Original Article

Study of Thyroid and Lipid Profile in Patients with Chronic Kidney Disease

Prateet Kaur Sidhu[1], RK Sharma[2], Manish Chandey[2], Mehak Kaur[1], Khushveen Sidhu[1]

[1]Senior Resident, Department of General Medicine, Sri Guru Ram Das Institute of Medical Sciences and Research, Vallah, Sri Amritsar, Punjab, India.
[2]Professor, Department of General Medicine, Sri Guru Ram Das Institute of Medical Sciences and Research, Vallah, Sri Amritsar, Punjab, India.

Abstract

Background: The Kidney Disease: Improving Global Outcomes (KDIGO) defines chronic kidney disease as kidney injury or a reduction in glomerular filtration rate (GFR) to <60ml/min/1.73 m^2 for at least 3 months. Thyroid hormone dysfunction and dyslipidemia are two parameters frequently observed in patients with CKD. It has been demonstrated that in individuals with CKD, dyslipidemia and thyroid hormone abnormalities play a significant role in the disease's early development and higher risk of cardiovascular problems. **Material and Methods:** The study was conducted in the Department of Medicine, Shri Guru Ram Das Institute of Medical Sciences and Research, Vallah, Sri Amritsar, in collaboration with Sri Guru Ram Das University of Health Sciences, Sri Amritsar. Patients diagnosed with chronic kidney disease (CKD) (newly diagnosed or previously known) presenting to the OPD/IPD between 01-08-2023 and 28-02-2025. **Results:** The majority (45.5%) are over 60 years old, followed by the 51-60 age group (28.2%). Males represent a slightly higher proportion (51.8%, n=57) compared to females (48.2%, n=53). The vast majority (78.4%) have CKD stage G5 (kidney failure), followed by stage G4 (18.2%), indicating severe kidney dysfunction in most cases. Early-stage CKD (G2, G3a, G3b) is rare, collectively representing only 5.5% of patients. The vast majority (84.6%, n=93) fall under A3, indicating severe albuminuria, which is consistent with advanced CKD (as 78.4% had stage G5). Only 15.4% (n=17) are in the A2 range. It was found that there was a substantial difference in mean TSH levels between patients and controls. While mean T4 levels were non-significant, mean T3 levels between patients and controls were shown to be significant. The comparison of means, L, HDL, and TGs was found to be substantial between cases and controls, whereas the comparison of mean cholesterol was found to be non-significant. The correlation between eGFR and TSH levels was statistically significant. The correlation between eGFR and TGs was found to be statistically significant. **Conclusion:** Early thyroid and lipid profile measurements in CKD patients help slow the progression of the illness and lower the risk of cardiovascular disease in these individuals. It facilitates prompt intervention and the implementation of appropriate preventative measures.

Keywords: CKD, LDL, HDL, TGs, TSH, T3, T4, eGFR.

Received: 01 November 2025 **Revised: 25 November 2025** **Accepted: 01 December 2025** **Published: 13 December 2025**

Introduction

A persistent and irreversible disorder, chronic kidney disease (CKD) is characterised by a progressive loss of kidney function over a period of three months or more. It involves structural or functional kidney impairments, with or without reduced glomerular filtration rate (GFR), or a sustained GFR below 60 mL/min/1.73 m^2 for at least three months, regardless of other kidney damage markers.[1] CKD arises from underlying conditions such as diabetes, hypertension, glomerulonephritis, polycystic kidney disease, and autoimmune disorders.[2] The disease is categorised into five stages based on GFR, with stages 4 and 5 indicating advanced disease requiring specialised care, including dialysis or transplantation. Staging helps identify high-risk patients, allowing targeted interventions to delay end-stage renal disease.[3]

Globally, CKD affects over 700 million people, representing approximately 9.1% of the population, with prevalence steadily rising.[4] It is currently the 12th leading cause of death worldwide and is projected to become the 5th by 2040.[5] In India, CKD is a growing epidemic, with prevalence rates ranging from 7.6% to 17.2%.[6]

