

Association Between Thyroid Dysfunction and Chronic Kidney Disease: A Cross-Sectional Study in a Rural Tertiary-Care Centre

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ABSTRACT

Background: Thyroid dysfunction is frequently reported in CKD, tends to increase with disease severity, and is often dominated by a low T3 pattern. It may also be overlooked in routine care because symptoms overlap with uremia, so screening is not consistently performed in many settings.

Aim: To determine the prevalence and spectrum of thyroid dysfunction among adults with chronic kidney disease.

Objectives: 1). To estimate the prevalence and describe the spectrum of thyroid dysfunction in CKD patients. 2). To assess the association between CKD stage and thyroid function status. 3). To compare mean T3, T4 and TSH levels across CKD stages.

Materials and Methods: Hospital-based cross-sectional study (May 2024–November 2025) among adults with CKD (n = 100). Serum T3, T4 and TSH were measured, and thyroid ultrasonography was performed.

Results: Mean age was 58.26 ± 10.79 years and 55% were men. CKD stage distribution was: Stage 1 4%, Stage 2 9%, Stage 3 21%, Stage 4 34%, Stage 5 32%. Mean thyroid values were T3 0.93 ± 0.35 ng/mL, T4 8.23 ± 1.99 µg/dL, and TSH 4.34 ± 5.42 µIU/mL. Overall, 54% had thyroid dysfunction: low T3 syndrome 34%, overt hypothyroidism 10%, and subclinical hypothyroidism 10%. Thyroid dysfunction increased with CKD stage (Chi-square 32.80, $p = 0.001$). Hormone levels differed across stages (ANOVA: T3 $p = 0.001$; T4 $p = 0.01$; TSH $p = 0.041$).

Conclusion: More than half of CKD patients had thyroid dysfunction, with a clear stage-wise rise and predominance of low T3 syndrome. Thyroid testing (T3, T4 and TSH) may be most informative in Stage 3–5 CKD, where abnormalities were concentrated.

Keywords: chronic kidney disease, thyroid dysfunction, low T3 syndrome, hypothyroidism, TSH

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INTRODUCTION

Chronic kidney disease (CKD) is increasingly encountered in routine Indian clinical practice. Delayed detection remains a major concern, because many patients first enter care after complications and comorbidities have already accumulated. Community screening studies from South India have demonstrated that CKD is prevalent beyond hospital settings and that awareness is limited, contributing to late presentation. [1] Pooled Indian evidence has similarly suggested a sustained, and possibly increasing, burden over successive study periods. [2]

Thyroid dysfunction is also frequently described in CKD, yet screening is not uniformly routine. Several mechanisms may contribute, including altered iodide handling with declining renal function, chronic inflammation, uremic toxins, changes in hormone-binding proteins, and reduced peripheral conversion of T4 to T3. Symptoms are often non-specific and overlap with the CKD phenotype. Fatigue, constipation, appetite change, dry skin, cold intolerance, bradycardia, and low mood may be attributed to uremia, anemia, malnutrition, or medications unless thyroid testing is deliberately considered. [3]

A common biochemical pattern in chronic illness is the low T3 state, often discussed under non-thyroidal illness syndrome. T3 may fall due to changes in deiodinase activity and central axis signalling, and TSH may not rise in the manner expected in

primary hypothyroidism. This pattern often tracks illness severity, which makes it clinically relevant in CKD rather than incidental. [4] In CKD cohorts, low T3 syndrome has been repeatedly linked to lower kidney function and more advanced disease. [5] Hypothyroid states are also important. Subclinical hypothyroidism has been reported with notable frequency in non-dialysis CKD, with large datasets suggesting a graded association with lower eGFR. [6] Beyond symptom burden, the concern extends to cardiovascular risk, particularly lipid abnormalities, vascular tone, cardiac function, and inflammatory pathways, in a population already vulnerable to cardiac events. [7]

Against this background, the present study assessed the frequency and pattern of thyroid dysfunction in adults with CKD in a tertiary-care setting and examined whether thyroid status varied across CKD stages using standard thyroid function testing (T3, T4 and TSH).

