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Original Article

A Case-Control Study to Evaluate the Association Between Insulin Resistance and Thyroid Hormone Using Triglyceride Glucose Index Among Subclinical Hypothyroid Patients

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ABSTRACT

Background: Subclinical hypothyroidism (SCH) is characterized by elevated thyroid stimulating hormone (TSH) levels with normal free triiodothyronine (fT3) and free thyroxine (fT4) levels. Increasing evidence suggests that SCH is associated with metabolic abnormalities including dyslipidemia, obesity and insulin resistance (IR). IR plays an important role in the development of metabolic syndrome, cardiovascular diseases and diabetes mellitus. The Triglyceride Glucose (TyG) index has emerged as a simple and reliable surrogate marker for assessing IR. Therefore, evaluation of IR using TyG index in SCH patients may facilitate early identification of metabolic and cardiovascular risk.

Objectives: To assess IR using the TyG index among SCH patients compared to euthyroid controls and to determine the correlation between TyG index and thyroid profile parameters.

Materials and Methods: A hospital-based case-control study was conducted among 100 participants, including 50 SCH patients and 50 age- and gender-matched euthyroid controls. Demographic characteristics, body mass index (BMI), fasting blood sugar (FBS), Triglycerides (TG), thyroid profile parameters (TSH, fT3, and fT4), and TyG index were evaluated. Statistical analysis was performed using appropriate tests and statistical correlation analysis was carried out using Pearson correlation coefficient. A p-value of < 0.05 was considered statistically significant.

Results: The mean BMI was significantly higher in SCH patients (29.35 ± 6.05 kg/m²) as compared to controls (20.73 ± 2.09 kg/m², $p<0.001$). TG levels and TSH values were also significantly elevated in SCH subjects (221.92 ± 94.55 mg/dL and 8.93 ± 2.26 µIU/mL, respectively; $p<0.001$). The TyG index was significantly higher in SCH patients (4.86 ± 0.33) as compared to controls (4.52 ± 0.30, $p<0.001$). A statistically significant positive correlation was observed between TyG index and TSH among SCH patients ($r=0.286$, $p=0.04$).

Conclusion: SCH is associated with increased IR and metabolic abnormalities. TyG index may serve as a useful, simple and cost-effective surrogate marker for early identification of IR in SCH patients.

Keywords: *Subclinical hypothyroidism, Triglyceride glucose index, Insulin resistance, Thyroid hormones, Thyroid stimulating hormone.*

INTRODUCTION

Subclinical hypothyroidism (SCH) is a common endocrine disorder characterized by elevated serum levels of TSH (Thyroid stimulating hormone) with normal circulating levels of fT4 (free thyroxine) and fT3 (free triiodothyronine). SCH is a mild and early form of thyroid dysfunction with biochemical abnormalities in the absence of overt clinical manifestations [(1)].SCH has been reported to affect 3-15% of the adult population across the globe [(2)]. A multi-centric

epidemiological study from India reported a prevalence of 8.02% in adults and a significantly higher burden was observed in the female population [3]. A regional level cross sectional study among medical students of Vijayapura district, Karnataka showed a prevalence of 8 % implying a significant burden even among the young apparently healthy persons [4]. Considering SCH as the most prevalent thyroid disorder in the Indian population, early screening and timely clinical intervention is crucial to prevent progression to overt hypothyroidism and its associated metabolic disturbances, cardiovascular disease risk, dyslipidemia, obesity and Insulin resistance(IR) [1].

Thyroid hormones play an essential role in regulating carbohydrate metabolism, lipid metabolism, thermogenesis and energy homeostasis. They influence glucose production, glucose utilization, insulin secretion and peripheral insulin sensitivity. Any alteration in thyroid function can therefore significantly affect metabolic processes [5]. Previous studies have suggested that sub-clinical thyroid dysfunction may contribute to impaired glucose metabolism and reduced insulin sensitivity even before progression to overt hypothyroidism. Altered thyroid hormone levels can affect insulin-mediated glucose uptake in peripheral tissues, hepatic glucose production and lipid metabolism, thereby increasing the risk of IR and subsequent metabolic complications [6].

IR is a pathological condition in which normal concentrations of insulin fail to produce adequate biological responses in target tissues such as skeletal muscle, adipose tissue and liver. It is recognized as a key mechanism underlying metabolic syndrome, type 2 diabetes mellitus, cardiovascular disease and several endocrine disorders. The relationship between thyroid dysfunction and IR has become an area of growing interest because both conditions share common metabolic pathways[7]. Various mechanisms have been proposed to explain the association between SCH and IR, including altered adipokine secretion, chronic low-grade inflammation, changes in lipid metabolism and endothelial dysfunction [8].

Several methods are available for assessing IR, including the hyperinsulinemic-euglycemic clamp technique, homeostatic model assessment for insulin resistance (HOMA-IR), Quantitative insulin sensitivity check index (QUICKI) and fasting plasma insulin measurements. Although the hyperinsulinemic-euglycemic clamp is considered the gold standard for evaluating IR, it is expensive, technically demanding and not feasible for routine clinical use (9). Consequently, simpler surrogate markers have gained increasing attention in clinical research. One such marker is the TyG index (Triglyceride glucose index), which has emerged as a practical, cost-effective and reliable indicator of IR [10].