CKD profoundly affects systemic health, leading to complications such as cardiovascular disease (CVD), anaemia, bone disorders, and metabolic imbalances. Thyroid dysfunction is a major endocrine complication, affecting 20%–40% of CKD patients.[7]

The thyroid regulates metabolism, protein synthesis, and energy production, but CKD disrupts thyroid hormone metabolism due to uraemia, iodine retention, and impaired conversion of thyroxine (T4) to triiodothyronine (T3).[8] Subclinical and overt hypothyroidism are most common, along with non-thyroidal

Address for correspondence: Dr. Prateet Kaur Sidhu, Senior Resident, Department of General Medicine, Sri Guru Ram Das Institute of Medical Sciences and Research, Vallah, Sri Amritsar, Punjab, India.
E-mail: prateetkaur97@gmail.com

DOI:
10.21276/amit.2025.v12.i3.240

How to cite this article: Sidhu PK, Sharma RK, Chandey M, Kaur M, Sidhu K. Study of Thyroid and Lipid Profile in Patients with Chronic Kidney Disease. Acta Med Int. 2025;12(3):1122-1126.

illness syndrome, characterised by low T3 levels.[9] Thyroid dysfunction in CKD is associated with faster disease progression, higher cardiovascular risk, and increased mortality.[10]

Another critical metabolic abnormality in CKD is dyslipidemia, which contributes significantly to the elevated cardiovascular mortality in this population. Largely atherogenic tiny dense low-density lipoprotein cholesterol (LDL-C) particles, reduced HDL-C, and increased triglycerides are the hallmarks of dyslipidemia in chronic kidney disease (CKD).[11]

These lipid abnormalities result from reduced activity of lipoprotein lipase, hepatic triglyceride lipase, and increased hepatic production of very-low-density lipoproteins (VLDLs). The prevalence of dyslipidemia increases with CKD progression, further compounding cardiovascular risks.[12]

Materials and Methods

The study was conducted in the Department of Medicine, Shri Guru Ram Das Institute of Medical Sciences and Research, Vallah, Sri Amritsar, in collaboration with Sri Guru Ram Das University of Health Sciences, Sri Amritsar.Patients diagnosed with chronic kidney disease (CKD) (newly diagnosed or previously known) presenting to the OPD/IPD between 01-08-2023 and 28-02-2025.

Inclusion criteria:

1. Age >18 years
2. All patients diagnosed with CKD

Exclusion criteria:

1. Acute kidney injury (AKI)
2. History of renal transplantation
3. Hematological disorders
4. Renal malignancy or ongoing chemotherapy
5. Pregnancy
6. Pre-existing thyroid disorders (hyper-/hypothyroidism)
7. Use of nephrotoxic drugs, lipid-lowering agents, or thyroid-altering medications

Controls:

- Age- and gender-matched healthy individuals (OPD visitors/admitted for unrelated illnesses).
- Underwent the same investigations as CKD patients.

Ethical Considerations

- Approved by the Institutional Ethics Committee.
- Written informed consent obtained in the vernacular language.

Statistical Analysis

Data analyzed using appropriate statistical tools (SPSS version 22). Descriptive statistics: Mean ± SD, frequencies. Chi-square/Fisher's test, t-test/ANOVA, Pearson/Spearman correlation (thyroid-lipid-CKD associations). $p < 0.05$ considered statistically significant.

Results

Following informed agreement, a total of 220 subjects—110 CKD patients and 110 healthy controls—were included. Patients with CKD are 56 years old on average.

48.2% of the recruited individuals were female, and 51.2% were male, according to an analysis of the gender split among the research participants.

The study's classification of CKD patients by management method, including or excluding hemodialysis. Of the 110 CKD patients included, 49 were receiving medical treatment, and 61 were receiving maintenance hemodialysis.

A greater percentage of patients with chronic kidney disease (CKD) had elevated urea and creatinine readings compared to the control group, according to a comparison of the means of the two research groups.

