

Exploring The Relationship Between Thyroid Dysfunction and Chronic Kidney Disease in Southern Chennai Population

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ABSTRACT

Chronic kidney disease (CKD) is frequently accompanied by endocrine disturbances, yet thyroid dysfunction remains under recognized in routine care. This hospital-based, unmatched case-control study assessed the prevalence and pattern of thyroid dysfunction among adults with CKD in Southern Chennai. A total of 300 participants were enrolled (150 CKD cases recruited from the Nephrology OPD and 150 non-CKD controls from Medicine/General Surgery departments). CKD was defined by KDIGO criteria (eGFR <60 mL/min/1.73 m² and/or evidence of kidney damage for >3 months). Thyroid function was evaluated using serum TSH and free thyroxine (FT4). CKD cases were older and had higher burdens of diabetes and hypertension, with significantly reduced eGFR compared with controls. Thyroid abnormalities were significantly more common in CKD: subclinical hypothyroidism was observed in 16.7% and overt hypothyroidism in 15.3% of cases, whereas controls were predominantly euthyroid (86%). In multivariable analysis, hypothyroidism showed a strong independent association with CKD (odds ratio 3.87), alongside established risk factors including age, diabetes, hypertension, and BMI. These findings highlight a substantial burden of hypothyroidism in CKD and support incorporating routine thyroid screening into CKD evaluation and follow-up.

Keywords: Renal Insufficiency, Chronic; Hypothyroidism; Thyroid Diseases; Thyrotropin; Case-Control Studies; India.

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INTRODUCTION

Chronic kidney disease (CKD) is a major global public health concern and is increasingly recognised as a systemic disorder associated with multiple metabolic and endocrine abnormalities.¹ Among these, thyroid dysfunction is one of the most frequently reported yet underdiagnosed conditions in patients with CKD.²⁻⁴ Alterations in thyroid hormone synthesis, metabolism, and clearance can begin early in renal impairment and intensify with declining kidney function, resulting in abnormalities ranging from low triiodothyronine (T3) syndrome (non-thyroidal illness syndrome) to subclinical and overt hypothyroidism.²⁻⁵ These changes are attributed to impaired peripheral conversion of thyroxine (T4) to T3, chronic inflammation and malnutrition-related factors, altered binding proteins, and dysregulation of the hypothalamic-pituitary-thyroid axis in the uraemic state.^{2,5-7} In addition, reduced

glomerular filtration decreases renal iodine clearance, potentially increasing iodide exposure and contributing to thyroid hypofunction through autoregulatory mechanisms.^{2,3}

A substantial body of recent literature has consistently demonstrated a higher prevalence of thyroid dysfunction among patients with CKD compared with the general population, with prevalence rising as estimated glomerular filtration rate (eGFR) declines and CKD stage advances.^{3,4,8,9} Subclinical hypothyroidism is commonly reported as a predominant abnormality in non-dialysis CKD cohorts.^{3,8,9} Evidence from Indian settings also supports a high burden, with studies from North and Central India reporting increased hypothyroidism/subclinical hypothyroidism in CKD and documenting worsening thyroid abnormalities with progressive renal dysfunction.^{4,9}

Thyroid dysfunction in CKD has important clinical implications. Both hypothyroidism and subclinical hypothyroidism have been linked with adverse cardiovascular risk profiles and outcomes, which may further compound the already elevated cardiovascular morbidity and mortality in CKD.¹⁰⁻¹² In haemodialysis populations, abnormal thyroid status has also been associated with higher mortality risk.¹³ Because clinical features of hypothyroidism overlap substantially with symptoms of chronic renal failure, thyroid dysfunction may remain underrecognized in routine practice, delaying appropriate detection and management.^{2,3}

Recent studies have evaluated biochemical markers to improve the detection of thyroid dysfunction in CKD; the FT3/FT4 ratio, reflecting impaired peripheral conversion, has shown prognostic value in CKD cohorts.¹⁴ While levothyroxine is standard for overt hypothyroidism, evidence for renal benefit in CKD with subclinical hypothyroidism remains inconsistent, with population-based data showing no clear reduction in adverse renal outcomes.¹⁵

Despite this evidence, controlled region-specific case-control data from Southern India remain limited. Therefore, this hospital-based case-control study in Southern Chennai assesses the prevalence and patterns of thyroid dysfunction in adults with CKD compared with non-CKD controls, while accounting for key confounders, to generate local evidence for earlier detection and improved management.

