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THYROID AND LIPID PROFILE ABNORMALITIES IN CHRONIC KIDNEY DISEASE: A CROSS-SECTIONAL STUDY FROM A TERTIARY CARE CENTRE IN NORTH INDIA

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ABSTRACT **Background:** Chronic kidney disease (CKD) is associated with multiple metabolic derangements including thyroid dysfunction and dyslipidemia, both of which significantly increase cardiovascular risk. However, data from the Indian subcontinent remain limited. **Objectives:** To assess thyroid and lipid profiles in CKD patients across disease stages and to examine their correlation with estimated glomerular filtration rate (eGFR). **Methods:** A hospital-based cross-sectional study was conducted at SMS Hospital, Jaipur, enrolling 50 adult CKD patients (Stages 3–5) from August 2023 to December 2024. Serum TSH, T3, T4, fasting lipid profile, eGFR, parathyroid hormone (PTH), calcium, and phosphorus were measured. Patients on dialysis, with thyroid or lipid-altering medications, autoimmune disorders, or acute kidney injury were excluded. Statistical analysis employed Student's t-test, Chi-square, Pearson/Spearman correlation, and ANOVA. **Results:** The mean age was 52.4 ± 12.3 years; 64% were male. CKD Stage 3 accounted for 42%, Stage 4 for 38%, and Stage 5 for 20%. Thyroid abnormalities were prevalent: elevated TSH (46%), low T3 (54%), and low T4 (18%). TSH rose from 4.5 ± 1.8 mIU/L (Stage 3) to 8.9 ± 3.2 mIU/L (Stage 5; $p<0.001$), while T3 and T4 declined significantly. Lipid abnormalities included hypertriglyceridemia (62%), low HDL (54%), elevated total cholesterol (48%), and elevated LDL (38%). eGFR correlated negatively with TSH ($r=-0.65$), triglycerides ($r=-0.52$), and PTH ($r=-0.72$), and positively with T3 ($r=0.58$) and HDL ($r=0.45$) (all $p<0.05$). Diabetic CKD patients had significantly worse lipid and thyroid profiles. **Conclusion:** Thyroid dysfunction and dyslipidemia worsen progressively with CKD severity and correlate significantly with declining eGFR. Routine metabolic screening in CKD patients, especially those with diabetes, is strongly recommended.

KEYWORDS : Chronic kidney disease; thyroid dysfunction; dyslipidemia; eGFR; subclinical hypothyroidism; cardiovascular risk

INTRODUCTION

Chronic kidney disease (CKD) is a global public health problem, affecting 8–16% of the population worldwide and disproportionately burdening low- and middle-income countries.[1] Defined by a glomerular filtration rate (GFR) below 60 mL/min/1.73 m^2 or markers of kidney damage persisting for more than three months, CKD carries a substantially elevated burden of cardiovascular morbidity and mortality—10 to 30 times higher than in the general population.[2]

Among the systemic complications of CKD, thyroid dysfunction and dyslipidemia are particularly significant yet frequently under-recognised comorbidities. The kidney participates actively in the metabolism, peripheral conversion, and elimination of thyroid hormones. Progressive reduction in GFR impairs iodine clearance, disrupts the hypothalamic-pituitary-thyroid axis, and inhibits the 5'-deiodinase enzyme, collectively resulting in reduced T3 synthesis, low circulating T3, and variable changes in T4 and TSH.[3-5] Low triiodothyronine (T3) syndrome is the most common thyroid abnormality reported in CKD and is independently associated with increased all-cause mortality.

Dyslipidemia in CKD is characterised by hypertrigly ceridaemia, reduced high-density lipoprotein cholesterol (HDL-C), and elevated small dense low-density lipoprotein (sdLDL), primarily attributable to impaired lipoprotein lipase (LPL) activity, decreased lecithin-cholesterol acyltransferase (LCAT) activity, and elevated apolipoprotein C-III (apoC-III) levels. Both thyroid dysfunction and dyslipidemia amplify the already heightened cardiovascular risk in CKD.

Despite a growing body of international literature, Indian data—especially from northern India—on the concurrent assessment of thyroid and lipid profiles across CKD stages remain scarce. Timely identification of these abnormalities could guide early therapeutic interventions and potentially slow CKD progression. The present study was therefore undertaken to systematically evaluate thyroid and lipid profiles across CKD stages 3 to 5, and to assess their correlation with eGFR, at a tertiary referral centre in Rajasthan, India.