MATERIALS AND METHODS

Study design and setting

A hospital-based cross-sectional study was conducted in the Department of Medicine, Adichunchanagiri Hospital and Research Centre. The study period was 18 months (May 2024 to November 2025).

Study population

Adult patients (>18 years, both sexes) admitted under General Medicine and/or Nephrology Department with a diagnosis of chronic kidney disease (CKD) were enrolled.

$n = (z^2pq) / d^2$, where $z = 1.96$ (95% confidence), $p = 34.9\%$, $q = 1 - p$, and $d = 10\%$ absolute precision. The minimum calculated sample size was 96, rounded to 100 participants.

Eligibility criteria

Inclusion criteria

- Age >18 years
- Diagnosed CKD
- Provided written informed consent

Exclusion criteria

- Pre-existing thyroid disease (prior to CKD diagnosis)
- Recent surgery, trauma, or burns
- Known liver disease
- Use of drugs known to alter thyroid function

Ethical considerations

Institutional Ethics Committee approval was obtained prior to study initiation. Written informed consent was secured from all participants. Privacy and confidentiality were maintained.

Data collection and study procedures

Data were recorded using a pre-tested structured proforma.

Clinical assessment: demographic details, CKD duration and stage, comorbidities, medication history, and focused examination for renal and thyroid-related features.

Investigations:

- Serum T3, T4 and TSH
- Ultrasound (USG) thyroid
- Renal function tests including serum creatinine; creatinine clearance calculated using the Cockcroft–Gault formula
- Complete blood count
- Serum electrolytes
- Routine urine analysis
- 24-hour urinary protein (when clinically indicated)
- FNAC thyroid (selected cases)
- ECG

Sample size

Sample size was estimated using the prevalence-based formula:

- Lipid profile
- Thyroid antibody testing (selected patients)
- Random blood sugar (RBS)

Outcomes and operational definitions

Primary outcome: Prevalence of thyroid dysfunction among adults with CKD.

Thyroid status classification: Participants were categorised as euthyroid, low T3 syndrome, overt hypothyroidism, or subclinical hypothyroidism based on serum T3, T4 and TSH, interpreted against the study-centre laboratory reference intervals used during the study period. The same reference intervals were applied uniformly to all participants.

CKD staging: CKD stage was assigned using standard staging criteria documented in the clinical record, and analysed across Stage 1 to Stage 5 categories.

Secondary outcomes:

1. Stage-wise distribution of thyroid status categories across CKD stages.
2. Comparison of mean thyroid hormone levels (T3, T4 and TSH) across CKD stages.

Thyroid ultrasound reporting: USG findings were recorded as normal, mildly enlarged, nodular, or atrophic based on radiology reporting documented in the case file.

Statistical analysis

Descriptive statistics were used for baseline characteristics. Association between CKD stage and thyroid status was assessed using Pearson chi-square. Mean thyroid hormone levels across CKD stages were compared using one-way ANOVA. A p-value <0.05 was considered statistically significant.

RESULTS

Participant and disease profile

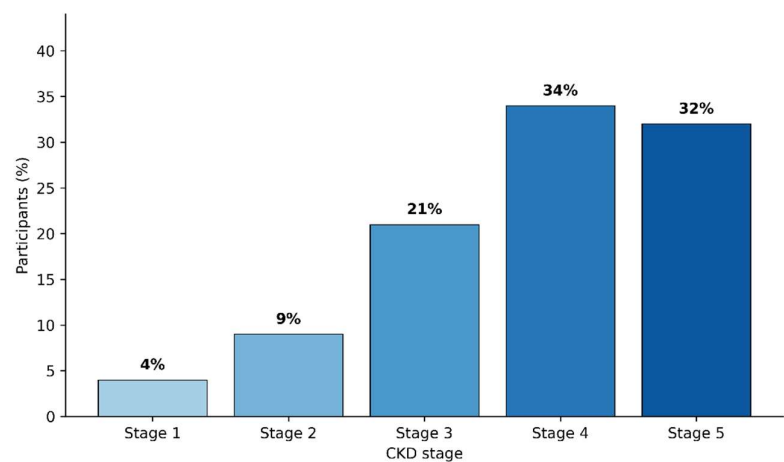
A total of 100 adults with CKD were analysed. Mean age was 58.26 ± 10.79 years, and 55% were men. Most participants had normal BMI (79%). Late-stage disease predominated: Stage 4 (34%) and Stage 5 (32%) together accounted for 66% of the cohort (Table 1; Figure 1).