The TyG index is calculated using FBS(fasting blood glucose) and fasting TG (Triglyceride) values and has demonstrated a strong correlation with IR measured by standard methods. The index offers several advantages including simplicity, reproducibility and easy applicability in routine clinical practice [11], Previous investigations have shown that the TyG index may serve as a useful predictor of IR, metabolic syndrome, diabetes mellitus and cardiovascular disease. Since SCH is associated with abnormalities in glucose and lipid metabolism, assessment of IR using the TyG index may provide valuable insights into the metabolic alterations occurring in these patients [12].

Several studies have explored the relationship between thyroid hormone status and IR; however, findings remain inconsistent. Some studies have reported significantly increased IR among patients with SCH [7,13], whereas others have observed weak or insignificant associations [14,15]. Furthermore, limited studies have specifically evaluated IR using the TyG index among SCH patients and correlated it with thyroid profile parameters.

Therefore, the present case-control study was designed to evaluate the role of TyG index as a surrogate marker for IR among SCH patients as compared to euthyroid controls and to determine the correlation between TyG index and thyroid profile parameters; thereby improving understanding of the metabolic consequences associated with SCH at an early stage and preventing the long-term metabolic and cardiovascular complications.

MATERIAL AND METHODS

Study Setting:

The present study was conducted at a tertiary care teaching hospital as a hospital-based case-control study over a period of 6 months to evaluate the association between IR and thyroid hormone status using the TyG index among patients with SCH. The study aimed to assess IR using the TyG index in SCH patients as compared to euthyroid controls and determine the correlation between the TyG index and thyroid profile parameters. The study compared metabolic and thyroid parameters between SCH patients and healthy euthyroid individuals.

Inclusion Criteria

- Patients willing to participate and provide written informed consent.
- Patients aged 18–60 years.
- Patients diagnosed with SCH, defined by elevated TSH levels with normal fT3 and fT4 values.
- Age and gender-matched euthyroid healthy individuals.

Exclusion Criteria

- Patients with overt hypothyroidism.
- Patients with diabetes mellitus.
- Pregnant women.
- Patients with chronic liver disease, renal disease.
- Patients receiving thyroid hormone replacement therapy.
- Patients taking medications affecting thyroid or glucose metabolism.
- Individuals unwilling to participate.

Sample size

The study was conducted on 100 participants recruited by convenience sampling. The subjects were categorized into 50 SCH patients and 50 age- and gender-matched euthyroid controls based on their thyroid profile values. Euthyroid controls were considered as subjects with normal serum fT3 level between 1.71-3.71pg/mL, fT4 level between 0.7-1.48 ng/dL and TSH level between 0.35–4.94 µIU/mL. Subjects with normal fT4 and fT3 and serum TSH > 4.5 µIU/mL were categorized as those with SCH.

Study Data Collection

Data was collected using a structured case record form. Demographic details, clinical findings, anthropometric measurements and laboratory parameters were systematically recorded. The demographic parameters included age, gender, family history, personal history and past medical history. The anthropometric measurements included height (cm), weight (kg) and body mass index (BMI). The biochemical parameters included FBS, fasting TG, TyG index and thyroid profile parameters.

Ethical Considerations

Ethical clearance for the study was obtained from the Institutional Ethics Committee and Scientific Committee [IEC NO: MIMS/IEC/2024/874] prior to commencement of the study. Written informed consent was obtained from all participants before enrollment. Participation in the study was entirely voluntary and participants had the right to withdraw at any stage without affecting their medical care.

Study Procedure

Following approval from the Institutional Ethics and Scientific Committee, eligible participants were screened and recruited after obtaining written informed consent. Detailed demographic information and medical history were recorded using a structured proforma. Anthropometric measurements were recorded.

Participants were instructed to fast overnight for about 8–12 hours prior to biochemical investigation. The following morning, after confirming their fasting status and taking aseptic precautions, about 5 mL of venous blood was drawn into plain non-vacuum vacutainer and then centrifuged at 3500 rpm for 15-20 minutes. Then separated serum was used to estimate blood glucose using glucose-oxidase peroxidase method and triglyceride levels using glycerol phosphate oxidase (GPO) method and thyroid profile parameters including TSH, fT3 and fT4 were estimated using chemiluminescence microparticles immunoassay (CMIA).

Blood samples were analyzed in a fully automated Abbott Architect Analyzer. Quality control materials at normal and pathological levels were assayed to ensure that the values of the biochemical parameters were within range - prior to analysis of patient samples.