METHODOLOGY

Study Design and Setting

A hospital-based, unmatched case-control study was conducted at a tertiary care facility in Southern Chennai, Tamil Nadu, India. Participants were recruited over a six-month period from the Nephrology Outpatient Department (OPD) for cases and the Medicine and General Surgery departments for controls.

Study Population and Selection

The study enrolled adults (≥ 18 years). Cases comprised patients with a documented diagnosis of CKD according to KDIGO criteria: an estimated glomerular filtration rate (eGFR) <60 mL/min/1.73 m² and/or evidence of kidney damage persisting for ≥ 3 months. Chronicity was verified via medical records or nephrologist documentation. Controls were selected from patient attenders, individuals accompanying patients but not seeking treatment themselves, to ensure they shared the same geographic and socioeconomic background as the cases. Controls were required to have an eGFR ≥ 60 mL/min/1.73 m² and no history of renal disease.

Both groups excluded individuals with acute systemic illness, pregnancy/postpartum status (≤ 6 months), known pituitary/hypothalamic disease, thyroid

malignancy, or use of medications significantly affecting thyroid function.

The sample size was estimated based on the association strength (AUC 0.914) reported in the Southern Chennai cohort by Elancheran et al. (2020).¹⁶ Using a baseline prevalence of 10% (Kaur et al., 2025)¹⁷ and an expected Odds Ratio of 2.5, the minimum required sample size per group was 138, according to Schlesselman's formula. To enhance statistical reliability, 150 cases and 150 controls were enrolled, for a total of 300 participants.

Data Collection and Laboratory Protocol

After obtaining written informed consent, we collected clinical data through structured interviews and medical record reviews. We recorded essential metrics, including BMI, blood pressure, and comorbidities such as diabetes and hypertension. Laboratory assessments were performed under standardised conditions: serum creatinine was measured via the enzymatic method to calculate eGFR using the CKD-EPI equation, and thyroid function was assessed using a standardised immunoassay platform.

Statistical Analysis and Ethics:

Continuous variables were compared using t-tests or Mann-Whitney U tests, while categorical data were analysed via Chi-square or Fisher's exact tests. Our analysis employed multivariable logistic regression to adjust for potential confounders, including age, sex, and metabolic status. Significance was set at $p < 0.05$. The protocol was approved by the Institutional Ethics Committee, and we ensured that any participant identified with new health abnormalities during screening was promptly referred for clinical care.

RESULTS

Table 1 highlights key demographic and clinical differences between patients with CKD and non-CKD controls. CKD patients are significantly older and exhibit a higher prevalence of diabetes and hypertension, both of which are major risk factors for CKD. Additionally, CKD patients show impaired renal function, reflected by elevated serum creatinine and reduced eGFR, alongside thyroid dysfunction, as indicated by higher TSH and lower FT4 levels compared to controls.

Table 2 presents the prevalence of thyroid dysfunction in both groups. CKD patients show a significantly higher rate of thyroid abnormalities, with 16.7% having subclinical hypothyroidism and 15.3% overt hypothyroidism. In contrast, controls have a higher proportion of euthyroid individuals (86%), suggesting that thyroid dysfunction is more prevalent in the CKD population and warrants regular screening.

Table 3 identifies factors independently associated with CKD. Hypothyroidism is strongly associated with CKD (OR: 3.87), as are age, diabetes, hypertension, and BMI. These findings underscore the role of thyroid dysfunction as a significant comorbidity in CKD patients and highlight other major risk factors for CKD progression.

Figure 1 visually supports the data in Table 2, showing a higher prevalence of thyroid dysfunction in CKD patients, particularly subclinical and overt hypothyroidism. This reinforces the need for targeted thyroid screening in patients with CKD to detect and manage thyroid abnormalities early.

Figure 2 shows the multivariable logistic regression results, presenting adjusted odds ratios for factors independently associated with CKD (age, BMI, diabetes, hypertension, and hypothyroidism).