MATERIALS AND METHODS

Study Design and Setting

This was a hospital-based, observational, cross-sectional study conducted in the Department of General Medicine, Sawai Man Singh (SMS) Hospital, Jaipur, Rajasthan, India, a government-run tertiary care centre. The study was conducted from August 2023 to December 2024.

Sample Size and Sampling

Sample size was computed at 95% confidence interval (= 0.05) based on an expected mean TSH of 16.70 ng/L in CKD Stage 3 patients with a standard deviation of 9.10 and an allowable error of 3%, yielding a minimum of 36 patients. To enhance statistical power and account for attrition, 50 consecutive eligible patients were enrolled using a purposive first-come, first-served approach.

Inclusion and Exclusion Criteria

Patients aged ≥18 years with a confirmed diagnosis of CKD (Stages 3–5) based on KDIGO 2012 guidelines were included. Patients were excluded if they had: haematological or autoimmune disorders; renal malignancy or ongoing chemotherapy; pregnancy; current use of nephrotoxic drugs, lipid-lowering agents, or thyroid hormone–altering medications; history of acute kidney injury or renal transplantation; or if they were on maintenance haemodialysis.

Clinical Assessment and Laboratory Investigations

Detailed clinical history, anthropometric measurements, and examination were performed. CKD stage was determined

using the CKD-EPI 2009 creatinine equation to estimate GFR. The following investigations were performed: serum TSH, free/total T3 and T4, fasting lipid profile (total cholesterol, LDL-C, HDL-C, triglycerides, VLDL-C), intact PTH, serum calcium, phosphorus, renal function tests (urea, creatinine), complete blood count, and abdominal ultrasonography.

Ethical Considerations

Written informed consent was obtained from all participants prior to enrolment. The study protocol received ethical approval from the Institutional Ethics Committee of SMS Medical College, Jaipur. Patient anonymity and confidentiality were maintained throughout.

Statistical Analysis

Data were entered in Microsoft Excel and analysed using SPSS version 21.0 (IBM Corp., Armonk, NY). Continuous variables are reported as mean ± standard deviation and categorical variables as frequencies and percentages. Inter-group comparisons used Student's independent t-test (two groups) or one-way ANOVA/Kruskal-Wallis test (three or more groups). Categorical associations were assessed using the Chi-square test. Pearson's or Spearman's correlation coefficient was used to assess associations between continuous variables. A two-tailed p-value <0.05 was considered statistically significant.

RESULTS

Demographic and Clinical Characteristics

Fifty patients with CKD were enrolled. The mean age was 52.4 ± 12.3 years; the 61–70 year age group was the most represented (28%). Males predominated (64%). Mean BMI was 22.8 ± 3.5 kg/m². Hypertension was present in 70% and diabetes mellitus in 40% of patients. Tobacco and alcohol use were noted in 12% and 16%, respectively, with combined use in 24%.

Table 1. Clinical Characteristics of the Study Population (n=50)

Characteristic	Value
Age (years, mean ± SD)	52.4 ± 12.3
Gender: Male / Female	32 (64%) / 18 (36%)
BMI (kg/m², mean ± SD)	22.8 ± 3.5
Diabetes mellitus	20 (40%)
Hypertension	35 (70%)
Tobacco use	6 (12%)
Alcohol use	8 (16%)
Both tobacco and alcohol	12 (24%)

CKD Staging and Aetiology

CKD Stage 3 was most common (42%), followed by Stage 4 (38%) and Stage 5 (20%). Mean eGFR declined significantly across stages: Stage 3: 46.2 ± 8.5, Stage 4: 22.6 ± 4.3, Stage 5: 10.8 ± 2.7 mL/min/1.73 m² ($p=0.001$). Diabetic nephropathy was the leading aetiology (36%), followed by hypertensive nephropathy (30%), chronic glomerulonephritis (20%), and others/unknown (14%).

Thyroid Profile Across CKD Stages

TSH increased significantly from Stage 3 to Stage 5 (4.5 ± 1.8 → 8.9 ± 3.2 mIU/L; $p<0.001$). T3 declined from 85 ± 15 ng/dL in Stage 3 to 55 ± 10 ng/dL in Stage 5 ($p<0.001$). T4 also declined significantly (6.8 ± 1.2 → 5.5 ± 0.9 μg/dL; $p=0.012$). Overall prevalence of thyroid abnormalities: elevated TSH 46%, low T3 54%, low T4 18%.

