Section: General Medicine

Original Research Article

THYROID DYSFUNCTION AND ITS PHENOTYPIC PATTERNS AMONG PATIENTS WITH CHRONIC KIDNEY DISEASE: A CROSS-SECTIONAL STUDY FROM A NORTH INDIAN TERTIARY CARE CENTRE

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Received *: 08/06/2026*
Received in revised form *: 23/07/2026*
Accepted *: 06/08/2026*

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DOI: 10.70034/ijmedph.2026.3.457

Source of Support: Nil,
Conflict of Interest: None declared

Int J Med Pub Health
2026; 16 (3); 2905-2910

ABSTRACT

Background: The kidneys play a critical role in thyroid hormone metabolism, clearance, and excretion, while thyroid hormones are essential for maintaining renal hemodynamics and tubular function. Consequently, chronic kidney disease (CKD) is associated with multiple alterations in the hypothalamic-pituitary-thyroid (HPT) axis. We aimed to evaluate the prevalence and phenotypic patterns of thyroid dysfunction across different stages of CKD and compared them to healthy controls.

Materials and Methods: We conducted a cross-sectional study of 100 participants: 50 healthy controls, 30 patients with CKD on conservative management, and 20 patients with CKD undergoing maintenance hemodialysis. Serum triiodothyronine (TT3), thyroxine (TT4), and thyroid-stimulating hormone (TSH) were measured. Kidney function was assessed using estimated creatinine clearance (eCrCl) by the Cockcroft-Gault formula and urine albumin-to-creatinine ratio (ACR). Thyroid dysfunction was classified into euthyroid, low-T3 syndrome, subclinical hypothyroidism, and overt hypothyroidism.

Results: The overall prevalence of thyroid dysfunction was 60.0% in conservatively managed patients and 95.0% in hemodialysis patients, compared to only 6.0% in controls. low-T3 syndrome was the dominant phenotype, present in 33.3% of conservatively managed patients and 55.0% of hemodialysis patients, while absent in controls. Multivariable logistic regression revealed that the odds of thyroid dysfunction were significantly higher in both conservative (OR = 21.43, 95% CI: 5.13–89.56) and dialysis groups (OR = 256.23, 95% CI: 23.75–2764.22) independent of age and sex. Within the CKD cohort, dialysis dependence was the primary predictor of dysfunction (OR = 15.43, 95% CI: 1.43–166.21). Overall, TT3 levels correlated positively with eCrCl ($r = 0.63$, $p < 0.001$) and negatively with ACR ($r = -0.36$, $p = 0.0002$), though no significant correlation was found within the CKD-only subgroup.

Conclusion: Thyroid dysfunction, particularly low-T3 syndrome, is highly prevalent in CKD and is strongly associated with CKD status and dialysis dependence. Within established CKD, thyroid abnormalities appear to reflect dialysis exposure and systemic illness burden rather than a simple linear relationship with eCrCl. These findings suggest that clinicians should consider structured thyroid screening in advanced CKD and dialysis patients.

Keywords: chronic kidney disease; hemodialysis; thyroid dysfunction; low-T3 syndrome; subclinical hypothyroidism; eCrCl.

INTRODUCTION

Chronic kidney disease (CKD) is a global health challenge associated with significant morbidity, mortality, and economic burden, particularly in developing nations like India.[1,2] The kidneys and the thyroid gland share a complex, bidirectional homeostatic relationship. The kidney is not only a target organ for thyroid hormone action but also participates in the clearance, excretion, and peripheral metabolism of thyroid hormones.[3,4] Thyroid hormones, in turn, play a crucial role in regulating renal development, renal blood flow, glomerular filtration rate (GFR), and tubular transport systems.[5,6] Advanced renal dysfunction is associated with profound alterations in thyroid hormone synthesis, transport, and peripheral action.[7,8] In patients with CKD, several factors contribute to thyroid abnormalities, including reduced peripheral deiodination of thyroxine (T4) to triiodothyronine (T3), altered thyroid hormone-binding protein levels, chronic uremic inflammation, malnutrition, and blunted pituitary TSH response to thyrotropin-releasing hormone (TRH).[9,10] The most frequent endocrine alteration observed in patients with CKD is the "low-T3 syndrome" (also known as euthyroid sick syndrome or non-thyroidal illness syndrome), characterized by low serum T3 levels in the presence of normal or low T4 and TSH levels.[11,12] Despite the known prevalence of these abnormalities, there remains clinical uncertainty regarding how thyroid dysfunction correlates with the severity of renal impairment across different stages of CKD and under different management modalities.[13,14] Specifically, whether maintenance hemodialysis helps to normalize or further impairs thyroid profiles remains controversial, with conflicting findings reported in local and international literature.[15,16] Furthermore, few studies in the Indian population have evaluated the association between thyroid status and the severity of renal impairment using both eCrCl and albuminuria categories.[1,2]