Table 1. Baseline characteristics, renal profile, and CKD stage distribution (n = 100)

Domain	Variable	Summary
Demography	Age (years), mean $\pm$ SD	58.26 $\pm$ 10.79
	Age group, n (%)	≤ 40 : 4 (4) · 41–50: 20 (20) · 51–60: 32 (32) · 61–70: 30 (30) · >70: 14 (14)
	Sex, n (%)	Male: 55 (55) · Female: 45 (45)
Anthropometry	BMI category, n (%)	Underweight: 7 (7) · Normal: 79 (79) · Overweight: 14 (14)
	BMI (kg/m ²), mean $\pm$ SD	22.10 $\pm$ 2.34
Renal indices	Urea (mg/dL), mean $\pm$ SD	89.04 $\pm$ 41.72
	Serum creatinine (mg/dL), mean $\pm$ SD	4.88 $\pm$ 3.35
	Creatinine clearance (mL/min), mean $\pm$ SD	28.69 $\pm$ 23.88
	eGFR (mL/min/1.73 m ²), mean $\pm$ SD	29.54 $\pm$ 24.72
CKD stage	Distribution, n (%)	Stage 1: 4 (4) · Stage 2: 9 (9) · Stage 3: 21 (21) · Stage 4: 34 (34) · Stage 5: 32 (32)

Values are reported as mean $\pm$ SD or n (%). Percentages are based on a denominator of 100.

Figure 1: CKD stage distribution among participants (% , n = 100)



Values are shown above bars as percentages.

Thyroid function patterns and ultrasound findings

Mean thyroid indices were T3 0.93 ± 0.35 ng/mL, T4 8.23 ± 1.99 µg/dL, and TSH 4.34 ± 5.42 µIU/mL (Table 2). Overall, 54% had thyroid dysfunction: low T3 syndrome 34%, overt

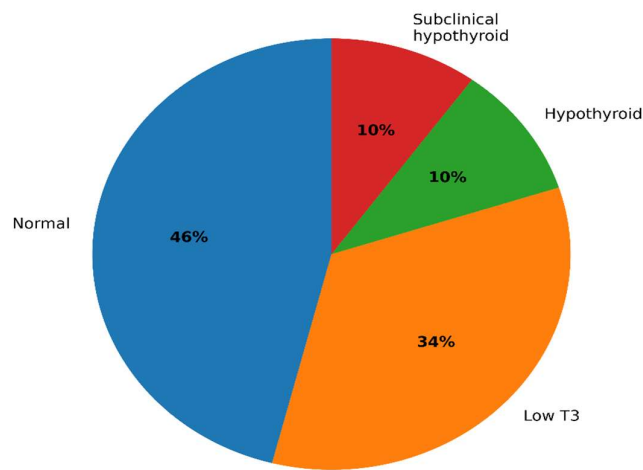
hypothyroidism 10%, and subclinical hypothyroidism 10% (Table 2; Figure 2 and Figure 3). On thyroid ultrasonography, 78% had a normal study, while enlargement (11%), nodularity (8%), and atrophy (3%) were less frequent (Table 2).