The TyG index was calculated using the formula:

$$\textbf{Triglyceride-glucose index (TyG)} = \ln \frac{\text{Fasting triglyceride mg/dl} \times \text{Fasting glucose mg/dl}}{2}$$

Statistical Analysis

Data was analyzed using IBM SPSS Statistics version 22. Continuous variables were summarized as mean ± standard deviation (SD). Categorical variables were expressed as frequencies and percentages. To compare continuous variables such as the TyG index, TSH, fT3 and fT4 between SCH patients and euthyroid controls, the Independent Student t-test was used. Pearson's correlation was used to assess the relationship between TyG index, BMI and thyroid profile parameters. A value close to +1 indicates a strong positive correlation, while a value close to -1 indicates a strong negative correlation. A p-value of < 0.05 was considered statistically significant. Scatter plots were used to visually examine these associations.

RESULTS

The study included a total of 100 participants, with 50 SCH patients and 50 age- and gender-matched euthyroid controls. In the control group, the majority of participants were in the 21–30 years age group (36%), followed by 41–50 years (26%), 31–40 years (22%) and 51–60 years (16%). In the SCH group, most participants were in the 31–40 years age group (28%), followed by 51–60 years (26%), 41–50 years (24%) and 21–30 years (22%).

Table 1: Distribution of study participants according to age group and gender among control and SCH groups

	Age group (years)				Count
Group	21 - 30	31 - 40	41 - 50	51 - 60	N=100
Control	18 (36%)	11 (22%)	13 (26%)	8 (16%)	50 (11males, 39 females)
SCH	11 (22%)	14 (28%)	12 (24%)	13 (26%)	50 (12males, 38 females)

The gender distribution was similar in both groups. The control group consisted of 11 males(22%) and 39 females(78%), while the SCH group included 12 males(24%) and 38 females(76%).

Table 2: Comparison of demographic, anthropometric, biochemical, thyroid profile parameters and TyG index between control and SCH groups

	Control group	**SCH group**	**p- value**
Age [years]	37.42 ± 12.18	40.74 ± 11.50	0.164
BMI [kg/m2]	20.73 ± 2.09	29.35 ± 6.05	<0.001*
FBS [mg/dl]	82.6 ± 12.26	86.46 ± 9.91	0.087
TG [mg/dL]	119 ± 49.80	221.92 ± 94.55	<0.001*
TSH [µIU/mL]	2.178 ± 1.04	8.93 ± 2.26	<0.001
fT3 [pg/mL]	2.706 ± 0.50	2.65 ± 0.56	0.600
fT4 [ng/dL]	0.996 ± 0.11	1.046 ± 0.85	0.683
TyG INDEX	4.52 ± 0.30	4.86 ± 0.33	<0.001*

*p-value of < 0.05 was considered statistically significant.

The mean age of the SCH group was 40.74 ± 11.50 years, while the control group had a mean age of 37.42 ± 12.18 years. This difference was not statistically significant (p = 0.164), confirming age comparability between groups. BMI was significantly higher in the SCH group (29.35 ± 6.05 kg/m²) compared to the control group (20.73 ± 2.09 kg/m²) with a p-value of <0.001. FBS levels were slightly higher in the SCH group (86.46 ± 9.91 mg/dL) than in controls (82.6 ± 12.26 mg/dL), but this difference was not statistically significant (p = 0.087). The TG showed a significant elevation in the SCH group (221.92 ± 94.55 mg/dL) compared to the control group (119 ± 49.80 mg/dL) with a p-value of <0.001.

TSH levels were significantly higher in the SCH group (8.93 ± 2.26 µIU/mL) compared to controls (2.18 ± 1.04 µIU/mL)
with a p-value of <0.001.There were no significant differences in fT3 (2.65 ± 0.56 pg/mL vs. 2.70 ± 0.50 pg/mL, p = 0.600) or fT4 levels (1.046 ± 0.85 ng/dL vs. 0.996 ± 0.11 ng/dL, p = 0.683) between the control and SCH groups.

TyG Index was significantly higher in the SCH group (4.86 ± 0.33) compared to the control group (4.52 ± 0.30) with a p-value of <0.001.

