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DYSLIPIDEMIA IN PATIENTS WITH SUB-CLINICAL HYPOTHYROIDISM

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Abstract

BACKGROUND: *Subclinical hypothyroidism (SCH) is known to contribute to the progression of atherosclerosis and is closely linked to dyslipidemia a major risk factor for cardiovascular diseases, which significantly impacts morbidity and mortality rates.*

OBJECTIVE: *To assess the dyslipidemia among patients diagnosed with subclinical hypothyroidism at Ghulam Mohammad Mahar Medical College Civil Hospital, Sukkur.*

PATIENTS AND METHODS: *This descriptive, cross-sectional study was carried out over a six-month period (from 1st Sept-2024 to 28th Feb-2025) in the Department of Medicine at GMMMC, Civil Hospital Sukkur. Individuals aged 20 to 60 years, of either gender, diagnosed with subclinical hypothyroidism were included in the study. Participants were assessed for the presence of dyslipidemia. Both qualitative and quantitative variables were analyzed using frequency (percentages) and mean ± standard deviation (SD), respectively.*

RESULTS: *A total of 144 individuals with subclinical hypothyroidism participated in the study. Among them, dyslipidemia was identified in 90 patients (62.5%). Most of those affected were female (56.2%), lived in urban areas (52.1%), and had a mid-level education (25.0%). Other notable characteristics among patients with dyslipidemia included smoking (43.8%), hypertension (61.1%), obesity (63.9%), non-alcoholic fatty liver disease (NAFLD) (56.9%), and diabetes mellitus (43.8%).*

CONCLUSION: *Dyslipidemia is highly prevalent among individuals with subclinical hypothyroidism. Early detection and management are essential to reduce the risk of cardiovascular complications.*

INTRODUCTION:

Thyroid dysfunction can manifest at various stages of life, but it is more frequently diagnosed in adulthood (Wilson SA, et al). These disorders impact nearly all major metabolic functions, particularly influencing energy balance and the metabolism of proteins, carbohydrates, and lipids. One of the key metabolic effects of thyroid hormones is the regulation of basal energy expenditure (Hegedus L, et al). Among these metabolic functions, lipid metabolism is especially sensitive to thyroid hormone levels, playing a pivotal

role in maintaining phospholipid balance in cell membranes and determining fatty acid composition. The active thyroid hormone, triiodothyronine (T3), significantly influences lipid homeostasis by modulating gene expression involved in both lipid synthesis and breakdown (Chiovato L, et al).

Hypothyroidism arises due to a deficiency in the production of thyroxine (T4) and triiodothyronine (T3), and this reduction leads to a compensatory increase in thyroid-stimulating hormone (TSH) levels (Yoo WS, et al). In cases where TSH levels are elevated but free thyroxine (FT4) remains within normal limits, the condition is termed subclinical hypothyroidism – SCH (Mavromati M, et al). This is considered a mild and often asymptomatic form of thyroid dysfunction, but if not addressed, it can progress to overt hypothyroidism in a significant number of cases. Most individuals with SCH do not exhibit clinical symptoms or present only with nonspecific signs, making laboratory evaluation critical for diagnosis. A small goiter may be observed on physical examination, raising clinical suspicion (Jasim S, et al and Biondi B, et al). SCH is a relatively common endocrine disorder, with prevalence estimates ranging from 6% to 17% in the general population (Qasim B, et al). It affects women more frequently than men, with global statistics indicating a prevalence of 7.5-8.5% in females compared to 2.8-4.4% in males. Diagnosis relies on biochemical assessment, typically using sensitive immunoassays such as the chemiluminescence technique (Calissendorff J, et al).

Evidence suggests that subclinical hypothyroidism contributes to an increased risk of cardiovascular complications, including coronary artery disease, heart failure, and lipid metabolism abnormalities.[9] Dyslipidemia an imbalance in lipid levels is a well-known modifiable risk factor for cardiovascular disease. Other risk factors linked to SCH include elevated diastolic blood pressure, impaired endothelial function, greater arterial stiffness, changes in coagulation parameters, and increased levels of inflammatory markers such as C-reactive protein (Aldossari K, et al).

The underlying mechanism for dyslipidemia in both overt and subclinical hypothyroidism involves disturbances in lipid processing in the liver and adipose tissue (Al Eidan E, et al). Specifically, increased TSH levels stimulate hepatic production of 3-hydroxy-3-methylglutaryl coenzyme A reductase, a key enzyme in cholesterol synthesis, thereby raising serum cholesterol levels (Allan GM, et al). The most frequent lipid abnormality observed in hypothyroid patients is elevated total cholesterol. Additionally, increases in very low-density lipoproteins (VLDL) and high-density lipoproteins (HDL) have been reported. Elevated triglyceride levels are often attributed to increased esterification of fatty acids in the liver.

