



CORRELATION OF THYROID FUNCTION AND LIPID PROFILE LEVELS IN PATIENTS WITH CHRONIC KIDNEY DISEASE: A COMPARATIVE CROSS-SECTIONAL STUDY

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ABSTRACT

Background: Chronic kidney disease (CKD) is a progressive systemic disorder characterized by multiple metabolic and endocrine disturbances. Thyroid dysfunction and dyslipidemia commonly coexist in CKD, potentially exacerbating cardiovascular risk. This study aimed to evaluate the correlation between thyroid function and lipid profile levels in patients with CKD compared to healthy controls.

Methods: This comparative cross-sectional study was conducted from March 2024 to August 2025 at a tertiary care hospital in Jaipur, India. A total of 240 participants aged 18–65 years were enrolled, including 120 diagnosed CKD patients and 120 age- and sex-matched healthy controls. Patients with a clinical history of pre-existing primary hypothyroidism, dyslipidaemia, diabetes mellitus were excluded. Serum levels of free T3 (FT3), free T4 (FT4), thyroid-stimulating hormone (TSH), total cholesterol (TC), triglycerides (TG), HDL, LDL, VLDL, serum creatinine, and estimated glomerular filtration rate (eGFR) were measured. Statistical analysis was performed using SPSS software, with independent t-tests and ANOVA as appropriate.

Results: Mean age of CKD patients was significantly higher than controls (52.89 ± 12.44 vs. 43.69 ± 11.74 years; $p < 0.001$). CKD patients showed significantly lower hemoglobin (10.30 ± 1.43 vs. 13.42 ± 1.12 g/dL), elevated serum urea (86.45 ± 44.56 vs. 27.63 ± 5.60 mg/dL) and creatinine (3.44 ± 1.94 vs. 0.90 ± 0.16 mg/dL), and reduced eGFR (23.07 ± 13.80 vs. 84.67 ± 22.27 mL/min/1.73m²) (all $p < 0.001$). Thyroid function analysis revealed significantly lower FT3 (2.25 ± 0.47 vs. 3.10 ± 0.46 pg/mL; $p < 0.001$) and higher TSH (3.28 ± 1.29 vs. 2.04 ± 0.75 μ IU/mL; $p < 0.001$) in CKD patients, with preserved FT4. Lipid profile showed significantly higher TC (220.68 ± 40.33 vs. 187.98 ± 31.06 mg/dL), TG (222.11 ± 77.46 vs. 122.22 ± 45.17 mg/dL), LDL (137.15 ± 45.90 vs. 114.68 ± 32.25 mg/dL), VLDL (44.42 ± 15.49 vs. 24.44 ± 9.04 mg/dL), and lower HDL (39.11 ± 7.45 vs. 48.85 ± 9.63 mg/dL) in CKD patients (all $p < 0.001$).

Conclusion: CKD is associated with significant thyroid dysfunction (low T3 syndrome and subclinical hypothyroidism) and an atherogenic lipid profile. Routine screening of thyroid function and lipid profile in CKD patients is recommended for early risk stratification and management to reduce cardiovascular morbidity.

KEYWORDS: Chronic Kidney Disease, Thyroid Dysfunction, Dyslipidemia, Low T3 Syndrome, eGFR, Cardiovascular Risk.

Introduction

Chronic kidney disease (CKD) represents a major global health challenge, affecting approximately 10–15% of the adult population worldwide. It is characterized by progressive and irreversible loss of kidney function, leading to end-stage renal disease (ESRD) if untreated. Beyond its renal manifestations, CKD is a systemic disorder associated with numerous metabolic and endocrine disturbances, including alterations in thyroid function and lipid metabolism.^{1,2}

The thyroid gland plays a fundamental role in regulating whole-body metabolism through secretion of thyroxine (T4) and triiodothyronine (T3). In CKD, the hypothalamus-pituitary-thyroid axis is frequently disrupted due to uremia, chronic inflammation, and altered peripheral metabolism of thyroid hormones. The most common pattern observed is “low T3 syndrome,” characterized by decreased serum T3 with normal or low T4 and TSH concentrations.³ Concurrently, CKD leads to characteristic dyslipidemia—elevated triglycerides, decreased HDL cholesterol, and accumulation of atherogenic lipoproteins—which significantly contributes to cardiovascular disease, the leading cause of mortality in CKD patients.⁴

The interplay between thyroid dysfunction and lipid abnormalities is biologically plausible: thyroid hormones regulate key enzymes in lipid synthesis and degradation, including HMG-CoA reductase, lipoprotein lipase, and LDL receptors. Hypothyroidism exacerbates the atherogenic lipid profile already present in CKD, potentially amplifying cardiovascular risk.⁵ According to the most recent KDIGO (Kidney Disease: Improving Global Outcomes) 2024 guidelines, CKD staging should incorporate both GFR and albuminuria categories to optimize cardiovascular and kidney risk prediction.⁶ Despite this, region-specific data, particularly from India, remain limited.

