International Journal of Research in Medical Sciences
Jamatia M et al. Int J Res Med Sci. 2026 Aug;14(8):3379-3386
www.msjonline.org
pISSN 2320-6071 | eISSN 2320-6012
DOI: https://dx.doi.org/10.18203/2320-6012.ijrms20262625

Original Research Article

Biochemical correlation of serum insulin, high-sensitivity C reactive protein and lipid tetrad index with thyroid-stimulating hormone levels in patients with hypothyroidism

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Received: 20 May 2026
Revised: 15 June 2026
Accepted: 04 July 2026

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ABSTRACT

Background: Hypothyroidism is a prevalent endocrine disorder associated with insulin resistance, dyslipidemia, chronic low-grade inflammation, and an increased risk of cardiovascular disease. Thyroid hormone deficiency adversely affects glucose and lipid metabolism, contributing to metabolic dysfunction. This study investigated the relationship between thyroid-stimulating hormone (TSH) levels and markers of insulin resistance, systemic inflammation, and atherogenic risk in patients with hypothyroidism.
Methods: A hospital-based cross-sectional analytical study was conducted among 70 patients with hypothyroidism attending a tertiary care teaching hospital in Indore, Madhya Pradesh, India. Based on thyroid function tests, participants were categorized as having overt or subclinical hypothyroidism. Serum triiodothyronine (T3), thyroxine (T4), TSH, fasting insulin, high-sensitivity C-reactive protein (hs-CRP), and lipid profile were measured using standard laboratory methods. Insulin resistance was assessed using the homeostatic model assessment for insulin resistance (HOMA-IR), while atherogenic risk was evaluated using the lipid tetrad index (LTI). Correlation analysis was performed to examine associations between TSH and study variables.
Results: Serum TSH demonstrated significant positive correlations with fasting insulin ($r=0.42$, $p<0.01$), hs-CRP ($r=0.38$, $p<0.01$), and LTI ($r=0.44$, $p<0.001$). Compared with patients with subclinical hypothyroidism, those with overt hypothyroidism had significantly higher fasting insulin, HOMA-IR, hs-CRP, and LTI values. Insulin resistance was identified in 58.6% of participants.
Conclusions: Higher TSH levels are associated with insulin resistance, systemic inflammation, and increased atherogenic risk in hypothyroidism. These findings highlight the importance of comprehensive metabolic assessment for improved cardiovascular risk stratification and timely clinical intervention.

Keywords: Hypothyroidism, Thyroid-stimulating hormone, Insulin resistance, HOMA-IR, High-sensitivity C-reactive protein, Lipid tetrad index, Cardiovascular risk

INTRODUCTION

Hypothyroidism is one of the most prevalent endocrine disorders globally and poses a significant public health challenge because of its multisystemic and metabolic effects. Thyroid hormones are integral to the regulation of carbohydrate metabolism, lipid homeostasis, thermogenesis, cardiovascular function, and protein synthesis. Deficiency of these hormones leads to extensive metabolic disturbances, including insulin resistance,

dyslipidemia, endothelial dysfunction, oxidative stress, and chronic low-grade inflammation.[1-10]

India bears a substantial burden of thyroid disorders, particularly among women, with hypothyroidism increasingly recognized as a key contributor to cardiometabolic morbidity.[11] Both overt and subclinical hypothyroidism are linked to abnormalities in glucose metabolism and lipid profiles, predisposing individuals to cardiovascular diseases.[3,8,9,24]

There is a growing association between insulin resistance and thyroid dysfunction. Reduced thyroid hormone activity impairs peripheral glucose utilization and alters adipokine secretion, thereby contributing to impaired insulin sensitivity.[2,5,24,39,40] Similarly, elevated hs-CRP levels indicate systemic inflammatory activation in hypothyroid patients and may serve as markers of cardiovascular risk.[4,14,15]

Hypothyroidism is also associated with significant alterations in lipid metabolism, including elevated levels of total cholesterol, triglycerides, and low-density lipoprotein cholesterol.[3,15,16] The LTI, which incorporates total cholesterol, triglycerides, lipoprotein(a), and HDL cholesterol, has emerged as a valuable composite marker for assessing cardiovascular risk.[25,26]

