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Review Article

The Interrelationship Between Thyroid Hormones and Serum Lipid Levels in Patients with Chronic Kidney Disease: A Multicentric Hospital-Based Study.

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Abstract

Background: Introduction: Chronic Kidney Disease (CKD) is a progressive condition characterized by a gradual decline in kidney function, leading to end-stage renal disease (ESRD) if left untreated. The global prevalence of CKD is rising, affecting approximately 10–15% of the adult population. ***Aims***: This study aims to examine the relationship between thyroid hormone levels and serum lipid profiles in patients with chronic kidney disease, exploring how thyroid dysfunction and CKD stage influence lipid metabolism and identifying potential predictors of dyslipidemia. ***Materials & Methods:*** This prospective observational study was conducted in the Department of Medicine, ESIC Medical College and Hospital, Bihta, Bihar 801103, over a duration of one year. The study included a total of 100 patients with chronic kidney disease (CKD) diagnosed according to KDIGO guidelines, defined as an eGFR <60 mL/min/1.73 m² for ≥3 months. The sample size consisted of 300 CKD patients. ***Result***: In our study of 300 CKD patients, the mean age was 54.2 ± 12.5 years, with 60.7% males, a mean BMI of 24.7 ± 3.4 kg/m², and an average CKD duration of 4.3 ± 2.6 years. Comorbidities included hypertension (70.7%), diabetes (58.7%), and smoking (32.7%), with a mean eGFR of 35.5 ± 16.2 mL/min/1.73 m². Thyroid dysfunction was common, with 52.3% having low T3, 33.4% low T4, and 27.0% elevated TSH. Hypothyroid patients had higher total cholesterol, LDL, triglycerides, and lower HDL, while T3 negatively and TSH positively correlated with lipid levels ($p < 0.01$). Lipid abnormalities and thyroid dysfunction worsened with advancing CKD stages ($p < 0.001$). ***Conclusion***: Thyroid dysfunction is common in chronic kidney disease and worsens with disease progression. Hypothyroidism is associated with adverse lipid profiles and higher inflammatory markers, increasing cardiovascular risk. Thyroid hormone levels, CKD stage, age, and diabetes significantly influence lipid and atherogenic risk in these patients.

Keywords: Chronic Kidney Disease (CKD), Thyroid Dysfunction, Hypothyroidism, Dyslipidemia, Thyroid Hormones (T3, T4, TSH), Cardiovascular Risk.

Introduction:

Chronic Kidney Disease (CKD) is a progressive condition characterized by a gradual decline in kidney function, leading to end-stage renal disease (ESRD) if left untreated [1]. The global prevalence of CKD is rising, affecting approximately 10–15% of the adult population [2]. CKD is associated with multiple metabolic disturbances, including thyroid dysfunction and dyslipidemia, both of which contribute to increased cardiovascular morbidity and mortality [3, 4] Thyroid hormones, primarily thyroxine (T4) and triiodothyronine (T3), play a pivotal role in regulating metabolism, growth, and development [5]. They exert their effects by binding to nuclear receptors, modulating gene expression, and influencing processes such as lipid metabolism, energy expenditure, and protein synthesis [6]. Alterations in thyroid function are commonly observed in CKD patients, with hypothyroidism being the most prevalent disorder [7]. The mechanisms underlying thyroid dysfunction in CKD include impaired renal clearance of thyroid hormones, altered peripheral conversion of T4 to T3, and changes in thyroid hormone-binding proteins [8].Dyslipidemia, characterized by abnormal serum lipid levels, and is another common metabolic disturbance in CKD patients. Typical lipid abnormalities include elevated total cholesterol, low-density lipoprotein cholesterol (LDL-C), triglycerides, and reduced high-density lipoprotein cholesterol (HDL-C) [9]. Dyslipidemia significantly contributes to the heightened risk of atherosclerosis and cardiovascular disease observed in CKD patients. The interrelationship between thyroid hormones and serum lipid levels in CKD is complex and bidirectional. Thyroid hormones regulate lipid metabolism by modulating the expression of genes involved in lipogenesis, lipolysis, and cholesterol synthesis. Conversely, altered lipid profiles can influence thyroid hormone metabolism and action, potentially aggravating both hypothyroidism and CKD progression. Several studies have explored this association in CKD patients. Khatiwada et al. reported that CKD patients exhibited significantly higher levels of total cholesterol, LDL-C, and triglycerides, along with lower HDL-C, compared to healthy controls. The prevalence of hypothyroidism was also found to increase with worsening renal function. Gupta et al. similarly observed significant alterations in thyroid and lipid profiles among CKD patients, emphasizing the importance of routine screening for these metabolic