Table 2. Thyroid profile, thyroid status, and ultrasound findings (n = 100)

Component	Category / measure	Result
Thyroid hormones	T3 (ng/mL), mean ± SD	0.93 ± 0.35
	T4 (µg/dL), mean ± SD	8.23 ± 1.99
	TSH (µIU/mL), mean ± SD	4.34 ± 5.42
Thyroid status	Euthyroid, n (%)	46 (46)
	Low T3 syndrome, n (%)	34 (34)
	Overt hypothyroidism, n (%)	10 (10)
	Subclinical hypothyroidism, n (%)	10 (10)
Thyroid ultrasound	Normal, n (%)	78 (78)
	Mildly enlarged, n (%)	11 (11)
	Nodular, n (%)	8 (8)
	Atrophic, n (%)	3 (3)

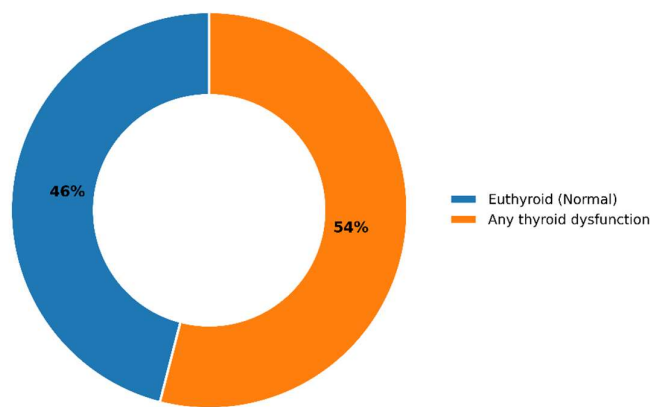
Values are reported as mean ± SD or n (%).

Figure 2: Distribution of thyroid status categories (pie chart, %)



Percentages reflect within-cohort proportions (n = 100).

Figure 3: Euthyroid versus any thyroid dysfunction (donut chart, %)



Any dysfunction combines low T3 syndrome, overt hypothyroidism, and subclinical hypothyroidism.

Relationship between CKD stage and thyroid dysfunction

Thyroid status varied meaningfully with CKD stage (Table 3; Figure 4). In Stages 1 and 2, all participants were euthyroid (100%). In Stage 5, only 18.8% remained euthyroid, while 59.4% had low T3 syndrome. The association between CKD stage and thyroid status was statistically significant (Pearson Chi-square =

32.80; p = 0.001) (Table 3). Mean hormone levels also differed across CKD stages: T3 declined progressively from 1.25 ± 0.19 (Stage 1) to 0.76 ± 0.31 (Stage 5), with significant between-stage differences by one-way ANOVA (T3 p = 0.001; T4 p = 0.01; TSH p = 0.041) (Table 3).

Table 3. Thyroid status and thyroid hormone levels across CKD stages
A. Thyroid status by CKD stage (n, within-stage %)