The mean values of T3, thyroxine (T4), and TSH in the CKD patients were 2.3 ng/dL, 1.4 ng/dL, and 6.7 mIU/L, respectively, when the thyroid profiles of the study groups were compared.

Dyslipidemia was more common in CKD patients than in the control group, according to a comparison of the lipid profiles of the two research groups.

Table 1: Distribution according to age

Age (in years)	Number of patients	Percent
18-20	3	2.7
21-30	5	4.5
31-40	11	10.0
41-50	10	9.1
51-60	31	28.2
>60	50	45.5
TOTAL	110	100.0
Mean±SD	56.7182±15.35401	

The majority (45.5%) are over 60 years old, followed by the 51-60 age group (28.2%). The mean age was 56.72 ± 15.35 years, indicating a relatively older population with moderate age variability. Younger age groups (18-20, 21-30) represent smaller proportions (2.7% and 4.5%, respectively). Males represent a slightly higher proportion (51.8%, n=57) compared to females (48.2%, n=53).

Table 2: Distribution according to gender

Gender	Number of patients	Percent
Female	53	48.2
Male	57	51.8
Total	110	100.0

Table 3: Distribution according to hemodialysis

Mode of Management	Number of patients	Percent
Hemodialysis	49	44.5
Medical management	61	55.5

The table presents the hemodialysis status of the 110 CKD patients, revealing that a slight majority (55.5%, n=61) are undergoing hemodialysis, while 44.5% (n=49) are not. This aligns with the earlier finding that 78.4% had stage G5 CKD, suggesting that over half of the advanced-stage patients are receiving dialysis. At the same time, some may be managed conservatively or are yet to initiate therapy.

Table 4: Mean distribution of Creatinine and BUN in cases and controls

Parameters	Cases		Controls		p-value
	Mean	Std. Deviation	Mean	Std. Deviation	
EGFR	11.8182	9.77000	101.9818	65.54514	0.001 (Sig.)
UACR	1247.7218	955.81775	222.6252	155.31187	0.001 (Sig.)

The analysis of renal function markers revealed highly significant differences (p=0.001) between CKD cases and controls for both estimated glomerular filtration rate (eGFR) and urinary albumin-to-creatinine ratio (UACR). CKD patients showed severely reduced eGFR (11.82 ± 9.77 mL/min/1.73m²) compared to controls (101.98 ± 65.55), confirming advanced kidney impairment. Similarly, UACR was markedly elevated in cases (1247.72 ± 955.82 mg/g) compared with controls (222.63 ± 155.31 mg/g), reflecting significant proteinuria.

Table 5: Mean distribution of Thyroid profile in cases and controls

	Groups	N	Mean	Std. Deviation	p-value
T3	CASE	110	2.3823	.66086	0.001*
	CONTROL	110	2.7036	.80319	
T4	CASE	110	1.4295	.63216	0.826
	CONTROL	110	1.4066	.88602	
TSH	CASE	110	6.7450	3.68156	0.040*
	CONTROL	110	4.4358	11.38855	

Analysis of thyroid function tests revealed significant disturbances in CKD patients compared with controls. T3 levels were significantly lower in cases (2.38 ± 0.66) than controls (2.70 ± 0.80, p=0.001), consistent with impaired peripheral conversion in CKD. While T4 levels showed no difference (cases: 1.43 ± 0.63 vs controls: 1.41 ± 0.89, p=0.826), TSH was significantly higher in cases (6.75 ± 3.68) than controls (4.44 ± 11.39, p=0.040), suggesting compensatory pituitary stimulation.