Recent evidence suggests that thyroid dysfunction contributes significantly to the development of metabolic syndrome and accelerated atherosclerosis through several interconnected mechanisms. Thyroid hormones regulate hepatic lipid metabolism, low-density lipoprotein receptor expression, and lipoprotein lipase activity. Deficiency of these hormones results in impaired lipid clearance and increased circulating atherogenic lipoproteins, thereby enhancing cardiovascular risk.[6,11,12] In addition, hypothyroidism is associated with endothelial dysfunction, increased arterial stiffness, and altered vascular reactivity, which further contribute to cardiovascular complications.[13,20]

Subclinical hypothyroidism, despite minimal clinical manifestations, may result in significant biochemical abnormalities and adverse outcomes. Several studies have demonstrated that elevated TSH levels, even within mildly increased ranges, are associated with insulin resistance, inflammatory activation, and dyslipidemia.[7,18,19,39,40] Therefore, early identification of metabolic and inflammatory derangements in patients with hypothyroidism is essential for timely therapeutic intervention and prevention of long-term complications.

The assessment of combined biochemical markers, such as fasting insulin, hs-CRP, and the LTI, may provide a more comprehensive understanding of cardiometabolic risk in hypothyroidism. Evaluating their relationship with TSH could help identify patients at a greater risk of cardiovascular morbidity and improve strategies for monitoring and clinical management.

There are limited data regarding the integrated relationship between TSH, insulin resistance, inflammatory biomarkers, and the LTI in hypothyroidism. Therefore, the present study aimed to evaluate the correlation between serum insulin, hs-CRP, and the LTI with TSH levels in patients with hypothyroidism.

METHODS

Study design and setting

This hospital-based cross-sectional analytical study was conducted in the Department of Medical Biochemistry at Index Medical College Hospital and Research Centre, Indore, Madhya Pradesh, India, a tertiary care teaching hospital. The study was carried out over a period of one year, from 22 February 2025 to 23 February 2026. A total of 70 adult patients diagnosed with hypothyroidism and attending the outpatient and inpatient departments during the study period were enrolled after obtaining written informed consent.

The study aimed to evaluate the association of serum insulin, hs-CRP, and LTI with TSH levels in patients with hypothyroidism. Participants were categorized into overt and subclinical hypothyroidism based on thyroid function test results. All biochemical investigations were performed in the Central Clinical Biochemistry Laboratory using standardized analytical methods with appropriate internal quality control procedures to ensure accuracy and reliability of the results.

Study population

The study population comprised 70 adult patients (≥18 years) diagnosed with hypothyroidism who attended the outpatient and inpatient departments of Index Medical College Hospital and Research Centre, Indore, Madhya Pradesh, India, during the study period. Based on serum TSH levels, participants were categorized into subclinical hypothyroidism (TSH >4.5-10 µIU/mL; n=20, 28.6%) and overt hypothyroidism (TSH>10 µIU/mL; n=30, 42.9%). Additionally, 20 individuals (28.6%) with normal TSH levels were included for comparative analysis. All participants fulfilled the predefined eligibility criteria and provided written informed consent prior to enrollment.

Inclusion criteria

The following participants were included in the study: Adults aged 18 years and above diagnosed with hypothyroidism based on clinical and biochemical findings, patients willing to participate and provide written informed consent and patients attending the outpatient or inpatient departments during the study period.

Exclusion criteria

Participants meeting any of the following criteria were excluded from the study: Known diabetes mellitus, chronic

inflammatory or autoimmune disorders, pre-existing hepatic or renal disease, pregnancy or lactation, established cardiovascular disease and malignancy or severe systemic illness.

Current use of lipid-lowering, anti-inflammatory, corticosteroid, or immunomodulatory medications that could influence metabolic or inflammatory parameters.

Sample collection

Approximately 5 ml of fasting venous blood was collected under aseptic conditions after an overnight fast of 8-12 h. The samples were centrifuged at 3000 rpm for 10 min for serum separation, and the serum was used for biochemical analyses.