The present study was designed to systematically characterize the prevalence and phenotypic patterns of thyroid dysfunction in patients with CKD (both conservatively managed and on maintenance hemodialysis) compared to healthy controls in a North Indian population. Age and sex were adjusted for in the multivariable analysis. We also sought to evaluate the association between thyroid hormone levels and key markers of kidney function, including eCrCl and ACR.

MATERIALS AND METHODS

This cross-sectional, hospital-based study was conducted at the Department of General Medicine, Santosh Medical College and Hospital, Ghaziabad, Uttar Pradesh, India. The study period ran from the initiation of patient screening until the target sample size was reached. The protocol was reviewed and approved by the Institutional Ethics Committee. All participants or their legal representatives provided written informed consent prior to enrolment.

Sample size calculation: The target sample size was calculated using the standard formula for comparing two independent group means:

$n = (s_1^2 + s_2^2) \times [Z\_(1-\alpha/2) + Z\_(1-\beta)]^2 / (\bar{x}_1 - \bar{x}_2)^2$

where n is the required sample size per group, s_1 and s_2 are the standard deviations of the two groups, Z_ (1-α/2) is the standard normal deviation for a two-tailed alpha level (1.96 for α = 0.05), Z_ (1-β) is the standard normal deviation for statistical power (0.84 for 80% power), and $\bar{x}_1$ and $\bar{x}_2$ are the expected group means. Based on prior studies of thyroid hormone levels in CKD patients and healthy controls, a total sample size of 100 participants (50 healthy controls and 50 patients with CKD) was determined to provide over 80% power at a 5% significance level to detect clinically meaningful differences in thyroid parameters.

Participants: A total of 100 participants were enrolled and divided into two main cohorts:

1. **Control Group (n = 50):** Healthy individuals without CKD, with normal renal function (estimated creatinine clearance [eCrCl] ≥ 90 mL/min) and no history of thyroid disease or other major systemic disorders.
2. **CKD Cohort (n = 50):** Adult patients (aged 18–65 years) diagnosed with CKD (stages 3–5) for at least 3 months, based on history, laboratory indicators, and ultrasonographic findings. This cohort was further subdivided into:

CKD-Conservative Subgroup (n = 30) : CKD patients managed conservatively without renal replacement therapy.

CKD-Dialysis Subgroup (n = 20) : CKD patients on maintenance hemodialysis for at least 1 month (median dialysis vintage: 42.0 months, interquartile range: 24.5–60.3 months). Exclusion criteria for all participants included a history of thyroid disorders, prior or current use of thyroid medications, acute kidney injury, pregnancy, active malignancy, history of autoimmune diseases, or family history of goiter.

Laboratory measurements: Under strict aseptic precautions, 2 mL of venous blood was collected from the median cubital vein after an overnight fasting period. Samples were allowed to clot for 30–45 minutes and then centrifuged at 3000 rpm for 5 minutes. The supernatant serum was used for immediate biochemical analysis. Blood urea (urease method) and serum creatinine (Jaffe's alkaline picrate method) were estimated on a Beckman Coulter AU 480 autoanalyzer (Beckman Coulter, Brea, CA, USA). Estimated creatinine clearance (eCrCl) was calculated using the Cockcroft-Gault formula:

eCrCl (mL/min) = [(140 - age) × weight (in kg)] / [72 × serum creatinine (in mg/dL)] × [0.85 if female]

Albuminuria was assessed via urine albumin-to-creatinine ratio (ACR) from a spot urine sample.

Total T3 (TT3), total T4 (TT4), and thyroid-stimulating hormone (TSH) were determined on the Access 2 Immunoassay System (Beckman Coulter) using the chemiluminescent immunoassay technique. Euthyroid status was defined as TT3 0.8–2.0 ng/mL, TT4 5.1–12.5 µg/dL, and TSH 0.4–4.5 µIU/mL. low-T3 syndrome was defined as TT3 < 0.8 ng/mL with TSH ≤ 4.5 µIU/mL. Subclinical hypothyroidism was defined as TSH > 4.5 µIU/mL with normal TT4. Overt hypothyroidism was defined as TSH > 4.5 µIU/mL with TT4 < 5.1 µg/dL.