Table 6: Distribution of Mean levels of Lipid profile in cases and controls

Parameters	Cases		Controls		p-value
	Mean	Std. Deviation	Mean	Std. Deviation	
LDL	110.7845	48.94673	79.2524	65.03723	0.001 (Sig.)
HDL	38.3005	21.37674	46.5491	26.52859	0.001 (Sig.)
Cholesterol	173.5118	65.87131	177.8118	60.17279	0.614 (NS)
Triglyceride	353.8864	135.45022	234.0345	190.30935	0.049 (Sig)

LDL was significantly higher in Cases (110.78 ± 48.95) compared to Controls (79.25 ± 65.04) (p = 0.001). HDL was significantly lower in Cases (38.30 ± 21.38) than in Controls (46.55 ± 26.53) (p = 0.001). Total Cholesterol showed no significant difference between groups (Cases: 173.51 ± 65.87; Controls: 177.81 ± 60.17, p = 0.614). Triglycerides were significantly elevated in Cases (353.89 ± 135.45) compared to Controls (234.03 ± 190.31) (p = 0.049).

Discussion

The interplay between thyroid dysfunction and dyslipidemia is particularly significant in CKD. Thyroid hormones regulate lipid metabolism, and hypothyroidism impairs LDL receptor function, raising LDL-C levels and reducing cholesterol clearance.[13] Dyslipidemia exacerbates inflammation and oxidative stress, further impairing thyroid function and creating a harmful cycle that worsens cardiovascular and renal complications.[14]

The majority of participants were aged >60 years (45.5%), with a mean age of 56.72±15.35 years, indicating an older cohort. This aligns with prior research showing that CKD prevalence increases with advancing age, particularly among those over 60 years (Hill et al., 2016; United States Renal Data System [USRDS], 2022).[15,16] Males slightly outnumbered females (51.8% vs 48.2%), consistent with previous findings that men are at a somewhat higher risk for CKD progression compared to women (Carrero et al., 2018).[17] Most participants hailed from rural areas (60.0%), suggesting socioeconomic and healthcare access differences that may affect CKD progression. Prior studies have highlighted that individuals in rural settings often face barriers to early CKD diagnosis and treatment, contributing to worse outcomes (Vassalotti et al., 2019).[18]

BMI data revealed that while 58.1% fell within the normal to overweight range, 34.5% were obese, highlighting the known association between obesity and kidney disease. This observation is supported by evidence that obesity is an independent risk factor for the development and progression of CKD (Garofalo et al., 2017).[19] CKD tagging revealed an alarmingly high proportion (78.4%) of patients with stage G5 (kidney failure), and only 5.5% were in early stages (G2–G3b). This distribution mirrors trends reported in tertiary care settings, where late-stage presentation is common due to delayed diagnosis and limited access to nephrology services (Saran et al., 2020, and Bello et al., 2019).[20,21] Haemoglobin was significantly lower in cases, indicating anaemia of CKD, a common complication attributed to reduced erythropoietin production and chronic inflammation (Stauffer& Fan, 2014)22, while platelet counts showed no significant difference, aligning with reports that thrombocytopenia is less consistently associated with early to moderate stages of CKD (Pecoits-Filho et al., 2010).[23]

Thyroid abnormalities were significant. T3 levels were lower in cases (p=0.001), and TSH was elevated (p=0.040), though T4 levels were comparable between groups. These findings are consistent with previous studies demonstrating that non-thyroidal illness syndrome, particularly "low T3 syndrome," is highly prevalent in CKD patients due to impaired peripheral conversion of T4 to T3 and altered hypothalamic-pituitary axis regulation (Kaptein et al., 2000; Iglesias & Díez, 2009).[8]

Low T3 syndrome was prominent among CKD patients (49.1% had low T3 vs. 28.2% of controls, p=0.001), which parallels prior observations that low T3 is a common metabolic adaptation in chronic illness and correlates with disease severity (Carrero et al., 2007).[19] No significant difference was observed in T4 distributions, aligning with earlier findings that T4 levels often remain within normal limits in CKD despite substantial reductions in T3. TSH was significantly elevated in CKD cases (67.3% had TSH >4.68 compared to 26.4% of controls, p=0.001), consistent with reports of subclinical hypothyroidism being more prevalent among CKD patients, potentially due to reduced renal clearance of TSH or direct thyroid gland dysfunction (Rhee et al., 2015).[14] Lipid disturbances were evident: LDL levels were significantly higher (p=0.001), and HDL levels were substantially lower (p=0.001) in CKD patients compared to controls. These findings are consistent with well-established evidence that dyslipidemia, particularly increased LDL and reduced HDL, is a common metabolic alteration in CKD and is closely linked to the accelerated cardiovascular risk in these patients (Jafar et al., 2003; Rader & Dufour, 2014).[25,26]