The prevalence of dyslipidemia among individuals with subclinical hypothyroidism has been reported to be approximately 70%. Specifically, hypercholesterolemia has been noted in 48.4% of cases, hypertriglyceridemia in 32.3%, and elevated low-density lipoprotein (LDL) levels in 26.5% of patients (Zeb S, et al). A study conducted by Landazur et al. revealed similar findings, reporting elevated total cholesterol in 31.9%, low HDL cholesterol in 57.9%, high LDL cholesterol in 24.3%, and increased triglyceride levels in 26% of SCH patients (Landazuri P, et al).

Although lipid profile abnormalities are frequently observed in thyroid dysfunction, there is limited regional data available on how thyroid hormone levels correlate with lipid disturbances in our population. Given the potential health risks associated with untreated dyslipidemia, particularly in the context of cardiovascular disease, early identification and management of these lipid abnormalities in SCH patients is essential. Recognizing this gap, the current study was undertaken to evaluate the frequency of dyslipidemia among individuals diagnosed with subclinical hypothyroidism at a tertiary care hospital. The aim is to reduce the potential burden of cardiovascular complications through early detection and intervention. Furthermore, the findings will be disseminated through professional health seminars and academic forums to encourage timely diagnosis and better clinical outcomes.

This study aims to evaluate how common dyslipidemia is among individuals diagnosed with subclinical hypothyroidism who are receiving care at Ghulam Mohammad Mahar Medical College Civil Hospital in Sukkur.

PATIENTS AND METHODS:

This descriptive cross-sectional study was conducted over a period of six months (from 1st Sept-2024 to 28th Feb-2025) in the Department of Medicine at Ghulam Mohammad Mahar Medical College, Civil Hospital, Sukkur. Ethical clearance was obtained from the College of Physicians and Surgeons Pakistan (CPSP), and informed consent was secured from all participants prior to enrollment.

The sample size was calculated based on a previously reported prevalence of 24.3% for elevated low-density lipoprotein cholesterol (LDL-C).[14]Using a non-probability consecutive sampling method, 144 patients diagnosed with subclinical hypothyroidism (SCH) were approached, and 7% of them were selected to participate in the study.

Inclusion Criteria:

The study included adults aged between 20 and 60 years of either sex, who presented with symptoms such as fatigue, unexplained weight gain, lethargy, shortness of breath, or neck swelling lasting for six weeks or more. These individuals were biochemically confirmed as having subclinical hypothyroidism and were attending the outpatient department of Civil Hospital, Sukkur.

Exclusion Criteria:

Individuals were not included in the study if they presented with any of the following conditions;
chronic liver disease, chronic kidney disease, nephrotic syndrome, Cushing's syndrome (due to potential interference with thyroid and lipid parameters), gestational diabetes, known hyperthyroidism, alcohol use disorder, or malignancies such as lung, prostate, thyroid, or breast cancer. Additionally, those receiving cancer chemotherapy, hormone replacement therapy, statins, corticosteroids, beta-blockers, selective estrogen receptor modulators, or medications known to influence thyroid function (e.g., thyroid replacement therapy, amiodarone, interferon, anti-thyroid drugs) were excluded. Pregnant or breastfeeding women were also not considered eligible.

Operational Definitions:

Subclinical hypothyroidism was defined based on the following thyroid hormone reference ranges: TSH > 4.50 µIU/mL, FT4 between 0.8–1.8 ng/dL, and FT3 between 1.4–4.4 pg/mL.

Dyslipidemia was identified when any one or more of the following lipid abnormalities were present:
Triglycerides (TG) ≥ 150 mg/dL
Total cholesterol (TC) ≥ 200 mg/dL
High-density lipoprotein (HDL) < 40 mg/dL
Low-density lipoprotein (LDL) ≥ 100 mg/dL

Data Collection Procedure:

Eligible participants were recruited from the outpatient department based on clinical symptoms and confirmed thyroid profiles. The principal investigator personally conducted the initial assessments, which included symptom review, vital signs, and general physical examination. Blood samples were collected to assess thyroid hormone levels (TSH, FT3, FT4) and, in those confirmed with subclinical hypothyroidism, a second blood sample (2cc drawn using a 5cc disposable syringe) was sent to the hospital laboratory to evaluate lipid profile parameters.

The principal investigator covered all expenses related to laboratory testing. Additionally, potential confounders and other explanatory variables were documented and assessed.