Statistical analysis: Continuous variables are reported as mean ± SD or median (IQR) based on normality, and categorical variables as counts and percentages. Comparisons between the three groups (Controls, CKD-Conservative, CKD-Dialysis) were performed using one-way ANOVA or Kruskal-Wallis tests for continuous variables, and Chi-square tests for categorical variables.

Logistic regression models were constructed to evaluate the association between study groups and thyroid dysfunction (defined as any thyroid phenotype other than euthyroid), adjusting for age and sex. A secondary logistic model evaluated the independent predictors of thyroid dysfunction within the CKD-only subgroup, including dialysis binary status, eCrCl, serum albumin, and hemoglobin. Pearson and Spearman correlation coefficients were calculated to explore relationships between thyroid hormone levels and renal markers. All statistical analyses were conducted using Python 3.13 (pandas, scipy. stats, statsmodels) and figures were plotted in R 4.2.2 using ggplot2. A two-sided p-value < 0.05 was considered statistically significant.

RESULTS

The baseline demographics and clinical variables of the 100 participants are summarized in [Table 1]. The mean age was 42.24 ± 16.41 years in the Control group, 51.20 ± 14.17 years in the CKD-Conservative group, and 54.50 ± 7.07 years in the CKD-Dialysis group (overall p = 0.0018). Male sex was more frequent in the CKD subgroups compared to the healthy controls (60.0% in conservative, 65.0% in dialysis, vs 34.0% in controls; p = 0.0186). Renal markers, serum albumin, and hemoglobin levels differed markedly across the three study groups (all p < 0.001). The mean eCrCl was 104.82 ± 13.18 mL/min in the Control group, 24.19 ± 11.91 mL/min in the CKD-Conservative group, and 8.66 ± 2.44 mL/min in the CKD-Dialysis group. Median ACR increased from 10.60 mg/g (IQR: 5.55–15.35) in controls to 128.30 mg/g (IQR: 50.43–260.47) in conservative CKD and 237.70 mg/g (IQR: 149.05–319.60) in dialysis patients. The prevalence of thyroid dysfunction was significantly higher in CKD patients compared to healthy controls (60.0% in CKD-Conservative, 95.0% in CKD-Dialysis vs 6.0% in controls; p < 0.001) [Table 1]. The phenotypic patterns of thyroid dysfunction also varied significantly across the groups (p < 0.001) (Supplementary [Table 1 and Figure 3]). In the Control group, 47/50 patients (94.0%) were euthyroid, and the remaining 3 patients (6.0%) had subclinical hypothyroidism. In contrast, in the CKD-Conservative group, only 12/30 (40.0%) were euthyroid; low-T3 syndrome was the most common abnormality (10/30, 33.3%), followed by subclinical hypothyroidism (5/30, 16.7%) and overt hypothyroidism (3/30, 10.0%). Among the CKD-Dialysis group, euthyroid status fell to just 1/20 (5.0%), with low-T3 syndrome affecting 11/20 (55.0%), subclinical hypothyroidism affecting 6/20 (30.0%), and overt hypothyroidism in 2/20 (10.0%). No controls demonstrated low-T3 syndrome. Multivariable logistic regression confirmed a highly significant, independent association between CKD management groups and thyroid dysfunction [Table 2]. After adjusting for age and sex, patients in the CKD-Conservative group had more than a 20-fold increased odds of thyroid dysfunction compared to controls (OR = 21.43, 95% CI: 5.13–89.56, p < 0.001). Patients undergoing maintenance hemodialysis demonstrated a massive increase in risk, with an odds ratio of 256.23 (95% CI: 23.75–2764.22, p < 0.001). Age and sex were not significantly associated with thyroid dysfunction in this model. Because of sparse events in some cells, odds ratios should be interpreted as directionally supportive rather than precise effect estimates. Within the CKD cohort (n = 50), a secondary logistic model evaluated predictors of thyroid dysfunction [Table 2]. Dialysis status was the only statistically significant independent predictor of dysfunction, associated with a 15-fold increased odds compared to conservative management (OR = 15.43, 95% CI: 1.43–166.21, p = 0.024). Within this subgroup, eCrCl, serum albumin, and hemoglobin were not independently associated with thyroid dysfunction. Because of sparse events in some cells, odds ratios should be interpreted as directionally supportive rather than precise effect estimates. In the overall study population, corrected TT3 levels demonstrated a strong positive linear correlation with eCrCl (r = 0.63, p < 0.001) and a moderate negative correlation with ACR (r = -0.36, p = 0.0002) (Supplementary Table 3, Figure 1 and Figure 2). TSH was weakly negatively correlated with eCrCl (r = -0.26, p = 0.008), while TT4 showed no significant correlation (r = 0.078, p = 0.44). The TT3/TT4 ratio also correlated strongly with eCrCl (r = 0.56, p < 0.001) and negatively with ACR (r = -0.36, p = 0.0002).Importantly, when analyzed within the CKD-only cohort (n = 50), the linear correlations between thyroid hormones and renal markers were no longer statistically significant (e. g., TT3 vs eCrCl: r = -0.12, p = 0.42; TSH vs eCrCl: r = -0.17, p = 0.24) (Supplementary Table 3). This suggests that while thyroid hormone levels decline globally as patients progress from healthy status to CKD, minor stage-to-stage eCrCl variations within the CKD cohort do not linearly predict thyroid hormone levels. Among CKD patients, thyroid dysfunction was also analyzed