Conclusion

In conclusion, this study offers a detailed snapshot of the clinical and biochemical landscape of patients with advanced chronic kidney disease, highlighting the multifaceted nature of CKD and its strong associations with age, rural residence, traditional metabolic risk factors, and profound endocrine and lipid disturbances. The findings emphasise not only the high burden of undiagnosed or late-presenting disease but also the systemic gaps in early detection, especially in underserved populations. The observed prevalence of thyroid dysfunction, anaemia, dyslipidemia, and malnutrition-inflammation-atherosclerosis (MIA) syndrome underscores the necessity for a more integrative and multidisciplinary approach to CKD management. Furthermore, the bidirectional relationships between thyroid function and renal impairment open new avenues for therapeutic research and suggest that endocrine evaluation may play a critical role in comprehensive renal care. Addressing these interlinked abnormalities through routine screening, individualised therapy, and improved healthcare access—particularly in rural regions—could significantly mitigate disease progression and improve patient outcomes. To confirm these correlations and investigate the potential benefits of early metabolic and hormonal correction in altering the course of CKD, further prospective research and interventional trials are necessary.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

References

1. Levey AS, De Jong PE, Coresh J, El Nahas M, Astor BC, Matsushita K, Gansevoort RT, Kasiske BL, Eckardt KU. The definition, classification, and prognosis of chronic kidney disease: a KDIGO Controversies Conference report. Kidney international. 2011 Jul 1;80(1):17-28.
2. Webster AC, Nagler EV, Morton RL, Masson P. Chronic kidney disease. The lancet. 2017 Mar 25;389(10075):1238-52.
3. KDIGO. KDIGO 2012 Clinical Practice Guideline for the Evaluation and Management of Chronic Kidney Disease. Kidney Int Suppl. 2013;3(1):1-150.
4. GBD Chronic Kidney Disease Collaboration. Global, regional, and national burden of chronic kidney disease, 1990–2017: a systematic analysis for the Global Burden of Disease Study 2017. Lancet. 2020;395(10225):709-33.
5. Foreman KJ, Marquez N, Dolgert A, Fukutaki K, Fullman N, McGaughey M, Pletcher MA, Smith AE, Tang K, Yuan CW, Brown JC. Forecasting life expectancy, years of life lost, and all-cause and cause-specific mortality for 250 causes of death: reference and alternative scenarios for 2016–40 for 195 countries and territories. The Lancet. 2018 Nov 10;392(10159):2052-90.
6. Singh AK, Farag YM, Mittal BV, Subramanian KK, Reddy SR, Acharya VN, Almeida AF, Channakeshavamurthy A, Ballal HS, P G, Issacs R. Epidemiology and risk factors of chronic kidney disease in India–results from the SEEK (Screening and Early Evaluation of Kidney Disease) study. BMC nephrology. 2013 Dec;14:1-0.
7. Rhee CM, Kalantar-Zadeh K, Streja E, Carrero JJ, Ma JZ, Lu JL, Kovesdy CP. The relationship between thyroid function and estimated glomerular filtration rate in patients with chronic kidney disease. Nephrology Dialysis Transplantation. 2015 Feb 1;30(2):282-7.
8. Iglesias P, Díez JJ. Thyroid dysfunction and kidney disease. European journal of endocrinology. 2009 Apr;160(4):503-15.
9. Kaptein EM, Quion-Verde HE, Chooljian CJ, Tang WW, Friedman PE, Rodriquez HJ, Massry SG. The thyroid in end-stage renal disease. Medicine. 1988 May 1;67(3):187.
10. Rhee CM, Alexander EK, Bhan I, Brunelli SM. Hypothyroidism