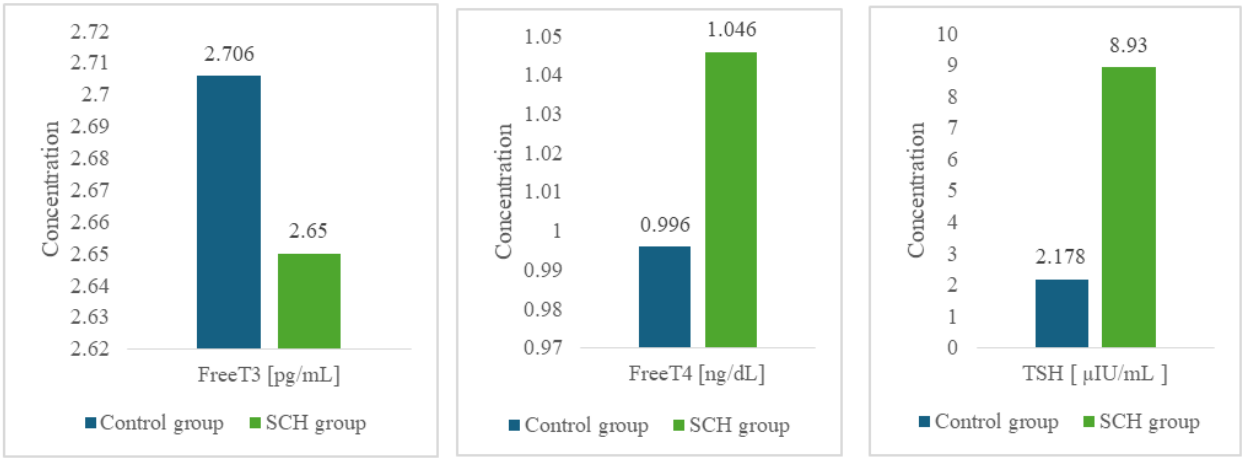

Fig 1:Comparison of thyroid profile parameters between control and SCH groups

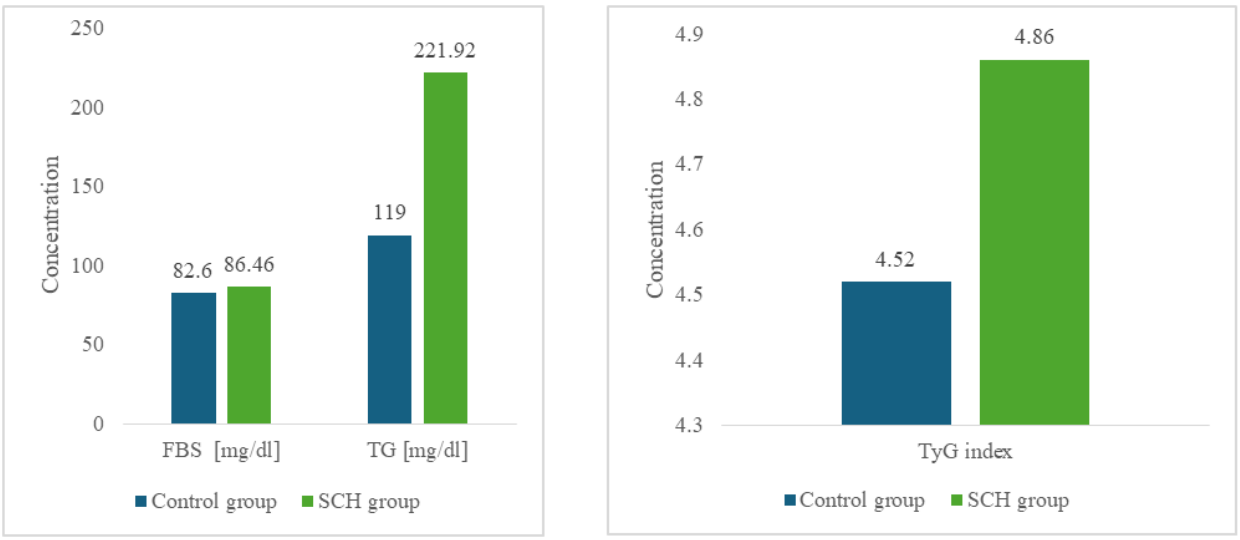

Fig. 2: Comparison of FBS, fasting TG and TyG index between control and SCH groups

Table 3: Correlation of TyG index with thyroid profile parameters (fT3, fT4 and TSH) in control and SCH groups

	Control			SCH		
	fT3	fT4	TSH	fT3	fT4	TSH
TyG Index	r=0.23	r=-0.151	p=0.05	r=0.15	r=0.19	r=0.286
	p=0.10	p=0.29	p=0.71	p=0.28	p=0.182	p=0.04*

*p-value of < 0.05 was considered statistically significant.

Pearson correlation analysis revealed no statistically significant association between the TyG index and thyroid profile parameters (fT3, fT4 and TSH) in the control group. In the SCH group, although weak positive correlations were observed between the TyG index and both fT3 ($r = 0.15$; $p = 0.28$) and fT4 ($r = 0.19$; $p = 0.182$); these were not statistically significant. Notably, a statistically significant positive correlation was found between the TyG index and TSH levels in the SCH group ($r = 0.286$; $p = 0.04$).