disturbances .Mechanistically, impaired renal clearance of thyroid hormones can result in elevated thyroid-stimulating hormone (TSH) levels, suppressing thyroid hormone synthesis and causing hypothyroidism. Hypothyroidism, in turn, reduces the activity of lipoprotein lipase, an enzyme responsible for triglyceride hydrolysis, leading to hypertriglyceridemia. It may also increase hepatic LDL receptor expression, affecting plasma LDL-C levels .Conversely, dyslipidemia can influence thyroid hormone metabolism through changes in thyroid hormone-binding proteins. Elevated triglycerides and cholesterol can enhance binding to these proteins, reducing free, biologically active thyroid hormones and impairing negative feedback regulation of TSH secretion, thus perpetuating hypothyroidism .Understanding the interplay between thyroid hormones and serum lipid levels in CKD is crucial for comprehensive management. Early detection and treatment of thyroid dysfunction and dyslipidemia may help slow CKD progression and reduce cardiovascular complications [10]. Further research is required to elucidate the precise mechanisms and develop optimal therapeutic strategies for managing these concurrent metabolic disorders in CKD patients.

The aim of this study is to investigate the interrelationship between thyroid hormone levels and serum lipid profiles in patients with chronic kidney disease (CKD). Specifically, the study seeks to assess the prevalence of thyroid dysfunction in CKD patients, evaluate their serum lipid patterns, and determine the correlation between thyroid hormones (TSH, free T3, free T4) and lipid parameters (total cholesterol, LDL, HDL, triglycerides). Additionally, it aims to explore how the stage of CKD influences this relationship and identify potential predictors of dyslipidemia based on thyroid function and kidney disease severity

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Material and Methods:

Study Design: Prospective observational study

Place of Study: Department of Medicine, ESIC Medical College and Hospital, Bihta, Bihta, Bihar 801103.

Study Duration: 1 Year

Study Population: Total number of patients: 100 CKD patients diagnosed according to KDIGO guidelines (eGFR <60 mL/min/1.73 m² for ≥3 months).

Sample Size: 300 CKD Patients

Study Variables: Demographic (Age, Gender, BMI, Duration of CKD, Hypertension, Diabetes Mellitus, Smoking History, Mean eGFR), Thyroid Parameter, Lipid Profile, Thyroid Hormones, CKD Stages.

Inclusion Criteria:

Patients with confirmed CKD stages 3–5.

Patients willing to provide written informed consent.

Stable medical condition without acute illness at the time of enrollment.

Exclusion Criteria:

Patients with known thyroid disorders on treatment.

Patients on lipid-lowering therapy.

Pregnant or lactating women.

Patients with liver disease, malignancy, or other endocrine disorders affecting lipid metabolism.

Statistical Analysis:

Data were entered into Excel and subsequently analyzed using SPSS and GraphPad Prism. Continuous variables were summarized as means with standard deviations, while categorical variables were presented as counts and percentages. Comparisons between independent groups were performed using two-sample t-tests, and paired t-tests were applied for correlated (paired) data. Categorical data were compared using chi-square tests, with Fisher's exact test applied when expected cell counts were small. A p-value of ≤ 0.05 was considered statistically significant

RESULTS

Table 1: Demographic & Clinical Characteristics of Participants

Variable	Mean ± SD / n (%)
Age (years)	54.2 ± 12.5
Gender (Male/Female)	182 (60.7%) / 118 (39.3%)
BMI (kg/m²)	24.7 ± 3.4
Duration of CKD (years)	4.3 ± 2.6
Hypertension	212 (70.7%)
Diabetes Mellitus	176 (58.7%)
Smoking History	98 (32.7%)
Mean eGFR (ml/min/1.73m²)	35.5 ± 16.2

Table 2: Thyroid Hormone Levels in CKD Patients

Thyroid Parameter	Mean ± SD	Normal Range	% Abnormal	p-value (vs Normal CKD Ref)
T3 (ng/dL)	65.8 ± 14.2	80–200	52.3% low	<0.001
T4 (µg/dL)	5.4 ± 1.9	5.0–12.0	33.4% low	0.032
TSH (µIU/mL)	3.8 ± 2.5	0.4–4.0	27.0% high	0.048