across KDIGO risk categories. The prevalence of thyroid dysfunction was 80.0% in the High risk category (4/5 patients) and 73.3% in the Very High risk category (33/45 patients) (p = 1.0) (Supplementary [Table 2] and Supplementary [Figure 1]).

Table 1: Demographics, clinical markers, and thyroid status by study group

Variable	Control (n = 50)	CKD-Conservative (n = 30)	CKD-Dialysis (n = 20)	P-value
Age, mean ± SD (years)	42.24 ± 16.41	51.20 ± 14.17	54.50 ± 7.07	0.002
Male sex, n/N (%)	17/50 (34.0%)	18/30 (60.0%)	13/20 (65.0%)	0.019
eCrCl, mean ± SD (mL/min)	104.82 ± 13.18	24.19 ± 11.91	8.66 ± 2.44	<0.001
ACR, median (IQR) (mg/g)	10.60 (5.55–15.35)	128.30 (50.43–260.47)	237.70 (149.05–319.60)	<0.001
TT3 corrected, mean ± SD (ng/mL)	1.17 ± 0.31	0.70 ± 0.23	0.69 ± 0.28	<0.001
TT4, mean ± SD (µg/dL)	7.66 ± 1.69	7.43 ± 2.07	7.32 ± 1.65	0.731
TSH, median (IQR) (µIU/mL)	2.34 (1.91–2.95)	3.23 (1.93–4.63)	3.73 (2.52–6.03)	0.006
TT3/TT4 ratio, mean ± SD	0.16 ± 0.05	0.10 ± 0.03	0.10 ± 0.04	<0.001
Serum Albumin, mean ± SD (g/dL)	4.24 ± 0.45	3.63 ± 0.52	3.29 ± 0.29	<0.001
Hemoglobin, mean ± SD (g/dL)	13.60 ± 1.19	10.34 ± 1.23	9.13 ± 0.92	<0.001
RDW, mean ± SD (%)	12.60 ± 0.77	13.87 ± 1.15	13.30 ± 0.90	<0.001
Thyroid Dysfunction, n/N (%)	3/50 (6.0%)	18/30 (60.0%)	19/20 (95.0%)	<0.001

Table 2: Multivariable logistic regression models for thyroid dysfunction

Model and Term	Odds Ratio (OR)	95% Confidence Interval (CI)	P-value
Model 1: All Participants (n = 100)			
CKD-Conservative (vs. Control)	21.43	5.13–89.56	<0.001
CKD-Dialysis (vs. Control)	256.23	23.75–2764.22	<0.001
Age (per year)	1.02	0.98–1.06	0.342
Male sex (vs. Female)	0.89	0.26–3.07	0.849
Model 2: CKD Patients Only (n = 50)			
Dialysis Dependence (vs. Conservative)	15.43	1.43–166.21	0.024
eCrCl (per mL/min)	1.04	0.95–1.13	0.388
Serum Albumin (per g/dL)	0.43	0.09–2.09	0.296
Hemoglobin (per g/dL)	0.94	0.45–1.94	0.857