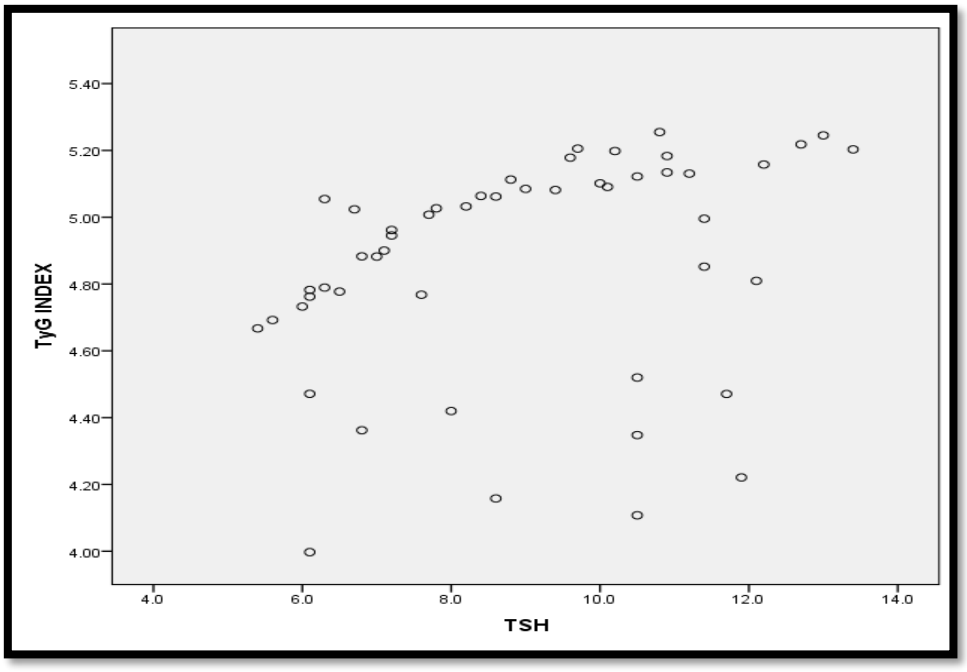

Fig 4: Scatter plot showing the correlation between TSH and TyG index in the SCH group.

DISCUSSION

Hypothyroidism is increasingly recognized as metabolic disorder with far-reaching consequences beyond classical symptom; particularly due to its association with IR, dyslipidemia, obesity and cardiovascular diseases. It has been

proven that overt hypothyroidism leads to increased risk of cardiovascular and metabolic disease.Mahajan et al. (2025) found that 64.3% of overt hypothyroid patients develop IR, with HOMA-IR rising to 5.00 and fasting insulin reaching 17.66 µIU/mL compared to 1.65 in individuals with normal thyroid levels (16). Nada et al. (2013) further showed that in overt hypothyroidism TG levels correlate directly with TSH and inversely with fT3 thereby augmenting the risk of serious cardiovascular disease (17).

SCH has been associated with several cardiovascular abnormalities, including endothelial dysfunction, increased systemic vascular resistance, impaired left ventricular diastolic function, dyslipidemia, and accelerated atherosclerosis (18,19). These changes may contribute to an increased risk of coronary artery disease, heart failure and cardiovascular mortality particularly in individuals with TSH levels ≥10 mIU/L or those with pre-existing cardiovascular disease (1–3). SCH exerts measurable metabolic effects even before progressing to overt disease. Yang et al. (2023) demonstrated that SCH worsens IR despite normal blood glucose levels (20), a finding quantitatively supported by Sridevi et al. (2012) who reported higher HOMA-IR scores (2.30 vs. 1.78) and higher total cholesterol levels (169.37±32.83 vs. 154.5±18.71 mg/dL) in SCH patients as compared to euthyroid controls(21). This IR does not act in isolation but combines with elevated triglycerides to amplify cardiovascular risk, as shown by Suh and Kim (2015) (18). Left untreated, these subclinical derangements can intensify substantially. Timely treatment of SCH represents a critical intervention point that can interrupt the insulin resistance–dyslipidemia–cardiovascular disease cascade before it progresses to the more severe metabolic and cardiac complications seen in overt hypothyroidism. Pandrc et al. (2017) demonstrated that early levothyroxine therapy normalizes thyroid function and lipid profiles while significantly improving the quality of life (22).

The present case-control study was conducted to evaluate the association between IR and thyroid hormone status using the TyG index among SCH patients and euthyroid controls and to determine the correlation between TyG index and thyroid profile parameters. A total of 100 participants were included comprising of 50 SCH patients and 50 euthyroid controls. The demographic distribution demonstrated comparable age and gender match between the groups. In the control group, the majority of subjects belonged to the 21–30 years age group (36%), while in the SCH group most participants belonged to the 31–40 years age category (28%). Females predominated in both groups with 39 females and 11 males in controls and 38 females and 12 males in SCH subjects thereby indicating greater prevalence of thyroid disorders among females. Similar female predominance has been reported in a study by Cooper and Biondi et al, who described SCH as being considerably more common among women because of autoimmune and hormonal influences on thyroid function (1). Likewise, another study by Peeters et al reported that SCH occurs more frequently in females and middle-aged individuals, particularly after the third decade of life; supporting the demographic characteristics observed in the present study (23).

The present study showed that the mean age in the SCH group (40.74 ± 11.50 years) was slightly higher than controls (37.42 ± 12.18 years), although the difference was statistically insignificant ($p=0.164$); suggesting appropriate age comparability between groups. This finding was similar to the study conducted by Thakur et al. (2024) where age- and gender-matched SCH patients and controls were selected to minimize confounding effects while evaluating TyG index and thyroid profile parameters (24).