Biochemical analysis

Serum levels of T3, T4, TSH, fasting insulin, hs-CRP, and lipid profile parameters were quantified using standard biochemical and immunoassay techniques on an automated analyzer. The estimation and interpretation of hs-CRP were conducted in accordance with the established guidelines for cardiovascular inflammatory markers.[4,14,15]

Assessment of insulin resistance

Insulin resistance was evaluated using the HOMA-IR, as described by Matthews et al.[22]

HOMA-IR=Fasting insulin (μU/ml)×Fasting glucose (mg/dl)/405

LTI

The LTI was calculated using the formula described by Enas et al and Das et al.[25,26]

LTI=Total cholesterol × triglycerides × lipoprotein(a)/ HDL-C

Statistical analysis

Data were entered and analyzed using IBM SPSS statistics version 26 and Microsoft excel. Continuous variables are expressed as mean±standard deviation (SD), and categorical variables are presented as frequencies and percentages. Differences between groups were evaluated using the student's t test for continuous variables and the chi-square test for categorical data. Correlation analysis between serum TSH levels and biochemical parameters, including serum insulin, HOMA-IR, hs-CRP, and LTI, was performed using Pearson's correlation coefficient for normally distributed variables and Spearman's rank correlation coefficient for non-normally distributed variables. Comparative analysis between the groups was further assessed using multiple linear regression models to adjust for potential confounding factors such as age. A p value of less than 0.05 was considered statistically significant and $p<0.01$ was considered highly significant. The statistical analysis aimed to evaluate the relationship between thyroid dysfunction, insulin resistance, systemic inflammation, and cardiovascular risk markers in patients with hypothyroidism.

RESULTS

A total of 70 participants were enrolled in the present study. Their baseline demographic and anthropometric characteristics, including age, sex, height, weight, body mass index (BMI), and TSH status, are summarized in Table 1. The study population comprised a balanced distribution of males and females, with the largest proportion of participants belonging to the 26-35-year age group. Based on serum TSH concentrations, 20 (28.6%) participants were euthyroid, 20 (28.6%) had subclinical hypothyroidism, and 30 (42.9%) had overt hypothyroidism.

The distribution of participants according to serum TSH categories is presented in Table 2. Overt hypothyroidism constituted the largest subgroup, accounting for 42.9% of the study population, whereas normal thyroid function and subclinical hypothyroidism each represented 28.6% of participants.

Serum TSH levels demonstrated a significant positive correlation with fasting serum insulin concentrations ($r=0.42$, $p<0.01$), indicating that increasing severity of thyroid dysfunction was associated with progressive impairment in insulin sensitivity. This relationship is illustrated in Figure 1.

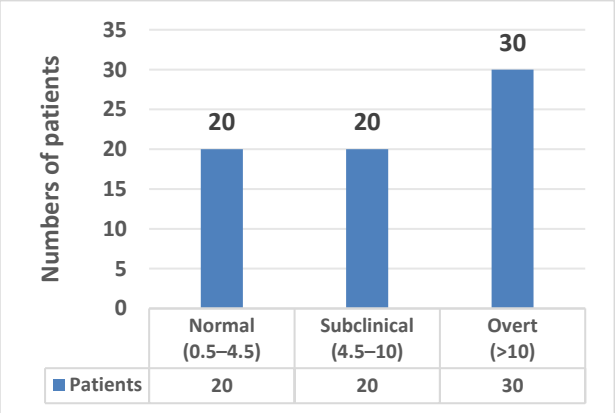

Figure 1: Distribution of patients according to TSH levels.

*Distribution of study participants according to serum TSH levels, showing normal, subclinical hypothyroid, and overt hypothyroid categories within the study population.

Assessment of insulin resistance revealed a mean HOMA-IR value of 3.62±1.37, which exceeded the accepted reference range. More than half of the participants (58.6%) were classified as insulin resistant, while 25.7% exhibited borderline insulin resistance and 15.7% remained insulin

sensitive. The distribution of HOMA-IR values is presented in Table 3.

A statistically significant positive correlation was also observed between serum TSH and hs-CRP levels (r=0.38, p<0.01), suggesting an increase in systemic inflammatory activity with worsening thyroid dysfunction. The correlation between TSH and hs-CRP is shown in Figure 2, while distribution of participants according to hs-CRP cardiovascular risk categories is summarized in Table 4.