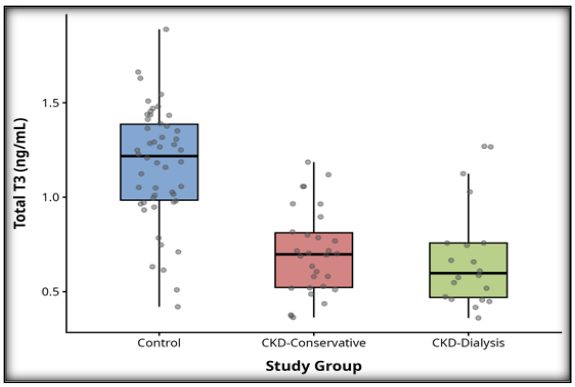

Figure 1: Corrected total T3 levels by study group

Boxplot showing the distribution of corrected total T3 levels in healthy controls, CKD patients on conservative management, and CKD patients undergoing hemodialysis.

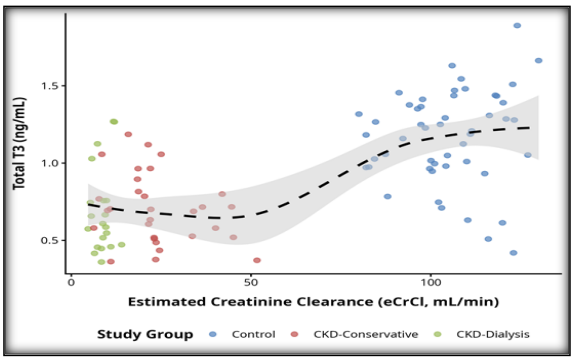

Figure 2: Scatter plot of eCrCl versus corrected total T3 levels

Scatter plot demonstrating the correlation between eCrCl and corrected total T3 levels across all participants, colored by study group, with a LOESS fit line.

Supplementary Material

Supplementary Table 1: Thyroid phenotype counts by study group

Study Group	Euthyroid	low-T3 syndrome	Subclinical Hypothyroidism	Overt Hypothyroidism	Total
Control	47 (94.0%)	0 (0.0%)	3 (6.0%)	0 (0.0%)	50
CKD-Conservative	12 (40.0%)	10 (33.3%)	5 (16.7%)	3 (10.0%)	30
CKD-Dialysis	1 (5.0%)	11 (55.0%)	6 (30.0%)	2 (10.0%)	20
Total	60 (60.0%)	21 (21.0%)	14 (14.0%)	5 (5.0%)	100

Supplementary Table 2: Thyroid status by KDIGO risk category among CKD patients (n = 50)

KDIGO Risk Category	Euthyroid	Thyroid Dysfunction	Total
High	1 (20.0%)	4 (80.0%)	5
Very high	12 (26.7%)	33 (73.3%)	45
Total	13 (26.0%)	37 (74.0%)	50

Supplementary Table 3: Correlation coefficients between thyroid hormones and biochemical markers

Scope and Variables	Pearson r	P-value	Spearman rho	P-value
All Participants (n = 100)				
TT3 vs. eCrCl	0.63	<0.001	0.56	<0.001
TT3 vs. ACR	-0.36	<0.001	-0.55	<0.001
TT3 vs. Albumin	0.53	<0.001	0.53	<0.001
TT3 vs. Hemoglobin	0.50	<0.001	0.51	<0.001
TT3 vs. RDW	-0.38	<0.001	-0.36	<0.001
TSH vs. eCrCl	-0.26	0.008	-0.32	0.001
TT4 vs. eCrCl	0.08	0.441	0.02	0.855
CKD Patients Only (n = 50)				
TT3 vs. eCrCl	-0.12	0.416	-0.03	0.813
TT3 vs. ACR	0.09	0.514	0.07	0.626
TSH vs. eCrCl	-0.17	0.240	-0.22	0.128

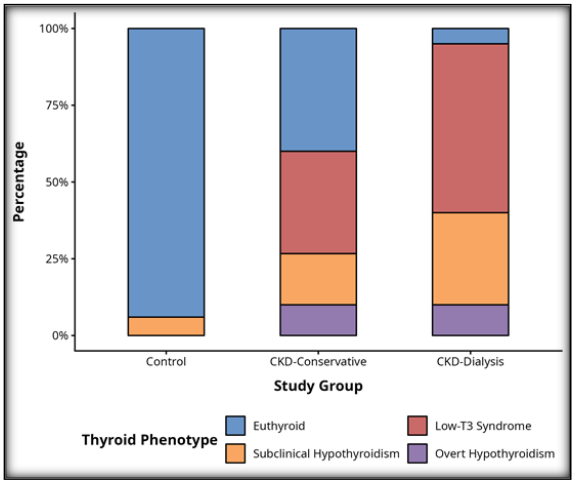

Figure 3: Thyroid phenotype distribution by study group

Stacked bar chart showing the percentage of each thyroid phenotype (Euthyroid, low-T3 syndrome, Subclinical Hypothyroidism, Overt Hypothyroidism) across the three study groups.

