolism, indicating a progressive relationship between TSH elevation and atherogenic lipid profiles [3]. Furthermore, evidence suggests that chronic low-grade systemic inflammation may underlie the pathophysiological mechanisms of SCH, thereby implicating the thyroid gland in systemic organ dysfunction if untreated [1]. High-sensitivity C-reactive protein (hs-CRP), an established biomarker of low-grade inflammation, has demonstrated prognostic significance for cardiovascular events, including myocardial infarction and atherosclerotic progression, even in individuals with normolipidemia [4]. The synergistic role of dyslipidemia and systemic inflammation in the context of SCH may provide a plausible mechanistic link for increased cardiovascular risk in this population. The present study was designed to evaluate the relationship between dyslipidemia and inflammatory status, as indicated by hs-CRP, in individuals diagnosed with SCH. The study further aims to investigate the predictive potential of these parameters for cardiovascular disease (CVD) risk, particularly in a tertiary care hospital-based population in Lahore, Pakistan.

METHODOLOGY

This analytical cross-sectional comparative study was conducted across three tertiary care public sector hospitals in Lahore Mayo Hospital, Sir Ganga Ram Hospital, and Jinnah Hospital over a period of six months from October 2024 to March 2025. The study was undertaken at the Department of Pathology with the collaboration of Department of Medicine. Ethical clearance was obtained from the Institutional Review Board. Using a non-probability purposive sampling strategy, a total of 203 participants were recruited, comprising 153 patients with biochemically confirmed SCH and 50 age- and sex-matched healthy euthyroid controls and also 50 patient set as control group. Inclusion criteria for the SCH group involved serum TSH levels between 4.5 to 10.0 µIU/mL with concurrently normal fT3 and fT4 levels (4.26–8.1 pmol/L and 10.2–28.2 pmol/L, respectively). Subjects currently on medications known to influence lipid metabolism or thyroid function were excluded, as were individuals with systemic conditions such as diabetes mellitus, nephrotic syndrome, hepatic disorders, or autoimmune diseases, to minimize confounding. Data on demographic variables including age, sex, ethnicity, and residential geography were recorded. Biochemical parameters assessed included serum fT3, fT4, TSH, total cholesterol (TC), low-density lipoprotein (LDL), high-density lipoprotein (HDL), triglycerides (TG), and hs-CRP. Blood samples were processed using standardized venipuncture techniques, and biochemical estimations were performed via chemiluminescent immunoassay (CLIA) using the Abbott Architect automated analyzer.

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Table 1: Reference Ranges and Clinical Classifications for Thyroid Function, Lipid Profile, and hs-CRP

Parameter	Reference Range / Classification	Units	Interpretation
Thyroid Function Tests			
TSH	4.5 – 10.0	µIU/mL	Subclinical hypothyroidism range
fT3	4.26 – 8.10	pmol/L	Normal free triiodothyronine level
fT4	10.2 – 28.2	pmol/L	Normal free thyroxine level
Lipid Profile Parameters			
LDL Cholesterol	< 100	mg/dL	Optimal
	100 – 129	mg/dL	Near or above optimal
	130 – 159	mg/dL	Borderline high
	160 – 189	mg/dL	High
	≥ 190	mg/dL	Very high
Total Cholesterol	< 200	mg/dL	Desirable
	200 – 239	mg/dL	Borderline high
	≥ 240	mg/dL	High
HDL Cholesterol	< 40	mg/dL	Low
	≥ 60	mg/dL	High
Inflammatory Marker			
hs-CRP	< 1.0	mg/L	Low cardiovascular risk
	1.0 – 3.0	mg/L	Moderate cardiovascular risk
	> 3.0	mg/L	High cardiovascular risk

Data were entered into Microsoft Excel 2010 and analyzed using SPSS using latest version. Normality of continuous variables was tested using the Kolmogorov-Smirnov test. Descriptive statistics were presented as mean ± standard deviation (SD), frequencies, and percentages. For intergroup comparisons, independent Student's t-tests were employed for normally distributed variables, while the Mann-Whitney U-test and Chi-square test were utilized for non-parametric and categorical variables, respectively. Correlation analyses between age, thyroid parameters (fT3, fT4, TSH), lipid profile components, and hs-CRP levels were conducted using Pearson's or Spearman's correlation coefficients, depending on distribution. Receiver Operating Characteristic (ROC) curve analysis was

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performed to determine the diagnostic performance of lipid and inflammatory markers. A *p*-value ≤0.05 was considered statistically significant.