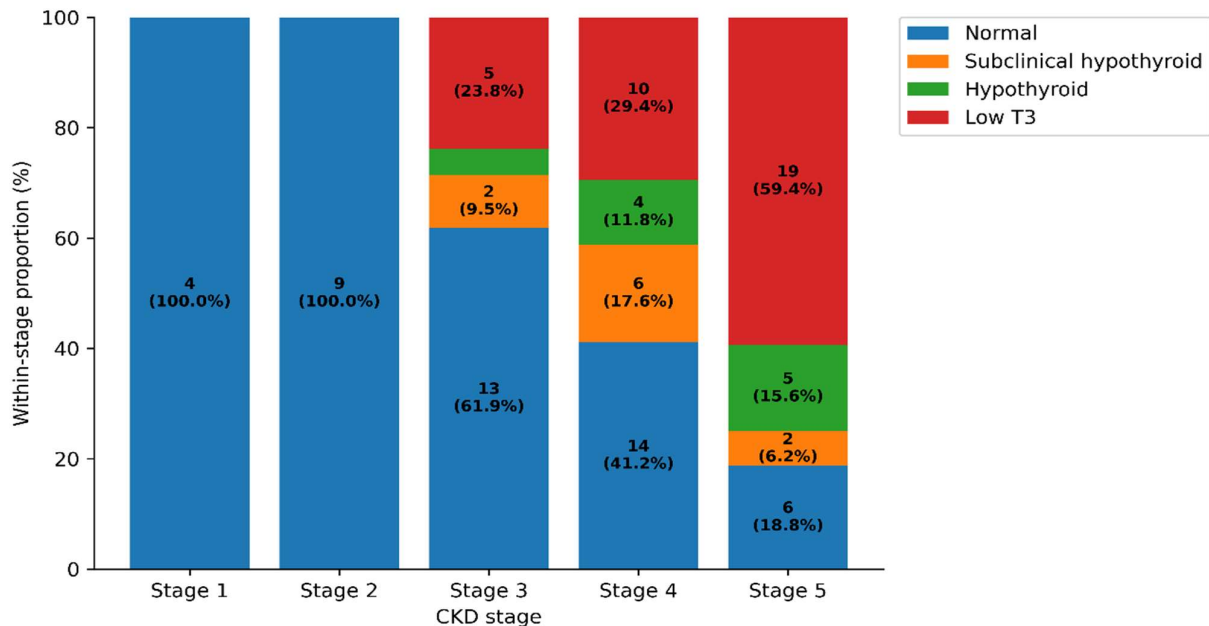
CKD stage (n)	Euthyroid	Subclinical hypothyroid	Overt hypothyroid	Low T3 syndrome
Stage 1 (4)	4 (100.0)	0 (0.0)	0 (0.0)	0 (0.0)
Stage 2 (9)	9 (100.0)	0 (0.0)	0 (0.0)	0 (0.0)
Stage 3 (21)	13 (61.9)	2 (9.5)	1 (4.8)	5 (23.8)
Stage 4 (34)	14 (41.2)	6 (17.6)	4 (11.8)	10 (29.4)
Stage 5 (32)	6 (18.8)	2 (6.3)	5 (15.6)	19 (59.4)

Panel A values are n (within-stage %). Pearson Chi-square = 32.80; p = 0.00

B. Thyroid hormones across CKD stages (mean $\pm$ SD) with ANOVA p-values

CKD stage	T3 (ng/mL)	T4 (μ g/dL)	TSH (μ IU/mL)
Stage 1	1.25 $\pm$ 0.19	8.54 $\pm$ 1.96	2.52 $\pm$ 1.07
Stage 2	1.19 $\pm$ 0.19	8.55 $\pm$ 1.05	1.85 $\pm$ 0.87
Stage 3	1.04 $\pm$ 0.39	8.31 $\pm$ 0.94	3.94 $\pm$ 4.88
Stage 4	0.92 $\pm$ 0.34	8.27 $\pm$ 2.06	4.35 $\pm$ 4.31
Stage 5	0.76 $\pm$ 0.31	7.90 $\pm$ 2.24	5.53 $\pm$ 7.39
p-value (ANOVA)	0.001	0.01	0.041

Panel B values are mean $\pm$ SD; between-stage comparisons were tested using one-way ANOVA (T3 $p = 0.001$; T4 $p = 0.01$; TSH $p = 0.041$).

Figure 4. Thyroid status distribution across CKD stages (100% stacked bar chart)

Segment labels display n and within-stage % where space permits; each bar totals 100% within stage.

DISCUSSION

Thyroid dysfunction was seen in 54% of adults with CKD in this study. The most frequent pattern was low T3 syndrome (34%), while overt hypothyroidism (10%) and subclinical hypothyroidism (10%) contributed another one-fifth. What makes this clinically relevant is that it was not evenly distributed across CKD stages. Thyroid abnormalities clustered in advanced disease, and the association by stage was statistically significant (Chi-square $p = 0.001$). The hormone behaviour also moved in a direction that “fits” CKD physiology: T3 fell as stage worsened, while TSH showed an upward drift.

This pattern is biologically plausible. CKD alters thyroid hormone handling through multiple concurrent mechanisms. Iodide clearance falls, plasma iodide can rise, and thyroid autoregulation may shift. Add uremic toxins, chronic inflammation, altered binding proteins, and reduced peripheral conversion of T4 to T3, and the biochemical picture becomes difficult to interpret using a single simplistic model. [8] In a busy ward, this matters because symptoms are shared territory—fatigue, constipation, appetite change, dry skin, cold intolerance, bradycardia, low mood. Without a deliberate thyroid check, it is easy to label everything as “uremia” or “anemia” and move on.