Therefore, this study was undertaken to evaluate the correlation between thyroid function and lipid profile levels in patients with CKD and to compare these parameters with healthy controls.

Materials and Methods

Study Design and Setting

This comparative cross-sectional study was conducted in the Department of General Medicine at the National Institute of Medical Sciences & Research, Jaipur, Rajasthan, India, from March 1, 2024, to August 31, 2025 (18 months).

Study Population

A total of 240 participants were enrolled: 120 patients with CKD and 120 age- and sex-matched healthy controls.

Inclusion Criteria (CKD group)

- Age 18–65 years, both genders
- Diagnosed CKD as per KDIGO guidelines: GFR <60 mL/min/1.73 m² for >3 months, with or without

structural kidney damage

- Willing to provide written informed consent

Inclusion Criteria (Control group)

- Healthy individuals aged 18–65 years
- No known chronic illness, normal renal function, and not on any medications affecting thyroid or lipid metabolism

Exclusion Criteria (Both groups)

- Known primary hypothyroidism or hyperthyroidism
- Known primary dyslipidemia
- Diagnosed diabetes mellitus
- Medications altering thyroid/lipid profile (amiodarone, phenytoin, beta-blockers, dopamine, statins, fibrates, steroids)
- Pregnancy, liver disease, malignancy, or acute infections

Sample Size Calculation

Sample size was calculated using the formula: $n = (Z\alpha/2^2 \times p \times (1-p)) / d^2$, assuming a prevalence of thyroid dysfunction in CKD of 13% (from prior literature), with 95% confidence interval and 6% absolute precision.

Substituting the values, $n = (1.96)^2 \times 0.13 \times (1-0.13) / (0.06)^2 = 120.69$

Minimum sample size was 121 per group; we enrolled 120 per group for feasibility. A purposive sampling technique was employed to recruit participants.

Data Collection and Laboratory Methods

A detailed clinical history, physical examination, and anthropometric measurements were recorded using a semi-structured proforma. Fasting (8–10 hours) venous blood samples were collected from all participants.

- **Renal Function Tests:** Serum urea (enzymatic method), serum creatinine (modified Jaffe’s kinetic method). eGFR was calculated using the CKD-EPI equation.
- **Thyroid Function Tests:** Free T3, free T4, and TSH measured by Electrochemiluminescence Immunoassay (ECLIA).
- **Lipid Profile:** Total cholesterol (cholesterol oxidase-peroxidase method), triglycerides (GPO-PAP method), HDL (direct immune inhibition), LDL and VLDL calculated using Friedewald’s formula (when TG <400 mg/dL).

All investigations were performed in the Central Laboratory of NIMS & Research, Jaipur, under standardized quality control.

Statistical Analysis

Data were entered into Microsoft Excel and analyzed using SPSS version 23.0 (IBM Corp., Armonk, NY, USA). Continuous variables were expressed as mean $\pm$ standard deviation (SD) and compared using independent Student’s t-test (two groups) or one-way ANOVA (≥ 3 groups).

Categorical variables were expressed as frequencies and percentages and compared using Chi-square test. A p-value <0.05 was considered statistically significant.

Ethical Approval

The study was approved by the Institutional Ethics Committee, NIMS University Rajasthan, Jaipur (Approval date: May 10, 2024) with Reference no: NIMSUR/IEC/2024/1004. Written informed consent was obtained from all participants in their preferred language (English/Hindi). The study was conducted in accordance with the Declaration of Helsinki and ICMR guidelines.

Results

Demographic and Clinical Characteristics

The CKD group had a significantly higher mean age compared to controls (52.89 ± 12.44 vs. 43.69 ± 11.74 years; $p < 0.001$). Sex distribution was comparable (CKD: 50% male; control: 54.2% male, $p = 0.51$). BMI, blood pressure, pulse rate, and smoking status did not differ significantly between groups (Table 1).

Renal Function and Haematological Parameters

CKD patients demonstrated significantly impaired renal function: markedly elevated serum urea (86.45 ± 44.56 vs. 27.63 ± 5.60 mg/dL; $p < 0.001$), serum creatinine (3.44 ± 1.94 vs. 0.90 ± 0.16 mg/dL; $p < 0.001$), and reduced eGFR (23.07 ± 13.80 vs. 84.67 ± 22.27 mL/min/1.73m²; $p < 0.001$). Hemoglobin was significantly lower in CKD patients (10.30

± 1.43 vs. 13.42 ± 1.12 g/dL; $p < 0.001$), consistent with anemia of CKD (Table 2).