and mortality among dialysis patients. Clinical Journal of the American Society of Nephrology. 2013 Apr 1;8(4):593-601.
11. Vaziri ND. Dyslipidemia of chronic renal failure: the nature, mechanisms, and potential consequences. Am J Physiol Renal Physiol. 2006;290(2):F262-72.
12. Tsimihodimos V, Dounousi E, Siamopoulos KC. Dyslipidemia in chronic kidney disease: an approach to pathogenesis and treatment. Am J Nephrol. 2008;28(6):958-73.
13. Duntas LH, Brenta G. The effect of thyroid disorders on lipid levels and metabolism. EndocrinolMetabClin North Am. 2018;47(1):175-90.
14. Chonchol M, Lippi G, Salvagno G, Zoppini G, Muggeo M, Targher G. Prevalence of subclinical hypothyroidism in patients with chronic kidney disease. Clin J Am SocNephrol. 2008;3(5):1296-300.
15. Hill NR, Fatoba ST, Oke JL, Hirst JA, O'Callaghan CA, Lasserson DS, Hobbs FR. Global prevalence of chronic kidney disease–a systematic review and meta-analysis. PloS one. 2016 Jul 6;11(7):e0158765.
16. United States Renal Data System. USRDS Annual Data Report: Epidemiology of kidney disease in the United States. Bethesda, MD: National Institutes of Health, National Institute of Diabetes and Digestive and Kidney Diseases; 2022.
17. Carrero JJ, Hecking M, Chesnaye NC, Jager KJ. Sex and gender disparities in the epidemiology and outcomes of chronic kidney disease. Nat Rev Nephrol. 2018;14(3):151-64.
18. Vassalotti JA, Centor R, Turner BJ, Greer RC, Choi M, Sequist TD. Practical approach to detection and management of chronic kidney disease for the primary care clinician. Am J Med. 2019;132(3):278-86.
19. Garofalo C, Borrelli S, Minutolo R, Chiodini P, De Nicola L, Conte G. A systematic review and meta-analysis suggests obesity predicts onset of chronic kidney disease in the general population. Kidney Int. 2017;91(5):1224-35.
20. Saran R, Robinson B, Abbott KC, Agodoa LYC, Bragg-Gresham J, Balkrishnan R, et al. US Renal Data System 2020 Annual Data Report: Epidemiology of kidney disease in the United States. Am J Kidney Dis. 2020;75(1):A1-A8.
21. Bello AK, Levin A, Tonelli M, Okpechi IG, Feehally J, Harris D, et al. Global kidney health atlas: a report by the International Society of Nephrology on the current state of organization and structures for kidney care across the globe. Kidney Int Suppl. 2019;9(2):e1-e88.
22. Stauffer ME, Fan T. Prevalence of anemia in chronic kidney disease in the United States. PLoS ONE. 2014;9(1):e84943.
23. Pecoits-Filho R, Heimbürger O, Bárány P, Suliman M, Fehrman-Ekholm I, Lindholm B, et al. Associations between circulating inflammatory markers and residual renal function in CRF patients. Am J Kidney Dis. 2010;45(5):931-40.
24. KAPTEIN EM, QUION-VERDE HE, CHOOLJIAN CJ, TANG WW, FRIEDMAN PE, RODRIQUEZ HJ, MASSRY SG. The thyroid in end-stage renal disease. Medicine. 1988 May 1;67(3):187.
25. Jafar TH, Stark PC, Schmid CH, Landa M, Maschio G, de Jong PE, et al. Proteinuria as a modifiable risk factor for the progression of non-diabetic renal disease. Kidney Int. 2003;64(3):1131-40.
26. Rader DJ, Dufour DR. Lipid and lipoprotein disorders in chronic kidney disease. Clin Chem. 2014;60(1):45-53.