DISCUSSION

This study evaluated the prevalence and pattern of thyroid dysfunction in chronic kidney disease (CKD) patients in Southern Chennai, revealing a significantly higher prevalence of thyroid abnormalities, particularly subclinical and overt hypothyroidism. These findings are consistent with recent studies indicating that thyroid dysfunction is common in CKD patients, with prevalence rates increasing as CKD severity worsens.^{18,19}

Thyroid dysfunction, especially low T3 syndrome, is frequently observed in CKD and is associated with renal impairment. The mechanisms underlying this include impaired peripheral conversion of thyroxine (T4) to triiodothyronine (T3), dysregulation of the hypothalamic-pituitary-thyroid axis, and altered renal iodine clearance.²⁰ These mechanisms align with prior research suggesting that thyroid abnormalities, particularly low T3 syndrome, are common in advanced stages of CKD.^{4,21}

Our study adds to the growing body of evidence linking thyroid dysfunction to increased cardiovascular risk in patients with CKD. Subclinical hypothyroidism has been associated with dyslipidaemia and endothelial dysfunction, which are known to contribute to the elevated cardiovascular morbidity and mortality observed in CKD populations.²²⁻²⁴ This highlights the importance of thyroid screening in CKD management to help identify at-risk patients and reduce cardiovascular complications.²⁵

The need for standardized diagnostic protocols for thyroid dysfunction in CKD is evident. While TSH, free T3, and free T4 assays are commonly used, variability in diagnostic thresholds and test methodologies complicates comparisons across studies and clinical settings.²⁶⁻²⁹ A more consistent approach would improve diagnostic accuracy and enhance the effectiveness of early intervention strategies.

Several limitations of this study should be considered. This hospital-based, single-centre case-control study used single-time-point thyroid measurements, which limits causal inference and may reduce generalisability beyond the study setting. Selection bias and residual confounding are possible, particularly from unmeasured factors such as comorbidity severity, medication use, inflammatory status, and nutritional parameters. In addition, thyroid autoantibodies and iodine status were not assessed, limiting aetiological characterisation of hypothyroidism in this cohort.

CONCLUSION

This study highlights the high prevalence of thyroid dysfunction, particularly subclinical hypothyroidism, in CKD patients in Southern Chennai. The association between thyroid dysfunction and CKD progression, as well as the increased cardiovascular risk in affected patients, underscores the importance of routine thyroid screening in CKD management. Our findings contribute to the growing body of evidence that thyroid abnormalities should be routinely monitored in patients with CKD to mitigate cardiovascular risk and improve long-term patient outcomes. However, further research, particularly longitudinal and interventional studies, is needed to clarify the causal relationship between thyroid dysfunction and CKD progression and to guide clinical decision-making regarding thyroid hormone replacement therapy.

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Conflicts of Interest:

The authors declare no conflicts of interest.

Table 1. Baseline Characteristics of Study Participants

Variable	CKD cases (n = 150)	Controls (n = 150)	p-value
Age, years (mean ± SD)	55.2 ± 12.1	48.6 ± 11.3	<0.001*
Male sex, n (%)	93 (62.0)	84 (56.0)	0.29
BMI, kg/m ² (mean ± SD)	24.8 ± 3.9	24.2 ± 3.7	0.18

Diabetes mellitus, n (%)	83 (55.3)	42 (28.0)	<0.001*
Hypertension, n (%)	108 (72.0)	51 (34.0)	<0.001*
Smoking, n (%)	29 (19.3)	21 (14.0)	0.22
Serum creatinine, mg/dL (median, IQR)	2.18 (1.52–3.84)	0.92 (0.76–1.10)	<0.001*
eGFR, mL/min/1.73 m ² (mean ± SD)	36.4 ± 15.2	92.8 ± 14.1	<0.001*
TSH, mIU/L (median, IQR)	3.9 (2.1–7.8)	2.3 (1.6–3.4)	<0.001*
FT4, ng/dL (mean ± SD)	1.02 ± 0.24	1.15 ± 0.18	<0.001*
*Statistically significant			

Table 2. Distribution of Thyroid Status Among CKD Cases and Controls

Thyroid status	CKD cases (n = 150), n (%)	Controls (n = 150), n (%)	p-value
Euthyroid	94 (62.7)	129 (86.0)	<0.001
Subclinical hypothyroidism	25 (16.7)	11 (7.3)	
Overt hypothyroidism	23 (15.3)	3 (2.0)	
Treated hypothyroidism (controlled)	8 (5.3)	7 (4.7)	

Overall, hypothyroidism (subclinical + overt + treated) was significantly more prevalent among CKD cases compared to controls.