Thyroid Function Abnormalities

CKD patients showed significantly lower FT3 (2.25 ± 0.47 vs. 3.10 ± 0.46 pg/mL; $p < 0.001$), higher TSH (3.28 ± 1.29 vs. 2.04 ± 0.75 μ IU/mL; $p < 0.001$), and slightly lower but non-significantly different FT4 (1.03 ± 0.15 vs. 1.08 ± 0.11 ng/dL; $p = 0.06$). This pattern is consistent with low T3 syndrome and subclinical hypothyroidism (Table 3, Figure 1).

Lipid Profile Abnormalities

CKD patients exhibited a characteristic atherogenic dyslipidemia: significantly higher total cholesterol (220.68 ± 40.33 vs. 187.98 ± 31.06 mg/dL; $p < 0.001$), triglycerides (222.11 ± 77.46 vs. 122.22 ± 45.17 mg/dL; $p < 0.001$), LDL (137.15 ± 45.90 vs. 114.68 ± 32.25 mg/dL; $p < 0.001$), VLDL (44.42 ± 15.49 vs. 24.44 ± 9.04 mg/dL; $p < 0.001$), and lower HDL (39.11 ± 7.45 vs. 48.85 ± 9.63 mg/dL) (Table 4).

CKD patients exhibited significant thyroid dysfunction characterized by lower Free T3 levels (2.25 ± 0.47 vs. 3.10 ± 0.46 pg/mL; $p < 0.001$) and higher TSH levels (3.28 ± 1.29 vs. 2.04 ± 0.75 μ IU/mL; $p < 0.001$) compared to controls, while regarding lipid profile, CKD patients demonstrated a significant atherogenic pattern with higher total cholesterol (220.68 ± 40.33 vs. 187.98 ± 31.06 mg/dL) and triglycerides (222.11 ± 77.46 vs. 122.22 ± 45.17 mg/dL), illustrated in Figure 1.

Table 1: Comparison of Demographic Characteristics

Parameter	CKD (n=120)	Control (n=120)	p-Value
Age (years)	52.89 ± 12.44	43.69 ± 11.74	< 0.001
Male sex, n (%)	60 (50.0)	65 (54.2)	0.51
BMI (kg/m ²)	25.48 ± 3.27	25.66 ± 3.87	0.69
SBP (mmHg)	133.80 ± 13.83	130.61 ± 13.05	0.07
DBP (mmHg)	81.28 ± 8.12	82.62 ± 8.46	0.21
Pulse (bpm)	77.77 ± 8.36	78.78 ± 7.74	0.33

Data presented as mean $\pm$ SD unless specified; independent t-test / Chi-square test.

P-value < 0.05 is statistically significant.

Table 2: Comparison of Renal Function and Haematological Parameters

Parameter	CKD (n=120)	Control (n=120)	p-Value
Hemoglobin (g/dL)	10.30 ± 1.43	13.42 ± 1.12	<0.001*
Serum Urea (mg/dL)	86.45 ± 44.56	27.63 ± 5.60	<0.001*
Serum Creatinine (mg/dL)	3.44 ± 1.94	0.90 ± 0.16	<0.001*
eGFR (mL/min/1.73m ²)	23.07 ± 13.80	84.67 ± 22.27	<0.001*

*Statistically significant

Table 3: Comparison of Thyroid Function

Parameter	CKD (n=120)	Control (n=120)	p-Value
Free T3 (pg/mL)	2.25 ± 0.47	3.10 ± 0.46	<0.001*
Free T4 (ng/dL)	1.03 ± 0.15	1.08 ± 0.11	0.06
TSH (μIU/mL)	3.28 ± 1.29	2.04 ± 0.75	<0.001*

*Statistically significant.

Table 4: Comparison of Lipid Profile

Parameter (mg/dL)	CKD (n=120)	Control (n=120)	p-value
Total Cholesterol	220.68 ± 40.33	187.98 ± 31.06	<0.001*
Triglycerides	222.11 ± 77.46	122.22 ± 45.17	<0.001*
HDL	39.11 ± 7.45	48.85 ± 9.63	<0.001*
LDL	137.15 ± 45.90	114.68 ± 32.25	<0.001*
VLDL	44.42 ± 15.49	24.44 ± 9.04	<0.001*

*Statistically significant

Correlation Summary

While detailed correlation matrices are beyond this concise report, the parallel presence of reduced FT3/elevated TSH with worsened lipid parameters (higher TC, TG, LDL, VLDL; lower HDL) suggests a clinically meaningful association between thyroid dysfunction and atherogenic dyslipidemia in CKD patients.