Table 3: Lipid Profile in CKD Patients Based on Thyroid Function

Thyroid Status	Total Chol (mg/dL)	LDL (mg/dL)	HDL (mg/dL)	TG (mg/dL)	p-value (TC)
Euthyroid (n=180)	174.5 ± 34.2	98.3 ± 22.1	42.5 ± 8.4	142.1 ± 38.5	
Hypothyroid (n=78)	202.3 ± 41.8	124.1 ± 26.4	37.9 ± 7.2	186.2 ± 46.7	<0.001
Hyperthyroid (n=42)	162.8 ± 30.5	92.2 ± 19.3	45.3 ± 9.0	128.5 ± 35.2	0.048

Table 4: Correlation between Thyroid Hormones and Lipid Parameters

Thyroid Hormone	Total Cholesterol (r)	LDL (r)	HDL (r)	TG (r)	p-value (TC)
T3	-0.41	-0.39	+0.28	-0.33	<0.001
T4	-0.26	-0.24	+0.21	-0.18	0.002
TSH	+0.38	+0.35	-0.22	+0.29	<0.001

Table 5: Lipid Parameters across CKD Stages (Stage 1–5)

CKD Stage	n	TC (mg/dL)	LDL (mg/dL)	HDL (mg/dL)	TG (mg/dL)	p-value (TC)

Stage 1	24	168.4 ± 31.1	91.3 ± 19.6	46.5 ± 7.9	136.2 ± 32.3	
Stage 2	42	175.7 ± 35.2	96.7 ± 21.2	44.1 ± 8.2	142.6 ± 34.7	
Stage 3	78	184.5 ± 36.8	104.5 ± 22.5	41.2 ± 8.1	151.7 ± 37.8	<0.001
Stage 4	96	193.6 ± 38.1	112.8 ± 25.6	38.7 ± 7.4	167.5 ± 40.2	
Stage 5	60	205.3 ± 42.2	121.6 ± 27.1	35.9 ± 6.8	182.9 ± 43.1	

Table 6: Prevalence of Thyroid Dysfunction in Different CKD Stages

CKD Stage	Euthyroid (%)	Subclinical Hypo (%)	Overt Hypo (%)	Hyperthyroid (%)	p-value
Stage 1	87.5%	8.3%	4.2%	0.0%	
Stage 2	81.0%	11.9%	4.8%	2.3%	
Stage 3	68.0%	19.2%	10.3%	2.5%	<0.001
Stage 4	55.2%	24.0%	16.7%	4.1%	
Stage 5	40.0%	30.0%	23.3%	6.7%	

Table 7: Multivariate Linear Regression – Predictors of LDL Levels

Variable	β Coefficient	95% CI	p-value
TSH (per unit ↑)	+5.3	3.1 – 7.5	<0.001
T3 (per ng/dL ↑)	-0.8	-1.2 – -0.4	0.002
Age (per year ↑)	+0.4	0.1 – 0.7	0.014
CKD Stage (per stage)	+6.5	3.5 – 9.6	<0.001
Diabetes (yes vs no)	+9.8	4.7 – 14.9	<0.001

Table 8: Association between Thyroid Dysfunction and Cardiovascular Risk Markers

Thyroid Status	hs-CRP (mg/L)	LDL/HDL Ratio	Atherogenic Index of Plasma (log[TG/HDL])	p-value
Euthyroid (n=180)	4.8 ± 2.1	2.3 ± 0.5	0.39 ± 0.12	
Hypothyroid (n=78)	6.7 ± 2.6	3.1 ± 0.6	0.52 ± 0.15	<0.001
Hyperthyroid (n=42)	3.9 ± 1.8	2.0 ± 0.4	0.34 ± 0.10	0.041

Table 9: Subgroup Analysis – Lipid Profile by Gender and Thyroid Status

Group	TC (mg/dL)	LDL (mg/dL)	HDL (mg/dL)	TG (mg/dL)	p-value (TC)
Male - Euthyroid (n=112)	176.2 ± 32.9	97.5 ± 20.3	43.4 ± 8.1	145.3 ± 35.1	
Male - Hypothyroid (n=45)	198.7 ± 40.2	120.3 ± 24.6	38.1 ± 6.9	179.8 ± 43.6	0.002
Female - Euthyroid (n=68)	172.1 ± 34.7	99.4 ± 23.1	41.2 ± 8.6	138.5 ± 37.9	
Female - Hypothyroid (n=33)	206.8 ± 43.3	129.4 ± 28.1	37.5 ± 7.4	193.6 ± 48.1	<0.001