Body mass index was significantly elevated among SCH patients (29.35 ± 6.05 kg/m²) as compared to controls (20.73 ± 2.09 kg/m²; $p<0.001$). The observed increase in BMI may be explained by metabolic slowing, reduced thermogenesis, altered lipid metabolism, and decreased energy expenditure associated with thyroid dysfunction. Similar observations were reported by Okan K et al. (2022) who found BMI values to be significantly higher in SCH patients (24.04 ± 3.84 kg/m²) as compared to healthy controls (22.48 ± 2.74 kg/m²) (25). Likewise, Cui et al. (2025) also reported a relationship between increasing BMI and elevated IR markers including TyG index (26). These findings indicate that excess body weight may contribute to IR and metabolic abnormalities in SCH patients.

FBS values in the current study were slightly higher among SCH subjects (86.46 ± 9.91 mg/dL) than controls (82.6 ± 12.26 mg/dL); although the difference was not statistically significant ($p=0.087$). The absence of significant alterations in glucose levels may indicate that early thyroid dysfunction does not directly influence fasting glycemic status, especially in non-diabetic individuals. However, IR may still be present despite normal fasting glucose values because insulin resistance changes may precede alterations in blood glucose levels. Thakur et al. (2024) observed no significant difference in fasting blood glucose between SCH patients and euthyroid controls despite demonstrating a significantly higher TyG index in SCH subjects (24). Likewise, Inayatet al. reported that fasting glucose values often remain within the normal range in SCH patients, whereas the TyG index reflects underlying insulin resistance and metabolic dysfunction with more accuracy (27).

TG levels showed significant elevation among SCH patients (221.92 ± 94.55 mg/dL) as compared to controls (119 ± 49.80 mg/dL; $p<0.001$). Elevated TG in SCH may result from decreased lipoprotein lipase activity and impaired lipid metabolism associated with reduced thyroid hormone action. Similar findings were observed by Okan K et al. (2022)

who reported significantly elevated serum TG levels in SCH patients (25). Dyslipidemia has been recognized as one of the important metabolic consequences of SCH and may contribute to increased cardiovascular disease risk.
TSH values in the present study were significantly elevated among SCH patients (8.93 ± 2.26 μIU/mL) as compared to controls (2.18 ± 1.04 μIU/mL; p<0.001). These findings are in agreement with Thakur et al. (2024) who reported significantly higher TSH values among SCH patients (6.6 ± 1.7 μIU/mL) as compared to controls (2.5 ± 1.2 μIU/mL; p<0.0001) (24). In contrast, fT3 and fT4 levels in the present study did not differ significantly between SCH and controls (fT3: p=0.600; fT4: p=0.683) which is expected because SCH is characterized by elevated TSH levels with normal circulating thyroid hormone concentrations.

The principal finding of the present study was the significantly elevated TyG index among SCH patients (4.86 ± 0.33) as compared to euthyroid controls (4.52 ± 0.30; p<0.001), indicating increased IR among SCH subjects. These findings strongly support the hypothesis that SCH may contribute to increase in IR and associated metabolic dysfunction even in the absence of overt hypothyroidism. Similar observations were reported by Thakur et al. (2024) who found significantly increased TyG index values in SCH patients (4.8 ± 0.2) as compared to controls (4.7 ± 0.2; p=0.015) (24). Okan K et al. (2022) also demonstrated increased IR among SCH patients and concluded that SCH may act as a risk factor for metabolic syndrome and cardiovascular disease (25). Furthermore, Inayat et al. (2025) observed that TyG index served as an important predictor of metabolic health and thyroid dysfunction in SCH patients (27).

The correlation analysis in the present study demonstrated no statistically significant relationship between TyG index and fT3 or fT4 levels in either group. However, a significant positive correlation was observed between TyG index and TSH levels among SCH patients (r=0.286; p=0.04). The scatter plot findings further supported this positive association between increasing TSH and TyG index values. Similar findings were reported by Zhang et al. (2024) who demonstrated a linear positive association between TyG index and TSH levels and suggested that higher TyG index values significantly increased the risk of IR (28). Cui et al. (2025) also found positive correlations between thyroid hormones and TyG index across different BMI groups (22). However, Thakur et al. (2024) did not observe a statistically significant correlation between TyG index and thyroid profile parameters (24). The discrepancy may be related to differences in sample size, population characteristics and study methodology.

Overall, the findings of the present study indicate that SCH is associated with a statistically significant increase in BMI, dyslipidemia and TyG index values. The study findings also indicate a statistically significant positive relationship between TyG index and TSH levels among individuals with SCH. These observations support the concept that SCH contributes to IR and metabolic disturbances and suggest that TyG index may serve as a useful and simple surrogate marker for early identification of IR among SCH patients ensuring the prevention of long-term complications of metabolic and cardiovascular dysfunctions.

Strengths of the Study:
The strength of the current study lies in the use of a clinically viable and reproducible method to measure TyG index. It provides an efficient substitute to the complex hyperinsulinemic-euglycemic clamp for the estimation of IR.

Limitations of the Study:
Despite the clinical contribution made by the current study, there are several limitations inherent to it that need to be taken into consideration. First of all, it needs to be noted that the study has been carried out is a hospital-based study employing the method of convenience sampling with a limited number of participants. Although the TyG index was used as a marker, no direct comparisons were done with the gold standard method (hyperinsulinemic-euglycemic clamp).