Table 2. Thyroid Profile Across CKD Stages

Parameter	Stage 3 (n=21)	Stage 4 (n=19)	Stage 5 (n=10)	p-value
TSH (mIU/L)	4.5 ± 1.8	6.2 ± 2.5	8.9 ± 3.2	<0.001
T3 (ng/dL)	85 ± 15	70 ± 12	55 ± 10	<0.001
T4 (μg/dL)	6.8 ± 1.2	6.0 ± 1.0	5.5 ± 0.9	0.012

Lipid Profile Across CKD Stages

Total cholesterol increased from 190 ± 35 mg/dL (Stage 3) to 221 ± 45 mg/dL (Stage 5; $p=0.045$). HDL-C fell significantly (45 ± 10 32 ± 7 mg/dL; $p<0.001$). Triglycerides rose from 150 ± 40 to 218 ± 60 mg/dL ($p=0.003$). LDL showed an upward trend that did not reach statistical significance ($p=0.089$). Prevalence: high triglycerides 62%, low HDL 54%, high total cholesterol 48%, high LDL 38%.

Table 3. Lipid Profile Across CKD Stages

Parameter	Stage 3 (n=21)	Stage 4 (n=19)	Stage 5 (n=10)	p-value
Total Cholesterol (mg/dL)	190 ± 35	205 ± 40	221 ± 45	0.045
LDL-C (mg/dL)	115 ± 25	125 ± 30	135 ± 35	0.089
HDL-C (mg/dL)	45 ± 10	38 ± 8	32 ± 7	<0.001
Triglycerides (mg/dL)	150 ± 40	180 ± 50	218 ± 60	0.003

Parathyroid and Mineral Parameters

PTH rose progressively (78 ± 20 188 ± 50 pg/mL; $p<0.001$). Serum calcium declined significantly (9.1 ± 0.5 8.3 ± 0.7 mg/dL; $p=0.015$), and phosphorus increased (3.8 ± 0.6 5.2 ± 1.0 mg/dL; $p<0.001$). PTH was elevated (>65 pg/mL) in 72%, low calcium in 28%, and high phosphorus in 42% of patients.

Correlations with eGFR

eGFR correlated negatively with TSH ($r=-0.65$; $p<0.001$), triglycerides ($r=-0.52$; $p<0.001$), total cholesterol ($r=-0.28$; $p=0.048$), PTH ($r=-0.72$; $p<0.001$), and phosphorus ($r=-0.60$; $p<0.001$). Positive correlations were found with T3 ($r=0.58$; $p<0.001$), T4 ($r=0.32$; $p=0.024$), HDL-C ($r=0.45$; $p=0.001$), and calcium ($r=0.38$; $p=0.006$). LDL-C did not significantly correlate with eGFR ($r=-0.22$; $p=0.124$).

Table 4. Correlation of Key Parameters with eGFR

Parameter	Correlation (r)	p-value	Direction
TSH	−0.65	<0.001	Negative
T3	+0.58	<0.001	Positive
T4	+0.32	0.024	Positive
Total Cholesterol	−0.28	0.048	Negative
HDL-C	+0.45	0.001	Positive
Triglycerides	−0.52	<0.001	Negative
PTH	−0.72	<0.001	Negative
Calcium	+0.38	0.006	Positive
Phosphorus	−0.60	<0.001	Negative

Effect of Diabetes and Gender

Diabetic patients had significantly higher TSH (6.5 ± 2.8 vs. 4.7 ± 2.5 mIU/L; $p=0.02$), total cholesterol (218 ± 40 vs. 168 ± 38 mg/dL; $p=0.03$), LDL-C (130 ± 30 vs. 88 ± 28 mg/dL; $p=0.014$), and triglycerides (190 ± 55 vs. 144 ± 50 mg/dL; $p=0.016$), with significantly lower HDL-C (36 ± 8 vs. 44 ± 9 mg/dL; $p=0.02$) compared with non-diabetics. No statistically significant gender differences were observed in thyroid or lipid parameters (all $p>0.05$).

DISCUSSION

The present study demonstrates a high burden of thyroid and lipid abnormalities in CKD patients, with a clear stepwise worsening as disease severity advances from Stage 3 to Stage 5. These findings align with and extend previous literature from Indian and international cohorts.