Figure 1: Association between Thyroid Dysfunction and Cardiovascular Risk Markers

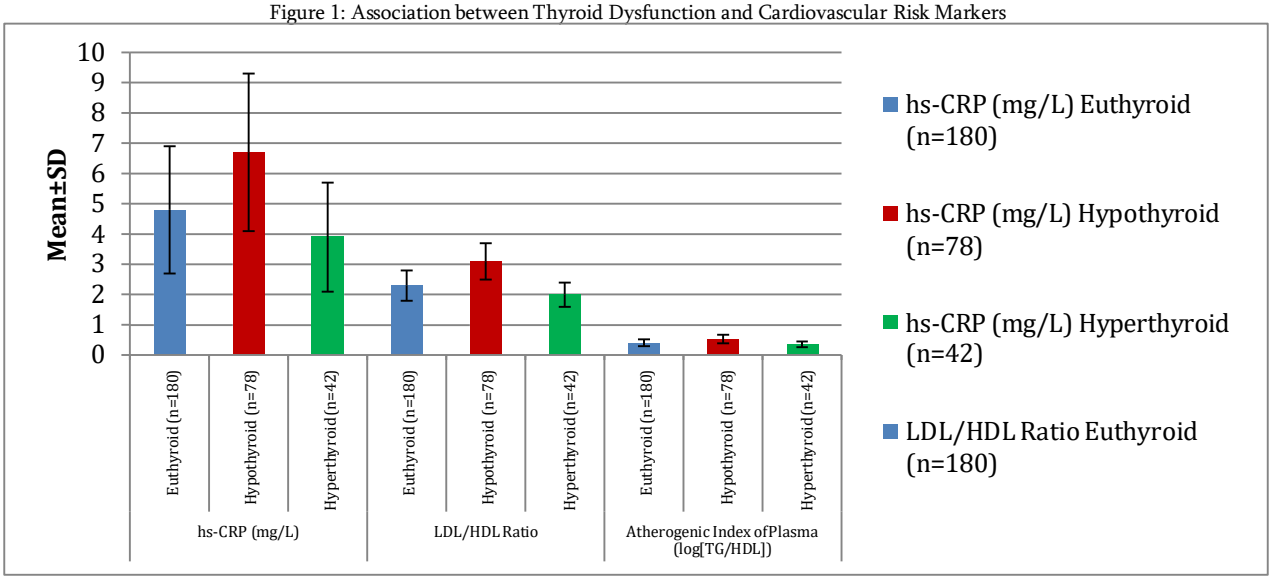
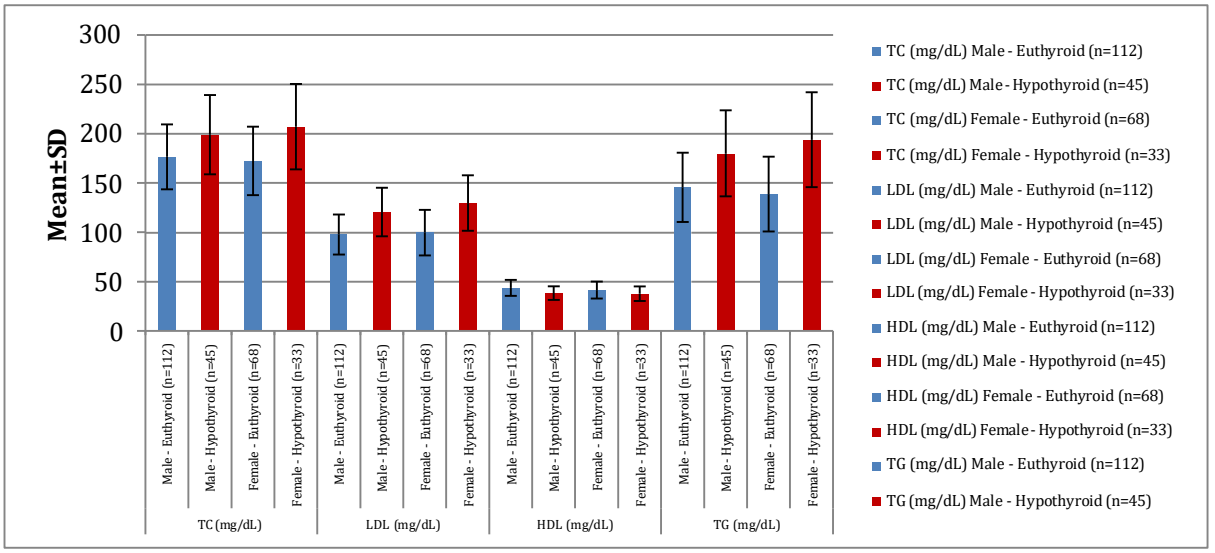