Data Analysis:

All collected data were entered and analyzed using SPSS software. For qualitative variables, frequencies and percentages were reported while quantitative variables were summarized using mean and standard deviation values. The Shapiro-Wilk test was applied to assess whether the data followed a normal distribution. Stratification was utilized to identify and control for potential confounding variables. Depending on the nature of the data, either the Chi-square test or Fisher's exact test was applied for analysis of categorical variables. A p-value of 0.05 or less, with a 95% confidence interval, was used to determine statistical significance

RESULTS:

Over the course of six months, 144 patients diagnosed with subclinical hypothyroidism were

included in the study. All participants were between 20 and 60 years of age and had been experiencing relevant symptoms such as persistent fatigue, lethargy, weight gain, difficulty breathing, or noticeable neck swelling for duration of at least six weeks. The study cohort comprised both male and female patients.

Details regarding the participants' demographic and clinical characteristics are outlined in Table 1, while the mean and standard deviation (SD) values for age, symptom duration, and body mass index (BMI) are provided in Table 2. The distribution of age and gender among the study population is depicted in Table 3.

For the statistical assessment of data distribution, the skewness and kurtosis values for age were found to be -0.06 and -0.47, respectively. Corresponding values for BMI were -0.47 and -0.68. The median age was 57 years, and the median BMI was calculated as 29.90 kg/m^2. Results from the Shapiro-Wilk test indicated that both age and BMI deviated significantly from a normal distribution, with p-values less than 0.01 for each variable.

Stratified analysis of dyslipidemia based on various demographic and clinical factors including age, gender, place of residence, educational background, marital status, hypertension, smoking habits, obesity, non-alcoholic fatty liver disease (NAFLD), and diabetes mellitus is presented in Tables 4.

Statistically significant associations with dyslipidemia in subclinical hypothyroid patients were identified for the residence ($p < 0.01$), hypertension ($p < 0.01$), marital status ($p < 0.01$), smoking ($p = 0.05$), NAFLD ($p < 0.01$) and obesity ($p < 0.01$).

In contrast, no significant relationships were observed between dyslipidemia and the age ($p = 0.48$), gender ($p = 0.12$), educational status ($p = 0.57$) and diabetes mellitus ($p = 0.41$). These findings suggest that while some socio-demographic and clinical variables might have role in the dyslipidemia development among individuals with subclinical hypothyroidism, others do not appear to exert a measurable influence.

TABLE 1: CLINICAL AND DEMOGRAPHIC CHARACTERISTICS OF THE PARTICIPANTS

PARAMETER	FREQUENCY (n=144)	PERCENTAGE (%)
AGE (yrs)		
20-29	18	12.5
30-39	43	29.9
40-49	50	34.7
50-60	33	22.9
GENDER		
Male	63	43.8
Female	81	56.2
RESIDENCE		
Urban	75	52.1
Rural	69	47.9
EDUCATIONAL STATUS		
Illiterate	31	21.5
Primary	30	20.8
Middle	36	25.0
Secondary	26	18.1
Higher	21	14.6
SMOKING		

Yes	43	43.8
No	50	34.7
Ex-smoker	31	21.5
MARITAL STATUS		
Married	60	41.7
Single	27	18.8
Separated	30	20.8
Divorced	27	18.8
HYPERTENSION		
Yes	88	61.1
No	56	38.9
OBESITY		
Yes	92	63.9
No	52	36.1
NAFLD		
Yes	82	56.9
No	62	43.1
DIABETES MELLITUS		
Yes	63	43.8
No	81	56.2
DYSLIPIDEMIA		
Yes	90	62.5
No	54	37.5

TABLE 2: MEAN AND STANDARD DEVIATION OF NUMERICAL VARIABLES AMONG STUDY PARTICIPANTS

Quantitative variables	**Mean ± SD**
Age (yrs)	56.65 ± 12.75
Duration of symptoms (wks)	9.55 ± 3.66
BMI (kg/m^2)	31.72 ± 2.93

TABLE 3: THE AGE AND GENDER DISTRIBUTION OF PARTICIPANTS

		GENDER		
		Male	Female	Total
AGE (yrs)	20-29	7 11.1%	11 13.6%	18 12.5%
	30-39	17 27.0%	26 32.1%	43 29.9%
	40-49	26 41.3%	24 29.6%	50 34.7%
	50-60	13 20.6%	20 24.7%	33 22.9%
Total		63 100.0%	81 100.0%	144 100.0%