Discussion

This comparative cross-sectional study demonstrates that patients with chronic kidney disease have significant alterations in both thyroid function and lipid profile compared to healthy controls. The study findings align with established literature and provide important region-specific data from North India.⁷

The observed patterns of low FT3, normal-to-low FT4, and elevated TSH were consistent with low T3 syndrome and subclinical hypothyroidism. This likely results from impaired peripheral conversion of T4 to T3 due to reduced deiodinase activity, accumulation of uremic toxins, and chronic inflammation.^{8,9} Our findings mirror those of Layal et al., who reported significantly lower FT3 and higher TSH in CKD patients, with prevalence of hypothyroidism increasing with CKD stage.⁷ Similarly, Khatiwada et al. documented progressive decline in FT3 with worsening renal function.³

The atherogenic lipid profile (elevated TC, TG, LDL, VLDL; low HDL) observed in our CKD patients is a hallmark of CKD-associated dyslipidemia. The mechanisms include reduced lipoprotein lipase activity, impaired hepatic clearance of triglyceride-rich lipoproteins, altered

apolipoprotein metabolism, and increased oxidative modification of lipoproteins.^{10,11} The study results are consistent with Vinayak et al. and Singh et al., who reported similar lipid derangements in Indian CKD cohorts.^{12,13}

The coexistence of thyroid dysfunction and dyslipidemia is particularly concerning because both independently contribute to cardiovascular mortality in CKD. Thyroid hormones regulate LDL receptor expression, cholesterol 7α-hydroxylase activity, and lipolysis.¹⁴ In CKD, the uremic milieu may amplify the lipid-altering effects of thyroid hormone deficiency, leading to a synergistic increase in cardiovascular risk.¹⁵ This study supports the hypothesis that screening for and managing thyroid dysfunction might improve lipid profiles and reduce cardiovascular events in CKD patients, though interventional studies are needed.

The present study findings are broadly consistent with the master article by Layal et al., which also reported low FT3, elevated TSH, hypertriglyceridemia, and low HDL in CKD patients, with worsening abnormalities across CKD stages 3–5.⁷ However, this study included a large control group (n=120) and used strict exclusion criteria to avoid confounding from primary thyroid or lipid disorders.

However, the study has certain limitations. Being a single-center study, the findings may have limited generalizability to broader populations. In addition, CKD patients were not stratified according to disease stages (Stages 1–5), which could have provided a clearer understanding of the progression of thyroid and lipid abnormalities with worsening renal function. The study also did not assess inflammatory biomarkers such as interleukin-6 (IL-6),

tumor necrosis factor-alpha (TNF- α), and C-reactive protein (CRP), which are known to play significant roles in both thyroid dysfunction and dyslipidemia among CKD patients. Furthermore, the study lacked longitudinal follow-up and interventional assessment, and therefore could not evaluate whether thyroid hormone replacement therapy has any beneficial effect on lipid parameters or cardiovascular outcomes in this patient population.

Future Directions

Future research should focus on longitudinal cohort studies to determine whether thyroid dysfunction precedes or develops secondary to lipid abnormalities during CKD progression. Additionally, randomized controlled trials are needed to evaluate the therapeutic impact of thyroid hormone replacement, such as low-dose levothyroxine, on lipid profile, endothelial dysfunction, and cardiovascular outcomes in CKD patients with subclinical hypothyroidism or low T3 syndrome. Further mechanistic studies should also investigate the contribution of inflammatory pathways, uremic toxins, and genetic polymorphisms, including deiodinase gene variants, in regulating the complex thyroid–lipid–renal axis and its role in CKD-associated metabolic disturbances.

Conclusion

This study confirms that chronic kidney disease is associated with significant thyroid dysfunction (predominantly low T3 syndrome and subclinical hypothyroidism) and an atherogenic lipid profile (hypertriglyceridemia, elevated LDL/VLDL, low HDL). These abnormalities likely contribute to the high cardiovascular morbidity and mortality observed in CKD patients.

Clinical Takeaway

Routine assessment of thyroid function (FT3, FT4, TSH) and lipid profile should be integrated into the standard care of all CKD patients, even in early stages, to enable timely risk stratification and intervention. Early detection and management of these modifiable risk factors may improve cardiovascular outcomes and slow renal disease progression.

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Conflict of Interest

The authors do not have any conflicts of interest.

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