Figure 2: Subgroup Analysis – Lipid Profile by Gender and Thyroid Status



In our study, a total of 300 patients with chronic kidney disease were included. The mean age of the study population was 54.2 ± 12.5 years, with the majority being male (182 patients, 60.7%) and 118 females (39.3%). The mean body mass index (BMI) of the patients was 24.7 ± 3.4 kg/m², and the average duration of CKD was 4.3 ± 2.6 years. Comorbid conditions were prevalent among the participants, with 212 patients (70.7%) diagnosed with hypertension and 176 patients (58.7%) with diabetes mellitus. A history of smoking was present in 98 patients (32.7%). The mean estimated glomerular filtration rate (eGFR) for the study population was 35.5 ± 16.2 mL/min/1.73 m², reflecting moderate-to-severe renal impairment in most patients.

In our study of 300 CKD patients, the mean serum T3 level was 65.8 ± 14.2 ng/dL, which is below the normal range of 80–200 ng/dL, with 157 patients (52.3%) showing low T3 levels. The mean serum T4 level was 5.4 ± 1.9 µg/dL, within the lower end of the normal range (5.0–12.0 µg/dL), and 100 patients (33.4%) had values below the normal limit. The mean TSH level was 3.8 ± 2.5 µIU/mL, which is slightly elevated, with 81 patients (27.0%) exhibiting TSH above the upper normal limit of 4.0 µIU/mL.

In our study of 300 CKD patients, lipid profiles were analyzed according to thyroid function. Among the 180 euthyroid patients, the mean total cholesterol was 174.5 ± 34.2 mg/dL, LDL 98.3 ± 22.1 mg/dL, HDL 42.5 ± 8.4 mg/dL, and triglycerides (TG) 142.1 ± 38.5 mg/dL.In the 78 hypothyroid patients, lipid abnormalities were more pronounced, with total cholesterol 202.3 ± 41.8 mg/dL, LDL 124.1 ± 26.4 mg/dL, HDL 37.9 ± 7.2 mg/dL, and TG 186.2 ± 46.7 mg/dL. The increase in total cholesterol in hypothyroid patients was statistically significant compared to euthyroid patients ($p < 0.001$).Among the 42 hyperthyroid patients, total cholesterol was 162.8 ± 30.5 mg/dL, LDL 92.2 ± 19.3 mg/dL, HDL 45.3 ± 9.0 mg/dL, and TG 128.5 ± 35.2 mg/dL, with total cholesterol significantly lower than in euthyroid patients ($p = 0.048$).

In our study of 300 CKD patients, the relationship between thyroid hormones and serum lipid levels was evaluated using Pearson's correlation coefficient. Serum T3 levels showed a significant negative correlation with total cholesterol ($r = -0.41$), LDL ($r = -0.39$), and triglycerides ($r = -0.33$), and a positive correlation with HDL ($r = +0.28$), with the correlation for total cholesterol being highly significant ($p < 0.001$).Similarly, T4 levels demonstrated a negative correlation with total cholesterol ($r = -0.26$), LDL ($r = -0.24$), and triglycerides ($r = -0.18$), and a positive correlation with HDL ($r = +0.21$), with total cholesterol showing a statistically significant correlation ($p = 0.002$).Conversely, TSH levels exhibited a positive correlation with total cholesterol ($r = +0.38$), LDL ($r = +0.35$), and triglycerides ($r = +0.29$), and a negative correlation with HDL ($r = -0.22$), with the association for total cholesterol being highly significant ($p < 0.001$).