DISCUSSION

This study evaluated the prevalence and phenotypic patterns of thyroid dysfunction in patients with CKD from a northwestern Indian population. Our results demonstrate that thyroid abnormalities are extremely common in CKD patients, affecting 60.0% of conservatively managed patients and 95.0% of patients on maintenance hemodialysis, compared to a baseline prevalence of only 6.0% in healthy controls. low-T3 syndrome was the predominant phenotype in both CKD groups, while being entirely absent in healthy individuals. Declining renal function was strongly and independently associated with thyroid dysfunction, and dialysis dependence further escalated this risk. Our findings align with the established physiological role of the kidney in thyroid hormone metabolism. The kidneys contain high concentrations of deiodinases that convert T4 to the active T3 hormone. In CKD, a reduction in functional renal mass leads to decreased peripheral conversion, explaining the high prevalence of low-T3 syndrome.[3,4,11] This "euthyroid sick syndrome" is widely regarded as a protective adaptation to reduce metabolic energy expenditure during chronic disease, but it may also represent a pathological state of tissue-level hypothyroidism.[12,18] In our multivariable regression analysis, CKD patients had significantly higher odds of thyroid dysfunction compared to controls, even after adjusting for age and sex. Within the CKD cohort, dialysis dependence emerged as the strongest predictor of thyroid dysfunction, associated with a 15-fold increased odds compared to conservative management. This is consistent with studies by Paudel et al. and Obasuyi et al., which reported high rates of thyroid abnormalities in maintenance hemodialysis cohorts.[17,19] The uremic state, coupled with dialysis-induced factors (such as heparin-mediated free fatty acid release and loss of thyroid hormones in dialysate), likely exacerbates thyroid hormone synthesis and protein-binding defects.[8] A key finding in our correlation analysis was that while TT3 and the TT3/TT4 ratio correlated strongly with GFR and ACR across the entire study population, these linear correlations disappeared when restricting the analysis to the CKD cohort. This indicates that the HPT axis dysfunction in CKD is not a simple linear function of GFR decline. Once a patient reaches moderate-to-severe CKD, systemic factors such as chronic uremic inflammation, malnutrition, metabolic acidosis, and dialytic clearance play a more dominant role in suppressing thyroid hormone levels than minor fluctuations in residual renal clearance. This interpretation is supported by Basu et al. and Narasaki et al., who highlighted the multifactorial, non-linear drivers of thyroid dysfunction in end-stage renal disease.[18,20] Given that the clinical features of hypothyroidism (e. g., fatigue, cold intolerance, dry skin, anemia) closely overlap with the symptoms of uremia and anemia in CKD, thyroid dysfunction is frequently missed in this population.[10,13] Our results suggest that relying solely on clinical presentation is insufficient, and advanced CKD patients, particularly those on hemodialysis, should undergo systematic biochemical screening. This study has several strengths, including the presence of a healthy control group, a clear sub-grouping of CKD patients by management modality, and the calculation of eCrCl and ACR. However, some limitations must be acknowledged. First, the cross-sectional design

prevents us from establishing a causal relationship between renal function decline and thyroid dysfunction. Second, the study was conducted at a single tertiary center with a moderate sample size, limiting the generalizability of our findings. Third, we did not measure free hormone fractions (free T3 and free T4) or markers of chronic inflammation (such as C-reactive protein or IL-6), which could have provided further mechanistic insights. Fourth, detailed dialysis parameters, such as the exact timing of blood draws relative to hemodialysis sessions, the avoidance of heparin prior to blood sampling, and measures of dialysis adequacy (Kt/V), were not available.

CONCLUSION

In conclusion, thyroid dysfunction, particularly low-T3 syndrome, is highly prevalent in CKD and is strongly associated with CKD status and dialysis dependence. Within established CKD, thyroid abnormalities appear to reflect dialysis exposure and systemic illness burden rather than a simple linear relationship with eCrCl. These findings support the clinical utility of considering structured thyroid screening in advanced CKD and dialysis patients.

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