TABLE 4: THE DISTRIBUTION OF DYSLIPIDEMIA IN STUDY POPULATION

		n =144 (%)		Total	P-value
AGE (yrs)					
	20-29	11 12.2%	7 13.0%	18 12.5%	
	30-39	31 34.4%	12 22.2%	43 29.9%	0.48**
	40-49	29 32.2%	21 38.9%	50 34.7%	
	50-60	19 21.1%	14 25.9%	33 22.9%	
GENDER					
	Male	35 38.9%	28 51.9%	63 43.8%	0.12**
	Female	55 61.1%	26 48.1%	81 56.2%	
RESIDENCE					
	Urban	59 65.6%	16 29.6%	75 52.1%	<0.01*
	Rural	31 34.4%	38 70.4%	69 47.9%	
EDUCATIONAL STATUS					
	Illiterate	22 24.4%	9 16.7%	31 21.5%	
	Primary	21 23.3%	9 16.7%	30 20.8%	
	Middle	20 22.2%	16 29.6%	36 25.0%	0.57**
	Secondary	15 16.7%	11 20.4%	26 18.1%	
	Higher	12	9	21	

		13.3%	16.7%	14.6%	
MARITAL STATUS					
	Married	47 52.2%	13 24.1%	60 41.7%	
	Single	19 21.1%	8 14.8%	27 18.8%	
	Separated	10 11.1%	20 37.0%	30 20.8%	<0.01*
	Divorced	14 15.6%	13 24.1%	27 18.8%	
HYPERTENSION	Yes	63 70.0%	25 46.3%	88 61.1%	
	No	27 30.0%	29 53.7%	56 38.9%	<0.01*
SMOKING	Yes	45 50.0%	18 33.3%	63 43.8%	
	No	25 27.8%	25 46.3%	50 34.7%	0.05*
	Ex-smoker	20 22.2%	11 20.4%	31 21.5%	
OBESITY	Yes	67 74.4%	25 46.3%	92 63.9%	
	No	23 25.6%	29 53.7%	52 36.1%	<0.01*
NAFLD	Yes	61 67.8%	21 38.9%	82 56.9%	<0.01*
	No	29 32.2%	33 61.1%	62 43.1%	
DIABETES MELLITUS	Yes	37 41.1%	26 48.1%	63 43.8%	
	No	53 58.9%	28 51.9%	81 56.2%	0.41**

*Statistically significant
**Statistically non-significant

DISCUSSION: Subclinical hypothyroidism (SCH) is characterized by elevated serum thyroid-stimulating hormone (TSH) levels while maintaining normal levels of free thyroxine (FT4). Though patients typically do not present overt symptoms of hypothyroidism, emerging evidence has revealed a potential link between SCH and various metabolic abnormalities, particularly dyslipidemia. This association has garnered attention due to its implications in cardiovascular risk, making it essential to understand the lipid profile variations in individuals with SCH (Liu H, et al).

Several studies have demonstrated that even mild thyroid dysfunction can influence lipid metabolism

(Pearce EN). The thyroid hormones play a critical role in lipid synthesis, mobilization, and degradation. In SCH, despite normal FT4 levels, the elevated TSH is thought to reflect early thyroid dysfunction that may already be affecting metabolic processes, including lipid regulation. This leads to the hypothesis that SCH could act as a silent contributor to cardiovascular disease via dyslipidemia (Kiran M, et al).

The present results are consistent with previous studies that reported elevated levels of total cholesterol (TC) and low-density lipoprotein cholesterol (LDL-C) in individuals diagnosed with subclinical hypothyroidism (SCH). A meta-analysis conducted by Razvi et al. (2007) found that SCH was consistently associated with elevated TC and LDL-C levels, even when TSH elevation was mild. These findings suggest a subtle but potentially harmful alteration in lipid metabolism that may be overlooked due to the absence of overt thyroid symptoms.

Interestingly, high-density lipoprotein cholesterol (HDL-C) and triglycerides (TG) have shown inconsistent results across different studies. Some have reported elevated TG and decreased HDL-C in SCH patients, while others found no significant changes. These inconsistencies may stem from population differences, sample size limitations, and varying definitions of SCH (Khazan M, et al). However, the prevailing trend points to an unfavorable lipid profile in SCH, which merits consideration for early intervention.