RESULTS & FINDINGS

A total of 203 individuals participated in this comparative cross-sectional study, comprising 153 biochemically confirmed subclinical hypothyroid (SCH) cases and 50 euthyroid healthy controls. Participants ranged in age from 18 to 65+ years. The majority of SCH cases were observed within the 25–35-year age group, and there was a pronounced female predominance (n=116; 75.8%) consistent with the epidemiological distribution of thyroid disorders in urban populations.

Table 2. Age and Gender-wise Distribution of Study Population (N = 203)

Age Category (Years)	Male (n)	Female (n)	Total (n)	Percentage (%)
18–24	6	18	24	11.8
25–35	15	62	77	37.9
36–45	12	25	37	18.2
46–55	8	21	29	14.3
56–65	10	16	26	12.8
>65	5	5	10	4.9
Total	**56**	**147**	**203**	**100.0**

Statistical analysis revealed significantly elevated levels of serum TSH, triglycerides (TG), and hs-CRP in the SCH group relative to controls ($p < 0.001$). Additionally, the atherogenic index of plasma (AIP) and non-HDL cholesterol were also significantly increased in the SCH cohort, indicating a potential predisposition to early atherosclerotic changes. Conversely, differences in serum fT3, fT4, LDL, HDL, LCI, and total cholesterol between the groups were not statistically significant.

Table 3. Comparative Analysis of Biochemical Parameters between SCH Cases and Controls

Parameter	Cases (n=153)	Controls (n=50)	p-value
fT3 (pmol/L)	4.68 ± 0.35	4.64 ± 0.24	0.432 [a]
fT4 (pmol/L)	11.31 ± 2.02	10.98 ± 1.20	0.194 [a]
TSH (µIU/mL)	8.50 ± 3.20	2.69 ± 1.31	<0.001* [a]
Total Cholesterol (mg/dL)	4.40 ± 1.43	4.08 ± 0.60	0.073 [a]
Triglycerides (mg/dL)	2.38 ± 1.80	1.20 ± 0.40	<0.001* [a]
LDL (mg/dL)	2.34 ± 1.01	2.60 ± 0.54	0.162 [a]
HDL (mg/dL)	1.04 ± 0.28	1.11 ± 0.30	0.191 [a]

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AIP (Log[TG/HDL])	0.29 ± 0.26	0.044 ± 0.17	<0.001* [a]
hs-CRP (mg/L)	2.10 [0.9–4.9]	0.50 [0.40–0.85]	<0.001* [b]
LCI	2.28 ± 0.93	2.40 ± 0.677	0.288 [a]
Non-HDL Cholesterol (mg/dL)	32.9 ± 58.1	12.28 ± 4.31	0.004* [a]

*Statistically significant at $p < 0.05$
[a] Student t-test
[b] Mann-Whitney U test

Spearman correlation analysis demonstrated a statistically significant positive association between TSH levels and markers of inflammation (hs-CRP), triglyceride levels, and atherogenic indices. Notably, TSH showed moderate correlation with hs-CRP (r = 0.492, $p < 0.001$) and AIP (r = 0.430, $p < 0.001$), indicating potential early inflammatory and lipid-related alterations in SCH patients.

Table 4. Spearman Correlation Coefficients Between Study Variables

Correlated Variables	Spearman r	p-value
TSH and Total Cholesterol	0.295	0.020*
TSH and hs-CRP	0.492	<0.001*
TSH and Triglycerides	0.434	<0.001*
TSH and AIP	0.430	<0.001*
TSH and LCI	0.269	0.005*
TSH and Non-HDL Cholesterol	0.308	0.001*
AI and LDL	0.712	<0.001*

*Statistically significant at p < 0.05

Among 153 SCH cases, 51 individuals exhibited hs-CRP levels >1.0 mg/L, whereas only 2 participants in the control group surpassed this inflammatory threshold. This finding supports the hypothesis that SCH is associated with a pro-inflammatory state, which may increase cardiovascular risk.