CONCLUSION

The present study concluded that patients with SCH demonstrated significantly increased IR and metabolic alterations as compared to euthyroid controls. Although age distribution was comparable between the groups, females were found to have a higher preponderance to SCH. Participants in the SCH group exhibited significantly higher BMI, TG levels, TSH levels and TyG index values as compared to euthyroid controls. The TyG index, which serves as a simple and cost-effective surrogate marker of IR, was significantly elevated in SCH subjects as compared to euthyroid controls; thereby indicating increased IR among participants with SCH. Furthermore, a significant positive correlation between TyG index and TSH levels in SCH group suggesting that an increase in thyroid dysfunction may contribute to worsening IR. These findings emphasize the importance of early metabolic screening among SCH patients and indicate that TyG index may be a useful surrogate marker for identifying individuals at higher risk of IR and related metabolic complications.

Statements and declaration:
Conflicts of interest: the authors declare that they do not have conflict of interest.
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REFERENCES

1. Cooper DS, Biondi B. Subclinical thyroid disease. Lancet. 2012;379(9821):1142–54. doi:10.1016/S0140-6736(11)60276-6 PubMed PMID: 22273398.
2. Gosi SKY, Kaur J, Garla VV. Subclinical Hypothyroidism. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2026 [cited 2026 Jul 1]. Available from: http://www.ncbi.nlm.nih.gov/books/NBK536970/ PubMed PMID: 30725655.
3. Rajput R, Bajaj S, Kalra P, Menon AS, Pillai MG, Ghosh S, et al. Subclinical hypothyroidism in adults: Consensus statement of Indian thyroid society. Thyroid Research and Practice. 2022;19(1):8. doi:10.4103/trp.trp_4_23
4. Totad RD. Sub-Clinical Hypothyroidism-A Cross-Sectional Study among Medical Students in Vijayapura District Karnataka. 2022.
5. Brenta G. Why can insulin resistance be a natural consequence of thyroid dysfunction? J Thyroid Res. 2011;2011:152850. doi:10.4061/2011/152850 PubMed PMID: 21941681; PubMed Central PMCID: PMC3175696.
6. Dimitridis G, Mitrou P, Lambadiari V, Boutati E, Maratou E, Panagiotakos DB, et al. Insulin action in adipose tissue and muscle in hypothyroidism. J Clin Endocrinol Metab. 2006;91(12):4930–7. doi:10.1210/jc.2006-0478 PubMed PMID: 17003097.
7. Maratou E, Hadjidakis DJ, Kollias A, Tsegka K, Peppa M, Alevizaki M, et al. Studies of insulin resistance in patients with clinical and subclinical hypothyroidism. Eur J Endocrinol. 2009;160(5):785–90. doi:10.1530/EJE-08-0797 PubMed PMID: 19141606.
8. Chaker L, Bianco AC, Jonklaas J, Peeters RP. Hypothyroidism. Lancet. 2017;390(10101):1550–62. doi:10.1016/S0140-6736(17)30703-1 PubMed PMID: 28336049; PubMed Central PMCID: PMC6619426.
9. Guerrero-Romero F, Simental-Mendía LE, González-Ortiz M, Martínez-Abundis E, Ramos-Zavala MG, Hernández-González SO, et al. The product of triglycerides and glucose, a simple measure of insulin sensitivity. Comparison with the euglycemic-hyperinsulinemic clamp. J Clin Endocrinol Metab. 2010;95(7):3347–51. doi:10.1210/jc.2010-0288 PubMed PMID: 20484475.
10. Vasques ACJ, Novaes FS, de Oliveira M da S, Souza JRM, Yamanaka A, Pareja JC, et al. TyG index performs better than HOMA in a Brazilian population: a hyperglycemic clamp validated study. Diabetes Res Clin Pract. 2011;93(3):e98–100. doi:10.1016/j.diabres.2011.05.030 PubMed PMID: 21665314.
11. Navarro-González D, Sánchez-Íñigo L, Pastrana-Delgado J, Fernández-Montero A, Martinez JA. Triglyceride–glucose index (TyG index) in comparison with fasting plasma glucose improved diabetes prediction in patients with normal fasting glucose: The Vascular-Metabolic CUN cohort. Preventive Medicine. 2016;86:99–105. doi:10.1016/j.ypmed.2016.01.022
12. Simental-Mendía LE, Rodríguez-Morán M, Guerrero-Romero F. The product of fasting glucose and triglycerides as surrogate for identifying insulin resistance in apparently healthy subjects. Metab Syndr Relat Disord. 2008;6(4):299–304. doi:10.1089/met.2008.0034 PubMed PMID: 19067533.
13. Singh BM, Goswami B, Mallika V. Association between insulin resistance and hypothyroidism in females attending a tertiary care hospital. Indian J Clin Biochem. 2010;25(2):141–5. doi:10.1007/s12291-010-0026-x PubMed PMID: 23105900; PubMed Central PMCID: PMC3453097.
14. Gutch M, Kumar S, Razi SM, Gupta KK, Gupta A. Assessment of insulin sensitivity/resistance. Indian J Endocrinol Metab. 2015;19(1):160–4. doi:10.4103/2230-8210.146874 PubMed PMID: 25593845; PubMed Central PMCID: PMC4287763.
15. Kirac CO, Sirikci V, Findikli HA. Comparison of triglyceride-glucose index and HOMA-IR as indicators of insulin resistance in obese women with subclinical hypothyroidism [Internet]. 2022 Dec. doi:10.15584/ejcem.2022.4.5
16. Mahajan J, Varma A, Kumar S, Acharya S, Shukla S, Kothari M, et al. Cross-sectional analysis of insulin resistance in hypothyroid patients at rural teaching hospital: An endocrinal synergy. J Family Med Prim Care. 2025;14(2):743–8. doi:10.4103/jfmpc.jfmpc_1579_24 PubMed PMID: 40115550; PubMed Central PMCID: PMC11922372.
17. Nada AM. Effect of treatment of overt hypothyroidism on insulin resistance. World J Diabetes. 2013;4(4):157–61. doi:10.4239/wjd.v4.i4.157 PubMed PMID: 23961327; PubMed Central PMCID: PMC3746089.
18. Suh S, Kim DK. Subclinical Hypothyroidism and Cardiovascular Disease. Endocrinol Metab (Seoul). 2015;30(3):246–51. doi:10.3803/EnM.2015.30.3.246 PubMed PMID: 26248862; PubMed Central PMCID: PMC4595347.
19. Jabbar A, Pingitore A, Pearce SHS, Zaman A, Iervasi G, Razvi S. Thyroid hormones and cardiovascular disease. Nat Rev Cardiol. 2017 Jan;14(1):39–55. doi:10.1038/nrcardio.2016.174 PubMed PMID: 27811932.
20. Yang W, Jin C, Wang H, Lai Y, Li J, Shan Z. Subclinical hypothyroidism increases insulin resistance in normoglycemic people. Front Endocrinol (Lausanne). 2023;14:1106968. doi:10.3389/fendo.2023.1106968 PubMed PMID: 37484968; PubMed Central PMCID: PMC10358968.