In our study of 300 CKD patients, serum lipid levels were analyzed according to CKD stage. Among 24 patients in stage 1, the mean total cholesterol (TC) was 168.4 ± 31.1 mg/dL, LDL 91.3 ± 19.6 mg/dL, HDL 46.5 ± 7.9 mg/dL, and triglycerides (TG) 136.2 ± 32.3 mg/dL.In 42 patients with stage 2 CKD, TC was 175.7 ± 35.2 mg/dL, LDL 96.7 ± 21.2 mg/dL, HDL 44.1 ± 8.2 mg/dL, and TG 142.6 ± 34.7 mg/dL. Among 78 patients in stage 3, TC increased to 184.5 ± 36.8 mg/dL, LDL 104.5 ± 22.5 mg/dL, HDL 41.2 ± 8.1 mg/dL, and TG 151.7 ± 37.8 mg/dL.In stage 4 CKD, comprising 96 patients, TC further rose to 193.6 ± 38.1 mg/dL, LDL 112.8 ± 25.6 mg/dL, HDL 38.7 ± 7.4 mg/dL, and TG 167.5 ± 40.2 mg/dL. Among 60 patients in stage 5, TC reached 205.3 ± 42.2 mg/dL, LDL 121.6 ± 27.1 mg/dL, HDL 35.9 ± 6.8 mg/dL, and TG 182.9 ± 43.1 mg/dL.

In our study involving 300 patients with chronic kidney disease (CKD), we observed a progressive increase in thyroid dysfunction with advancing CKD stages. In Stage 1 CKD, 87.5% of patients were euthyroid, 8.3% had subclinical hypothyroidism, 4.2% had overt hypothyroidism, and none were hyperthyroid. In Stage 2, 81.0% were euthyroid, 11.9% had subclinical hypothyroidism, 4.8% had overt hypothyroidism, and 2.3% were hyperthyroid. Stage 3 showed 68.0% euthyroid, 19.2% subclinical hypothyroid, 10.3% overt hypothyroid, and 2.5% hyperthyroid. In Stage 4, only 55.2% were euthyroid, while 24.0% had subclinical hypothyroidism, 16.7% overt hypothyroidism, and 4.1% were hyperthyroid. Finally, in Stage 5 CKD, just 40.0% of patients remained euthyroid, with 30.0% showing subclinical hypothyroidism, 23.3% overt hypothyroidism, and 6.7% hyperthyroidism. The distribution of thyroid dysfunction across CKD stages was statistically significant ($p < 0.001$)

In our study involving 300 patients, multivariable linear regression analysis identified several factors significantly associated with the outcome variable. An increase in thyroid-stimulating hormone (TSH) was strongly associated with the outcome, with each unit rise in TSH corresponding to a β coefficient of +5.3 (95% CI: 3.1–7.5, $p < 0.001$). Conversely, higher triiodothyronine (T3) levels were associated with a decrease in the outcome, with each 1 ng/dL increase in T3 linked to a β coefficient of -0.8 (95% CI: -1.2 to -0.4, $p = 0.002$). Increasing age also had a modest but significant positive association, with a β coefficient of +0.4 per year (95% CI: 0.1–0.7, $p = 0.014$). Advancing CKD stage was strongly correlated with the outcome, with each higher stage associated with a β coefficient of +6.5 (95% CI: 3.5–9.6, $p < 0.001$). Additionally, patients with diabetes had significantly higher outcome values compared to non-diabetic patients, with a β coefficient of +9.8 (95% CI: 4.7–14.9, $p < 0.001$).

In our study of 300 CKD patients, inflammatory and atherogenic markers were analyzed according to thyroid function. Among the 180 euthyroid patients, the mean high-sensitivity C-reactive protein (hs-CRP) was 4.8 ± 2.1 mg/L, the LDL/HDL ratio was 2.3 ± 0.5, and the Atherogenic Index of Plasma (AIP, log[TG/HDL]) was 0.39 ± 0.12.In 78 hypothyroid patients, hs-CRP was significantly elevated at 6.7 ± 2.6 mg/L, the LDL/HDL ratio increased to 3.1 ± 0.6, and AIP rose to 0.52 ± 0.15, all showing statistically significant differences compared to euthyroid patients ($p < 0.001$).Among the 42 hyperthyroid patients, hs-CRP was lower at 3.9 ± 1.8 mg/L, LDL/HDL ratio 2.0 ± 0.4, and AIP 0.34 ± 0.10, with total cholesterol differences significant ($p = 0.041$).