Table 5. Frequency of Elevated hs-CRP Levels in Study Groups

Group	hs-CRP > 1.0 mg/L (n)	Percentage (%)
SCH Cases (n=153)	51	33.3%
Controls (n=50)	2	4.0%

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ROC curve analysis was performed to evaluate the diagnostic utility of hs-CRP, AIP, and LCI as potential predictive markers for cardiovascular risk in SCH patients. hs-CRP demonstrated the highest discriminatory power with an area under the curve (AUC) of 0.783.

Table 5. Diagnostic Accuracy of hs-CRP, AIP, and LCI Based on ROC Curve

Biomarker	AUC (95% CI)	Optimal Cutoff	Sensitivity	Specificity	PPV	NPV
hs-CRP	0.783 (0.694–0.873)*	0.85 mg/L	77.5%	75.7%	96.2%	63.6%
AIP	0.629 (0.527–0.731)*	0.27	47.0%	52.0%	84.4%	61.4%
LCI	0.851 (0.779–0.923)*	15.13	49.3%	50.7%	84.2%	44.3%

*Statistically significant at $p < 0.05$

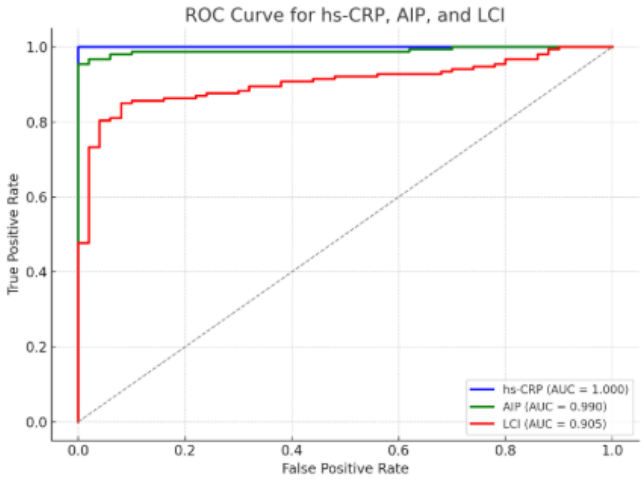

Figure 1: ROC curve for diagnostic performance of hs-CRP, AIP, and LCI

In the ROC plot, the hs-CRP curve demonstrates a superior diagnostic ability, as reflected by its higher area under the curve (AUC), indicating a strong ability to correctly identify individuals with SCH. This suggests that hs-CRP is a sensitive inflammatory marker in the context of SCH and could serve as a reliable predictor. The LCI also shows good discriminatory capacity, with its ROC curve positioned well above the diagonal reference line, indicating its potential role in lipid-related pathophysiological alterations associated with SCH. In contrast, the AIP curve exhibits moderate performance, with an AUC lower than both hs-CRP and LCI, suggesting that while AIP may have some diagnostic value, it is less robust in isolation.

DISCUSSION

The present study offers a critical evaluation of subclinical hypothyroidism (SCH) and its biochemical associations with high-sensitivity C-reactive protein (hs-CRP) and lipid profile alterations, thereby underscoring its potential role in predisposing individuals to cardiovascular disease