Low T3 Syndrome

Low T3 being the dominant phenotype is not unexpected. Across CKD literature, low T3 tends to appear early among thyroid

abnormalities and becomes more frequent as kidney function declines. [9,10] In this dataset, the rise in Stage 5 is particularly striking and mirrors that general trend. Low T3 also carries prognostic weight in other renal cohorts. Studies in hemodialysis populations have reported that low T3 is associated with higher long-term mortality even after adjustment for conventional and uremia-related risks. [11] A meta-analysis has also supported a mortality signal in chronic renal failure when low T3 is present. [12] So, for clinicians, a low T3 value in late CKD should not automatically be dismissed as “just a lab variation.” It may reflect physiological stress in a very real way.

One observation needs careful handling: Stages 1–2 were entirely euthyroid (100%). Some reports have shown low T3 even in earlier CKD, especially in outpatient cohorts and in those with intercurrent illness. [9] Here, early-stage numbers were small (Stage 1 $n=4$; Stage 2 $n=9$). So this “clean” early-stage profile may reflect limited sample size, selection by admission patterns, or differences in comorbidity load at the time of sampling, rather than a firm biological boundary.

Hypothyroidism and Subclinical Hypothyroidism

Overt and subclinical hypothyroidism together accounted for 20%, and these categories were concentrated in later stages. This direction matches epidemiologic work where thyroid abnormalities become more common as renal function worsens, even before end-stage disease. [13] Large outpatient datasets have also reported that subclinical hypothyroidism is relatively

common in non-dialysis CKD and shows an association with lower eGFR. [14] Clinically, the relevance is not only symptom overlap. Hypothyroid states can intersect with lipid profile changes, vascular resistance, and cardiac performance—problems that CKD patients already struggle with. Even a modest rise in TSH can feel “bigger” in someone with limited reserve.

Indian hospital-based cohorts have described a similar pattern, with a heavier hypothyroid burden in advanced CKD. [15] That makes sense in real-world wards, where late presentation, diabetes, hypertension, and malnutrition-inflammation features often travel together.

Thyroid Ultrasound

Most participants had a normal thyroid ultrasound (78%), and fewer showed enlargement, nodularity, or atrophy. This is useful clinically because it suggests that thyroid dysfunction in CKD is often functional rather than structural. In practical terms, T3, T4 and TSH remain the centre of evaluation, while ultrasound adds value mainly when nodularity, gland size change, or discordant clinical features raise a separate structural concern.

Clinical Implication

In moderate-to-advanced CKD, a low threshold for thyroid testing is reasonable, particularly when fatigue is disproportionate, bradycardia is unexplained, dyslipidemia is persistent, or nutritional trajectory is poor. T3 may also behave like a rough marker of physiological stress in late CKD, although treatment of low T3 syndrome itself remains uncertain. The

clearer actionable space is the identification of overt or subclinical hypothyroidism, with interpretation done cautiously in the presence of intercurrent illness or medications that alter the thyroid axis.

Limitations

This single-centre, hospital-based cross-sectional study may overestimate prevalence compared with community CKD populations. Early-stage CKD numbers were small, and dialysis status was not analysed separately within advanced CKD; thyroid antibody testing was done only in selected patients, limiting subgroup interpretation.

CONCLUSION

In this CKD cohort, thyroid dysfunction was common (54%) and became more frequent as CKD stage advanced. The dominant pattern was low T3 syndrome (34%). Overt hypothyroidism (10%) and subclinical hypothyroidism (10%) were also seen, mostly in later stages. Across stages, T3 declined and TSH tended to rise, which fits a clear association between worsening renal function and thyroid profile. In practice, checking T3, T4 and TSH can be considered, especially in Stage 3–5 CKD, where missed hypothyroid states may add to fatigue, poor functional status, and cardiovascular risk.

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