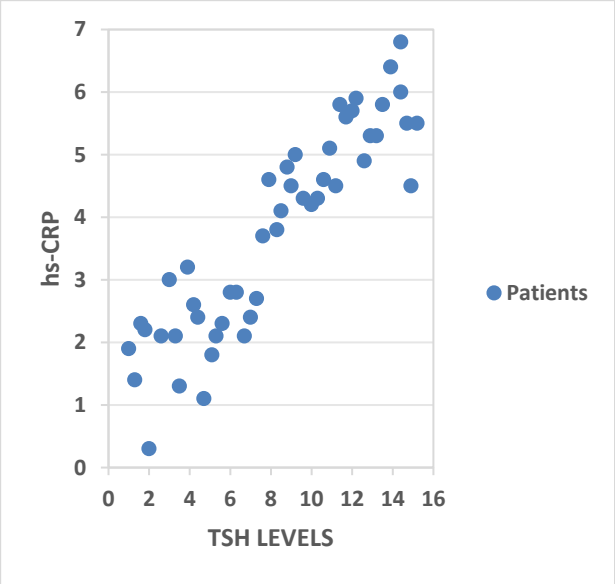

Figure 2: Correlation between hs-CRP and TSH.

*Correlation between hs-CRP and serum TSH levels, indicating the relationship between thyroid dysfunction and systemic inflammation.

Similarly, serum TSH demonstrated a significant positive correlation with the LTI (r=0.44, p<0.001), indicating a progressive increase in atherogenic cardiovascular risk as thyroid dysfunction became more severe.

This correlation is depicted in Figure 3, whereas the distribution of LTI values and corresponding cardiovascular risk categories is presented in the Table 5.

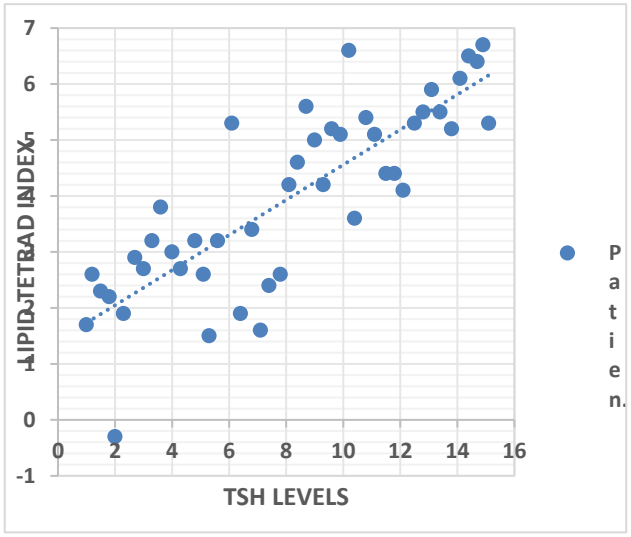

Figure 3: Correlation between LTI and TSH.

*Correlation between LTI and serum TSH levels, demonstrating the association between the severity of hypothyroidism and cardiovascular risk.

Comparative analysis between overt and subclinical hypothyroidism demonstrated that patients with overt disease exhibited higher fasting insulin levels, HOMA-IR, hs-CRP concentrations, triglyceride levels, and LTI alues than those with subclinical hypothyroidism (Table 6). These findings indicate that metabolic dysregulation, systemic inflammation, and atherogenic abnormalities become progressively more pronounced with increasing severity of thyroid dysfunction. Importantly, patients with subclinical hypothyroidism also demonstrated measurable biochemical alterations, suggesting that clinically mild thyroid dysfunction may be associated with early metabolic and inflammatory disturbances.

Table 1: Demographic and anthropometric characteristics of the study participants, (n=70).

Variables	Category	N	Percentage (%)
Age (in years)	18-25	17	22.7
	26-35	18	24.0
	36-45	13	17.3
	46-55	16	21.3
	56-60	6	8.0
Gender	Male	36	51.4
	Female	34	48.6
Height (cm)	150-159.9	12	17.1
	160-169.9	18	25.7
	170-179.9	17	24.3
	180-189.9	23	32.9
Weight (kg)	50-59.9	9	12.9
	60-69.9	17	24.3
	70-79.9	19	27.1
	80-89.9	25	35.7

Continued.

Variables	Category	N	Percentage (%)
BMI (kg/m²)	Underweight (<18.5)	5	7.1
	Normal weight (18.5-24.9)	26	37.1
	Overweight (25.0-29.9)	24	34.3
	Obese class I (30.0-34.9)	9	12.9
	Obese class II (35.0-39.9)	2	2.9
	Obese class III (≥40.0)	0	0.0
TSH category	Normal (0.5-4.5 µIU/mL)	20	28.6
	Subclinical hypothyroidism (>4.5-10 µIU/mL)	20	28.6
	Overt hypothyroidism (>10 µIU/mL)	30	42.9

*Demographic and anthropometric characteristics of the study participants categorized according to age, gender, height, weight, BMI, and TSH status.