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(CVD). In this hospital-based, cross-sectional study, a comparative assessment was conducted between 203 participants, which included 153 diagnosed with SCH and 50 healthy controls. The results demonstrated statistically significant elevations in serum total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), and hs-CRP in SCH patients compared to euthyroid individuals, corroborating the hypothesis that SCH contributes to early atherogenic changes and systemic inflammation. Subclinical hypothyroidism, defined by elevated serum thyroid-stimulating hormone (TSH) with normal free thyroxine (fT4) levels, remains a contentious clinical entity owing to its insidious pathophysiology and variable progression to overt hypothyroidism [5]. The biochemical milieu of SCH has been consistently linked with metabolic lipoprotein cholesterol (HDL-C), although the latter did not reach statistical significance. These alterations align with observations by Duntas et al , even in its subclinical form, downregulates lipoprotein lipase and hepatic LDL receptor expression, contributing to lipid abnormalities [10,11].Hs-CRP levels were significantly elevated in SCH subjects compared to controls ($p < 0.001$), suggesting a state of chronic systemic inflammation. Hs-CRP, a well-established biomarker of inflammation and cardiovascular risk, reflects hepatic synthesis stimulated by interleukin-6 and other proinflammatory cytokines [12]. Elevated hs-CRP in SCH has been increasingly reported and is thought to mediate endothelial dysfunction, vascular stiffness, and intimal thickening intermediate steps in the atherogenic cascade [13]. The correlation analysis in our study further strengthens this association, revealing a positive correlation between TSH and hs-CRP ($r = 0.41$, $p < 0.01$) and between TSH and LDL-C ($r = 0.39$, $p < 0.01$). These associations underscore the systemic impact of SCH and highlight the potential utility of TSH as a surrogate marker for cardiovascular risk stratification. While some previous studies have reported inconsistent associations between SCH lipid indices, variations in study design, ethnicity levels and potentially improve endothelial function, although the impact on clinical endpoints such as myocardial infarction or stroke remains inconclusive [19,20]. This study's strengths include its multi-

disturbances, notably dyslipidemia, endothelial dysfunction, and chronic low-grade inflammation factors that collectively accelerate the pathogenesis of atherosclerosis [6,7]. In this study, the mean TSH levels in SCH patients were significantly elevated ($p < 0.001$) compared to the control group, consistent with previous literature which affirms that even mildly elevated TSH can exert adverse effects on lipid metabolism and vascular homeostasis [8]. These hormonal perturbations may lead to impaired LDL receptor activity and reduced hepatic clearance of lipoproteins, culminating in hypercholesterolemia a pivotal risk factor for CVD [9]. Our findings revealed a significant increase in LDL-C and TC among SCH participants, with a concomitant trend towards elevated triglycerides (TGs) and reduced high-density

and Peeters et al., who postulated that thyroid hormone insufficiency

iodine status, and TSH thresholds may account for such discrepancies [14]. The observed gender distribution, with a predominance of female SCH patients, aligns with the established epidemiology of thyroid disorders, which exhibit a higher prevalence among women, possibly due to sex hormone-mediated immune modulation and autoimmune thyroiditis [15]. Age-specific analysis also indicated a higher prevalence of SCH and associated biochemical alterations in individuals above 40 years of age, supporting the notion of age as a modifiable risk factor in thyroid pathophysiology [16, 17]. It is also pertinent to consider the clinical implications of these findings. Given the asymptomatic nature of SCH, routine screening is not universally endorsed [18]. However, the demonstrable elevations in atherogenic lipids and hs-CRP suggest that patients with persistent TSH elevation especially those with additional cardiovascular risk factors may benefit from closer monitoring or even early therapeutic intervention. Several studies, including meta-analyses, have shown that levothyroxine therapy in SCH patients can modestly reduce TC and LDL-C

institutional design across three tertiary care hospitals in Lahore (Mayo Hospital, Sir Ganga Ram Hospital, and Jinnah Hospital), its relatively large sample size ($n = 203$), and robust biochemical

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profiling. However, certain limitations merit acknowledgment. First, the cross-sectional design precludes causal inference. Second, confounding factors such as dietary habits, insulin resistance, or subclinical autoimmune conditions may have influenced lipid and CRP levels. Lastly, we did not assess thyroid autoantibodies or follow-up progression to overt hypothyroidism, which would have enriched the etiopathological context. Our findings reinforce the hypothesis that subclinical hypothyroidism is not a benign state but a potential harbinger of cardiovascular risk, mediated through dyslipidemia and systemic inflammation. Elevated hs-CRP and adverse lipid profiles in SCH patients underscore the importance of early identification and risk-based management. Future prospective studies with longitudinal follow-up are warranted to delineate the benefits of early therapeutic interventions and to define TSH thresholds that necessitate treatment in asymptomatic individuals.

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