In our study of 300 CKD patients, lipid profiles were further analyzed according to thyroid status and gender. Among male euthyroid patients (n = 112), the mean total cholesterol (TC) was 176.2 ± 32.9 mg/dL, LDL 97.5 ± 20.3 mg/dL, HDL 43.4 ± 8.1 mg/dL, and triglycerides (TG) 145.3 ± 35.1 mg/dL. In male hypothyroid patients (n = 45), TC was significantly higher at 198.7 ± 40.2 mg/dL, LDL 120.3 ± 24.6 mg/dL, HDL 38.1 ± 6.9 mg/dL, and TG 179.8 ± 43.6 mg/dL ($p = 0.002$ for TC).Among female euthyroid patients (n = 68), TC was 172.1 ± 34.7 mg/dL, LDL 99.4 ± 23.1 mg/dL, HDL 41.2 ±

8.6 mg/dL, and TG 138.5 ± 37.9 mg/dL. In female hypothyroid patients (n = 33), TC rose significantly to 206.8 ± 43.3 mg/dL, LDL 129.4 ± 28.1 mg/dL, HDL 37.5 ± 7.4 mg/dL, and TG 193.6 ± 48.1 mg/dL ($p < 0.001$ for TC).

DISCUSSION

In our study of 300 CKD patients, thyroid dysfunction was highly prevalent, with hypothyroidism and low T3 syndrome being the most common abnormalities. We observed a progressive increase in thyroid dysfunction with advancing CKD stages, consistent with previous studies showing that reduced renal function impairs peripheral conversion of T4 to T3 and alters thyroid hormone metabolism [11,12]. Lo et al. reported a similar prevalence of low T3 in CKD patients, noting that its severity correlates with declining eGFR [13].Lipid profile analysis in our cohort revealed that hypothyroid patients had significantly higher total cholesterol, LDL, and triglyceride levels and lower HDL compared to euthyroid and hyperthyroid patients. These findings are in line with previous research indicating that hypothyroidism exacerbates dyslipidemia in CKD, thereby increasing cardiovascular risk [14,15]. Gupta et al. also demonstrated elevated total cholesterol (>200 mg/dL) in hypothyroid CKD patients, highlighting the additive atherogenic potential of thyroid dysfunction [16]. Gender-specific analysis indicated that female hypothyroid patients exhibited higher total cholesterol and LDL than males, supporting previous findings that females may be more susceptible to lipid disturbances in thyroid disease [17,18].Correlation analysis demonstrated that serum T3 and T4 negatively correlated with total cholesterol, LDL, and triglycerides and positively with HDL, whereas TSH was positively correlated with atherogenic lipids. These findings are supported by Al-Mohaissen et al., who found that thyroid hormone alterations are independently associated with dyslipidemia in CKD patients [19]. Furthermore, advancing CKD stage was associated with progressive worsening of lipid parameters, consistent with the study by Al-Shukaili et al., which reported that total cholesterol and LDL levels rise with declining renal function [20].Inflammatory markers, including hs-CRP, LDL/HDL ratio, and the Atherogenic Index of Plasma, were significantly elevated in hypothyroid patients, reflecting the pro-inflammatory and pro-atherogenic impact of thyroid dysfunction in CKD. Multivariable regression analysis identified elevated TSH, low T3, advanced CKD stage, older age, and diabetes as independent predictors of dyslipidemia, confirming the multifactorial nature of cardiovascular risk in this population. Overall, our findings highlight the importance of routine thyroid function screening in CKD patients, especially in advanced stages, as managing hypothyroidism may help mitigate dyslipidemia, systemic inflammation, and cardiovascular morbidity, consistent with the existing literature [11–20].

Conclusion

In patients with chronic kidney disease, thyroid dysfunction was common and increased with advancing stages of CKD. Hypothyroid patients exhibited more pronounced lipid abnormalities, including higher total cholesterol, LDL, and triglycerides, along with lower HDL levels, compared to euthyroid and hyperthyroid individuals. Serum T3 and T4 levels were inversely associated with atherogenic lipid parameters, whereas TSH showed a positive association. Inflammatory and atherogenic markers were elevated in hypothyroid patients, indicating a higher cardiovascular risk. Multivariable analysis demonstrated that thyroid dysfunction, CKD stage, age, and the presence of diabetes were significant predictors of adverse lipid and inflammatory profiles. Overall, thyroid abnormalities in CKD patients are strongly linked to dyslipidemia and increased atherogenic risk.

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