Table 2: Distribution of patients according to TSH levels.

TSH range (µIU/mL)	Interpretation	Count (%)
0.5-4.5	Normal	20 (28.6)
>4.5-10	Subclinical hypothyroidism	20 (28.6)
>10	Overt hypothyroidism	30 (42.9)

*Distribution of study participants according to serum TSH levels, categorized as normal thyroid function, subclinical hypothyroidism, and overt hypothyroidism.

Table 3: Distribution of HOMA-IR values among hypothyroid patients.

HOMA-IR value	Interpretation	Percentage (%)
<2.0	Insulin sensitive	15.7
2.0-2.9	Borderline/moderate insulin resistance	25.7
≥3.0	Insulin resistant	58.6
Parameter	Mean value	
HOMA-IR	3.62	

*Distribution of insulin resistance among hypothyroid patients based on HOMA-IR values. Most participants (58.6%) were insulin resistant, while 25.7% had borderline/moderate insulin resistance and 15.7% were insulin sensitive. The mean HOMA-IR was 3.62.

Table 4: Distribution and correlation of hs-CRP levels with TSH.

hs-CRP category	Range (mg/l)	Count (%)
Low risk	≤1.0	14 (20.0)
Moderate risk	1.01-2.0	31 (44.3)
High risk	>2.0	25 (35.7)
Correlation analysis		
Parameter	Correlation coefficient (r)	P value
TSH vs hs-CRP	+0.38	<0.01

*Distribution of hs-CRP levels according to inflammatory risk categories and their correlation with TSH. This table illustrates the relationship between systemic inflammation and thyroid dysfunction.

Table 5: Distribution and correlation of LTI with TSH.

LTI category	Cardiovascular risk	Percentage (%)
<10,000	Low risk	15.7
10,000-20,000	Moderate risk	45.7
>20,000	High risk	38.6
Correlation analysis		
Parameter	Correlation coefficient (r)	P value
TSH vs LTI	+0.44	<0.001
Descriptive statistic	**Value**	
Mean LTI	19,506.70	
Range	5,299.99-47,827.17	

*Distribution of LTI values indicating cardiovascular risk and their correlation with serum TSH levels.

Table 6: Comparative analysis between overt and subclinical hypothyroidism.

Parameters	Overt hypothyroidism (TSH >10)	Subclinical hypothyroidism (TSH 4.5-10)
Serum insulin	Higher	Moderately elevated
HOMA-IR	Higher	Moderate
hs-CRP	Higher	Mild-moderate elevation
Triglycerides	Higher	Moderate elevation
LTI	Higher	Elevated
Metabolic abnormalities	More pronounced	Present but milder

*Comparative evaluation of biochemical and metabolic parameters between overt and subclinical hypothyroidism groups.

DISCUSSION

The present study demonstrated a significant association between hypothyroidism and metabolic, inflammatory, and atherogenic abnormalities. Positive correlations between serum TSH concentrations and fasting insulin, hs-CRP, and the LTI indicate that progressive thyroid dysfunction is accompanied by worsening insulin resistance, enhanced systemic inflammation, and increased cardiovascular risk. These observations support the concept that hypothyroidism is a multisystem disorder extending beyond impaired thyroid hormone production.

The significant elevation of fasting insulin levels and HOMA-IR values observed in the present study suggests impaired glucose homeostasis and reduced peripheral insulin sensitivity in patients with hypothyroidism. Thyroid hormones play an essential role in regulating glucose metabolism by influencing hepatic glucose production, intestinal glucose absorption, and insulin-mediated glucose utilization. Consequently, thyroid hormone deficiency may promote insulin resistance through reduced glucose disposal and altered insulin signaling pathways. These findings are consistent with previous studies reporting increased insulin resistance in both overt and subclinical hypothyroidism.[2,5,24,39,40]

The observed positive correlation between serum TSH and hs-CRP further highlights the contribution of chronic low-grade systemic inflammation to the pathophysiology of hypothyroidism. Elevated hs-CRP concentrations may reflect persistent inflammatory activation, endothelial dysfunction, oxidative stress, and vascular injury, all of which contribute to accelerated atherosclerosis and increased cardiovascular morbidity. Similar associations between thyroid dysfunction and inflammatory biomarkers have been documented in earlier investigations.[4,9,14,39,40]