The demographic profile—mean age 52.4 years with male predominance and high prevalence of hypertension (70%) and diabetes (40%)—is consistent with reports by Mondal et al. (mean age 50.5 years, 67% males) and Al Hussaini et al. (mean age 51.3 years, 76.8% males).[7,18] Diabetic nephropathy as the leading aetiology (36%) mirrors the finding of Samir Singh et al. (44.66%).9

The progressive increase in TSH and decline in T3 and T4 with advancing CKD stages confirms the impairment of thyroid

hormone metabolism in uremia. The negative correlation of TSH with eGFR (r = −0.65) and positive correlation of T3 with eGFR (r = 0.58) are consistent with findings by Peters et al. and Raj et al., who identified eGFR as an independent predictor of fT3 levels.[10,11] The pathophysiological basis includes accumulation of iodine (Wolff-Chaikoff effect), metabolic acidosis impairing iodothyronine deiodinase, and cytokine-mediated (TNF-α, IL-1) inhibition of 5'-deiodinase activity.[12]

Low T3 syndrome, found in 54% of our patients, is of particular clinical significance given emerging evidence linking low fT3 to increased all-cause and cardiovascular mortality in CKD. The 46% prevalence of elevated TSH, predominantly subclinical, is higher than the 25% reported by Sinjari et al. (2017) but comparable to the 32.7% in the more contemporary Sinjari et al. (2022) report.[13] This likely reflects differences in CKD stage distribution and the exclusion of dialysis patients in the present study.

Lipid abnormalities were ubiquitous, with hypertriglyceridaemia (62%) and low HDL-C (54%) being most prevalent—an atherogenic pattern consistent with the impairment of lipoprotein lipase and LCAT activity described in uremia.[6] Mondal et al. reported even higher rates of hypertriglyceridaemia (89%) and low HDL (87.5%) in a predominantly Stage 4–5 cohort.[7] The strong negative correlation between eGFR and triglycerides (r = −0.52) and positive correlation with HDL-C (r = 0.45) support the mechanistic link between progressive renal dysfunction and lipid dysregulation. The absence of a statistically significant LDL-C increase across stages, despite an upward trend, likely reflects the complex remodelling of LDL particles (increased sdLDL fraction) rather than absolute LDL elevation, a finding also noted by Lecamwasam et al.[14]

Diabetic CKD patients exhibited a more adverse cardiometabolic profile, with significantly elevated LDL-C and triglycerides and lower HDL-C. This dual burden underscores the synergistic effect of diabetes and CKD on lipid metabolism and reinforces the need for intensified metabolic monitoring in this subgroup. The absence of significant gender differences in either thyroid or lipid profiles contrasts with some reports but is consistent with the small sample size of the present study and warrants confirmation in larger cohorts.

Parathyroid parameters confirmed the expected pattern of secondary hyperparathyroidism, with a robust inverse correlation between eGFR and PTH ($r = -0.72$; $p < 0.001$), serving as an internal validity check and underscoring the interplay between mineral-bone disorder and metabolic comorbidities in CKD.

LIMITATIONS

This study has several limitations. The cross-sectional design precludes causal inference. The sample size (n = 50), though adequately powered for primary outcomes, limits subgroup analyses. Dialysis patients were excluded, restricting generalisability to the predialysis CKD population. Free T3 and free T4 were not measured separately; total hormones were used. Residual confounding from dietary habits, unmeasured comorbidities, and medication use cannot be entirely excluded.

CONCLUSION

Thyroid dysfunction—predominantly low T3 and subclinical hypothyroidism—and dyslipidaemia characterised by hypertriglyceridaemia and low HDL-C are highly prevalent in pre-dialysis CKD patients and worsen progressively with declining renal function. eGFR correlates significantly with TSH, T3, T4, triglycerides, and HDL-C, establishing a clear metabolic-renal axis. Diabetic CKD patients carry a disproportionately higher cardiometabolic burden. These findings support routine screening of thyroid and lipid profiles in all CKD patients—particularly those with advanced disease or coexisting diabetes—to enable early detection and intervention, thereby potentially reducing cardiovascular morbidity and slowing CKD progression.

DECLARATIONS

Ethics Approval: Approved by the Institutional Ethics Committee, SMS Medical College, Jaipur. Written informed consent was obtained from all participants.

Conflict of Interest: The authors declare no conflicts of interest.

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Data Availability: Data supporting the findings of this study are available from the corresponding author upon reasonable request.

Author Contributions: H.B.: study design, data collection, analysis, manuscript writing. P.M.: supervision, manuscript review and revision. Both authors approved the final manuscript.

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