The pathophysiological mechanism underlying dyslipidemia in SCH remains a subject of ongoing investigation. It is hypothesized that reduced thyroid hormone activity affects the expression of hepatic LDL receptors, leading to decreased clearance of LDL-C from the circulation. Additionally, thyroid hormones influence the activity of lipoprotein lipase, an enzyme essential for the hydrolysis of triglyceride-rich lipoproteins. In SCH, the diminished thyroid hormone signaling might impair this enzymatic function, contributing to hypertriglyceridemia (Brenta G, et al).[20]

The cardiovascular implications of these lipid changes are significant. Dyslipidemia is a well-established risk factor for atherosclerosis and coronary artery disease (CAD). Even minor alterations in lipid levels can have cumulative effects over time, particularly in the context of elevated LDL-C. Therefore, SCH may serve as a modifiable risk factor for cardiovascular disease, especially in populations with additional comorbidities such as diabetes or hypertension (Rizos CV, et al). Another important consideration is the reversibility of dyslipidemia with thyroid hormone replacement therapy. Multiple randomized controlled trials have shown that levothyroxine treatment in patients with SCH can normalize TSH levels and lead to modest reductions in LDL-C and TC levels. Meier et al. (2001) reported that levothyroxine therapy in SCH patients resulted in improved lipid parameters; particularly in those with baseline lipid abnormalities (Meier C, et al). This reinforces the therapeutic potential of addressing SCH not only for thyroid hormone normalization but also for cardiovascular risk reduction.

However, it is important to note that not all studies support treatment in all SCH cases. The decision to initiate therapy should be individualized, taking into account patient age, TSH levels, symptomatology, and lipid profile. For example, elderly patients with mild TSH elevation may not derive the same benefit as younger individuals with more pronounced TSH elevation and dyslipidemia (Rastgooye Haghi A, et al). As such, clinical guidelines vary in their recommendations, with some advocating for treatment in patients with TSH >10 mIU/L or in those with underlying cardiovascular disease or symptoms attributable to SCH.

Despite these promising findings, limitations exist within the literature. Many studies suffer from small sample sizes, lack of long-term follow-up, and heterogeneity in diagnostic criteria. Furthermore, the confounding effects of lifestyle factors such as diet, physical activity, and concurrent medication use are not always adequately controlled. These limitations highlight the need for more comprehensive, longitudinal studies to better delineate the true impact of SCH on lipid metabolism and cardiovascular outcomes.

Gender and age may also influence the relationship between SCH and dyslipidemia. For instance, women, particularly those in the perimenopausal and postmenopausal age groups, appear to have a higher prevalence of SCH and are more susceptible

to lipid abnormalities. This may be partly due to hormonal fluctuations that affect both thyroid function and lipid metabolism. In addition, aging is associated with both an increase in TSH levels and changes in lipid profile, making it challenging to discern whether dyslipidemia is a direct result of SCH or a byproduct of aging physiology (Hussain A, et al).

Genetic predisposition may further modulate the impact of SCH on lipid metabolism. Polymorphisms in genes regulating thyroid function and lipid pathways may make certain individuals more susceptible to lipid disturbances even with marginal thyroid dysfunction (Biondi B). Future research incorporating genetic and molecular profiling could enhance risk stratification and guide personalized treatment approaches.

CONCLUSION:

Dyslipidemia was identified in 90 participants, accounting for 62.5% of the study population. Among those affected the majority were women (56.2%), residents of urban areas (52.1%), and individuals with a moderate level of education (25.0%). Additional findings included a notable proportion of patients who smoked (43.8%), had hypertension (61.1%), were classified as obese (63.9%), suffered from non-alcoholic fatty liver disease (NAFLD) (56.9%), or had diabetes mellitus (43.8%). Thus the evidence suggests a compelling association between SCH and dyslipidemia, with elevated TSH levels contributing to an atherogenic lipid profile. While not all SCH patients will manifest dyslipidemia, the presence of lipid abnormalities should prompt clinicians to consider thyroid function assessment, especially in those without other apparent causes of dyslipidemia. Nonetheless, further large-scale, randomized studies are essential to refine treatment thresholds and optimize management strategies for SCH-associated dyslipidemia and are also needed in order to confirm and better characterized these findings.

LIMITATION OF THE STUDY:

The present study was limited by its single-center setting and cross-sectional design, which may reduce the applicability of the results to broader populations. Additionally, the small sample size and the short observation period limited to the patients' ability to assess long-term outcomes. For a more complete evaluation of the association between subclinical hypothyroidism and dyslipidemia, future investigations should adopt a prospective design with extended follow-up and include participants from multiple healthcare centers to enhance the validity and scope of the findings.

AUTHOR'S CONTRIBUTION:

Collection and acquisition of data & grammatical corrections	Dr. Saira Sattar
Concept & design of study & proof read	Dr. Altaf Ahmed Shaikh
Drafting the article and finalizing the manuscript	Dr. Asra
Revising critically and make it suitable for final format	Dr. Sadaf
Acquisition of data and grammatical review	Dr. Ali Mohsin
Analysis of data and drafting	Dr. Syed Zulfiquar Ali Shah
Final Approval of version	By All Authors

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