21.Sridevi A, Vivekanand B, Giridhar G, Mythili A, Subrahmanyan KAV. Insulin resistance and lipid alterations in subclinical hypothyroidism. Indian J Endocrinol Metab. 2012;16(Suppl 2):S345–6. doi:10.4103/2230-8210.104085 PubMed PMID: 23565422; PubMed Central PMCID: PMC3603070.
22. Pandrc MS, Ristić A, Kostovski V, Stanković M, Antić V, Milin-Lazović J, et al. The Effect of Early Substitution of Subclinical Hypothyroidism on Biochemical Blood Parameters and the Quality of Life. J Med Biochem. 2017;36(2):127–36. doi:10.1515/jomb-2017-0007 PubMed PMID: 28680356; PubMed Central PMCID: PMC5471645.
23. Peeters RP. Subclinical Hypothyroidism. N Engl J Med. 2017;376(26):2556–65. doi:10.1056/NEJMcp1611144 PubMed PMID: 28657873.
24. Thakur R, Kumar S, Neeraj RK, Saleem M, Kumar C, Mohan L. Evaluation of the Association between Insulin Resistance and Subclinical Hypothyroidism Using Triglyceride-Glucose Index: A Cross-Sectional Study. Maedica (Bucur). 2024;19(2):255–9. doi:10.26574/maedica.2024.19.2.255 PubMed PMID: 39188835; PubMed Central PMCID: PMC11345053.
25. Okan K, Sencan M, Duman G. The relationship between thyroid hormone levels, insulin resistance and body mass index, in patients with subclinical hypothyroidism and euthyroid patients. International Journal of Endocrinology (Ukraine). 2022;18(1):41–8. doi:10.22141/2224-0721.18.1.2022.1144
26. Cui R, Li G, Wei Y, Liu J, Wang Y, Leng S, et al. Thyroid Hormone Resistance Indices and Their Correlations with Insulin Resistance in Chinese Euthyroid Subjects. Int J Endocrinol. 2025;2025:5397686. doi:10.1155/ije/5397686 PubMed PMID: 41040505; PubMed Central PMCID: PMC12488306.
27. Inayat F, Shams S, Ullah N, Ahmad Shah AW, Haq IU. Exploring the associations of the triglyceride–glucose index with thyroid function in subclinical hypothyroid patients: a cross-sectional study. Lipids Health Dis. 2025;24:337. doi:10.1186/s12944-025-02740-1 PubMed PMID: 41121321; PubMed Central PMCID: PMC12539201.
28. Zhang C, Wang H, Li Y, Wang X, Han Y, Gao X, et al. Association between the triglyceride-glucose index and thyroid disorders: a cross-sectional survey and Mendelian randomization analysis. Endocrine. 2024;86(1):173–85. doi:10.1007/s12020-024-03858-5 PubMed PMID: 38782862.