Table 3. Multivariable Logistic Regression Analysis of Factors Associated With CKD

Variable	Adjusted OR	95% Confidence Interval	p-value
Hypothyroidism (any)	3.87	2.01–7.45	<0.001*
Age (per year increase)	1.04	1.01–1.06	0.006*
Male sex	0.93	0.53–1.65	0.81
Diabetes mellitus	2.99	1.70–5.26	<0.001*
Hypertension	5.71	3.28–9.95	<0.001*
BMI (per kg/m ² increase)	1.10	1.01–1.19	0.02*
*Statistically significant			

Figure 1: Distribution of thyroid status among CKD cases and non-CKD controls

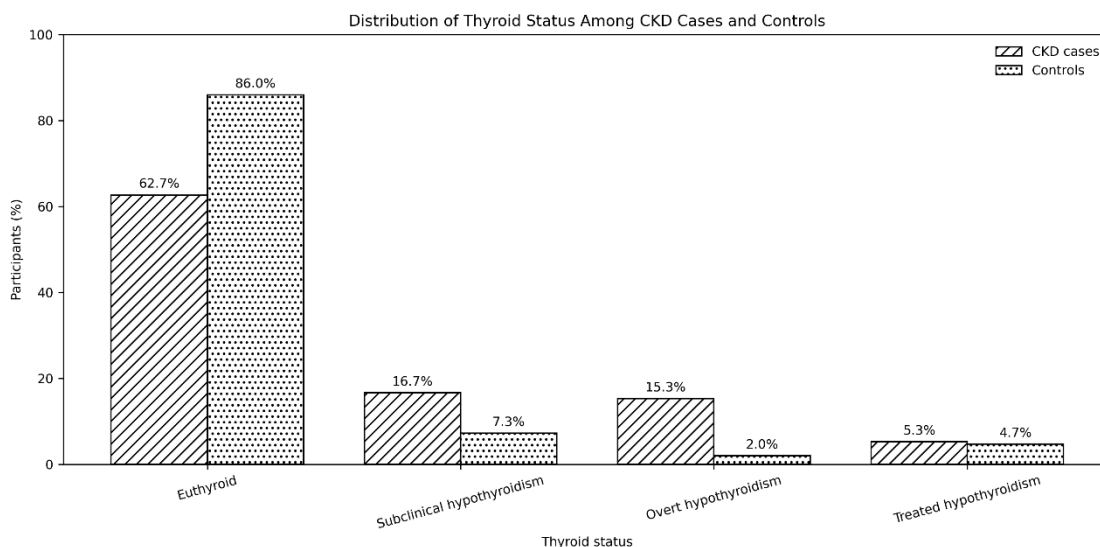
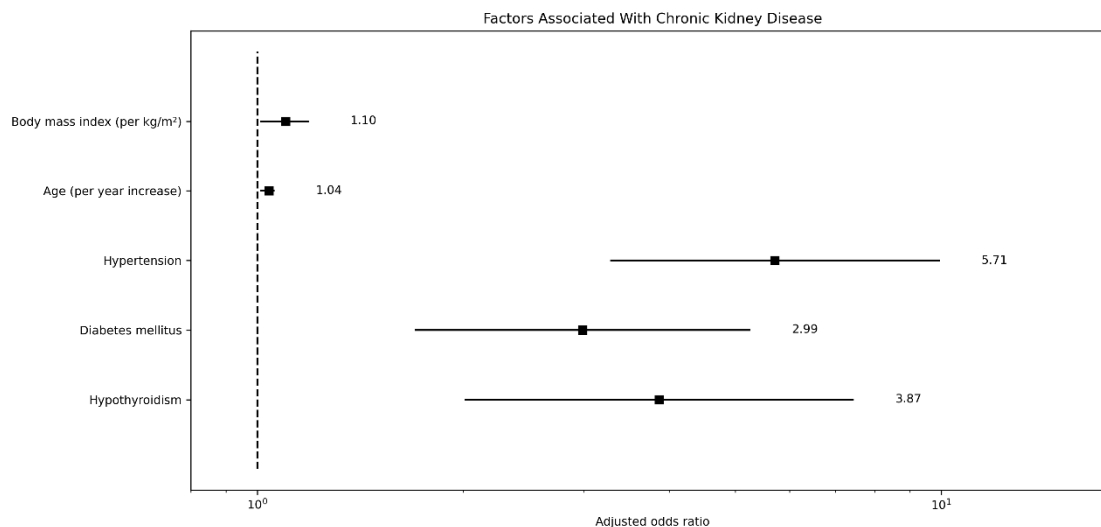


Figure 2: Factors Independently Associated with Chronic Kidney Disease



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