An important finding of the present study was the significant positive relationship between serum TSH and the LTI, indicating progressively increasing atherogenic risk with worsening thyroid dysfunction. Thyroid hormone deficiency is known to impair lipid metabolism by reducing hepatic LDL receptor activity, decreasing lipoprotein clearance, and promoting hypertriglyceridemia. When combined with insulin resistance and chronic inflammation, these alterations may substantially increase cardiovascular risk in hypothyroid patients. The present findings are in agreement with previous reports demonstrating an increased prevalence of dyslipidemia and cardiovascular disease among individuals with hypothyroidism.[3,9,25,26]

Notably, although patients with overt hypothyroidism exhibited more severe biochemical abnormalities than those with subclinical hypothyroidism, measurable metabolic and inflammatory disturbances were also evident in the subclinical group. This observation suggests that subclinical hypothyroidism should not be regarded as an entirely benign condition, as early metabolic derangements may already be present before the development of overt disease. Early identification, comprehensive metabolic assessment, and timely therapeutic intervention may therefore help reduce the long-term burden of cardiovascular complications in these patients.[24,31,36,39,40]

Overall, the present findings reinforce the close interplay between thyroid dysfunction, insulin resistance, systemic inflammation, and atherogenic dyslipidemia. Routine evaluation of metabolic and inflammatory biomarkers in patients with hypothyroidism may facilitate early cardiovascular risk stratification and improve clinical management through timely preventive and therapeutic strategies.

Clinical significance

Routine evaluation of serum insulin, hs-CRP, HOMA-IR, and LTI may assist in identifying hypothyroid patients at increased cardiovascular risk and may improve early therapeutic intervention and long-term clinical outcomes.

Limitations

Small sample size, single-center study, the cross-sectional design limits causal inference, lack of longitudinal follow-up and absence of a euthyroid control group were some limitations of the study.

CONCLUSION

The present study demonstrates that hypothyroidism is associated with significant metabolic, inflammatory, and atherogenic disturbances that may contribute to an

increased risk of cardiovascular disease. Serum TSH levels exhibited significant positive correlations with serum insulin, hs-CRP, and the LTI, suggesting that worsening thyroid dysfunction is accompanied by progressive insulin resistance, persistent low-grade systemic inflammation, and an increasingly adverse lipid profile.

Importantly, the observed alterations were evident in both overt and subclinical hypothyroidism, indicating that even mild thyroid dysfunction may have clinically meaningful cardiometabolic consequences. The elevated HOMA-IR values and inflammatory biomarkers observed in the study further support the role of thyroid hormone deficiency in the development of insulin resistance and chronic inflammatory processes, both of which are recognized contributors to cardiovascular morbidity.

The increased LTI also reflects enhanced atherogenic potential, underscoring the close interplay between thyroid dysfunction and cardiovascular risk. Collectively, these findings highlight the need for a comprehensive approach to the clinical evaluation of patients with hypothyroidism that extends beyond assessment of thyroid function alone. Incorporating metabolic, inflammatory, and atherogenic markers into routine clinical practice may facilitate earlier identification of individuals at increased cardiovascular risk, allowing timely therapeutic interventions and more effective long-term disease management.

Further prospective, multicenter studies with larger sample sizes are warranted to validate these findings and to determine whether early identification and management of these metabolic abnormalities can improve long-term cardiovascular outcomes in patients with hypothyroidism.

Recommendations

Larger multicenter studies are recommended. Longitudinal studies should be conducted to evaluate long-term cardiovascular outcomes. The impact of thyroid hormone replacement therapy on metabolic and inflammatory parameters should be assessed.

ACKNOWLEDGEMENTS

Authors would like to thank to Department of Medical Biochemistry, Index Medical College Hospital and Research Centre, for providing the necessary facilities, academic support, and institutional guidance to conduct this study. Also, to faculty members, technical staff, and participants involved in this study and to Prof. Ritesh Vishwakarma, Department of Medical Biochemistry for guidance.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: The study was approved by the Institutional Ethics Committee Malwanchal University under Registration Ref. No. MU/Acm/Ph.D./Med BioChem/2024/002.

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Cite this article as: Jamatia M, Sharma AK, Charak R. Biochemical correlation of serum insulin, high-sensitivity C reactive protein and lipid tetrad index with thyroid-stimulating hormone levels in patients with hypothyroidism. Int J Res Med Sci 2026;14:3379-86.