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Original Research Article

ASSOCIATION OF THYROID DYSFUNCTION WITH SEVERITY OF CHRONIC KIDNEY DISEASE: A CROSS-SECTIONAL STUDY AT A TERTIARY CARE CENTER

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ABSTRACT

Background: Chronic kidney disease (CKD) is associated with disturbances in thyroid hormone metabolism that may become more pronounced with progressive renal impairment. This study evaluated the association between thyroid dysfunction and severity of CKD.

Materials and Methods: This observational cross-sectional study included 150 adult CKD patients admitted to a tertiary care center. CKD was classified into stages 3, 4, and 5 according to estimated glomerular filtration rate. Serum triiodothyronine (T3), thyroxine (T4), and thyroid-stimulating hormone (TSH) were assessed, and thyroid status was categorized. Associations between CKD stage and thyroid dysfunction were evaluated statistically.

Results: Stage 3, 4, and 5 CKD accounted for 31.3%, 30.7%, and 38.0% of patients, respectively. Euthyroid status progressively declined from 59.6% in stage 3 to 34.8% in stage 4 and 24.6% in stage 5. Low T3 syndrome increased from 21.3% to 30.4% and 38.6%, respectively. CKD stage was significantly associated with thyroid dysfunction (χ^2=18.942, p=0.003).

Conclusion: Thyroid dysfunction increased with advancing CKD severity, particularly low T3 and hypothyroid patterns.

Keywords: Chronic kidney disease; CKD severity; Thyroid dysfunction; Low T3 syndrome; Hypothyroidism; eGFR.

INTRODUCTION

Chronic kidney disease (CKD) is a progressive disorder characterized by persistent structural or functional abnormalities of the kidneys lasting for at least three months. It represents an important global health problem because of its increasing prevalence, substantial cardiovascular burden, progression to end-stage renal disease, and associated mortality.[1-3] In India, the burden of CKD continues to increase, driven predominantly by diabetes mellitus, hypertension, chronic glomerular diseases, nephrotoxic exposures, and delayed diagnosis.[4] The consequences of CKD extend beyond progressive loss of renal filtration, as the disease produces widespread metabolic, cardiovascular, hematological, and endocrine disturbances.

The systemic effects of CKD become increasingly prominent as renal function deteriorates. Retention of uremic toxins, chronic inflammation, oxidative stress, metabolic acidosis, anemia, disturbances of mineral metabolism, and altered neurohormonal regulation contribute to multisystem dysfunction.[5] Endocrine abnormalities are particularly relevant because the kidney participates in the metabolism and regulation of several hormones. Among these, alterations in the hypothalamic-pituitary-thyroid axis are frequently observed in patients with impaired renal function.

The kidney plays an important role in thyroid hormone metabolism, degradation, and excretion. Progressive renal impairment may reduce peripheral conversion of thyroxine (T4) to the biologically active triiodothyronine (T3) because of decreased activity of type I 5′-deiodinase. Uremic toxins and metabolic acidosis can modify thyroid hormone binding to carrier proteins, while impaired iodine clearance and altered hypothalamic-pituitary responsiveness may further influence circulating thyroid hormone concentrations.[6,7] These

mechanisms create a complex biochemical pattern in which abnormalities of thyroid function may become increasingly apparent as CKD progresses.

Low T3 syndrome is among the most frequently reported thyroid abnormalities in CKD. It is generally characterized by reduced circulating T3 with relatively preserved T4 and normal or mildly altered TSH. Reduced T3 may represent an adaptive response to chronic systemic illness; however, increasing evidence suggests that low T3 concentrations are associated with impaired renal function and unfavorable clinical outcomes.[6,8] Subclinical and overt hypothyroidism are also reported more frequently among CKD patients than in the general population.

Importantly, the prevalence and spectrum of thyroid abnormalities appear to vary according to the severity of renal dysfunction. Pan et al. demonstrated progressive reductions in T3 and FT3 concentrations across advancing CKD stages and reported a high prevalence of euthyroid sick syndrome in stage 5 CKD.[8] Similarly, Khatiwada et al. observed a significantly greater risk of thyroid dysfunction among patients with stages 4 and 5 CKD compared with those with stage 3 disease.[9] These findings suggest that deterioration of renal function may be accompanied by progressive disruption of thyroid hormone homeostasis.

The relationship may also have clinical implications beyond biochemical abnormalities. Reduced thyroid hormone concentrations have been associated with cardiovascular risk, adverse renal outcomes, and mortality in CKD populations.[6,8] Nevertheless, interpretation remains challenging because manifestations such as fatigue, weakness, fluid retention, and metabolic disturbances may be attributable to either CKD or thyroid dysfunction.

Despite increasing evidence of an interaction between renal function and thyroid homeostasis, data examining thyroid abnormalities across different CKD stages in Indian populations remain limited. Characterizing this relationship may help identify patients more likely to exhibit thyroid dysfunction as renal impairment progresses.

Therefore, the present study was undertaken to evaluate the association between thyroid dysfunction and severity of chronic kidney disease, with particular emphasis on the distribution of euthyroid status, low T3 syndrome, and hypothyroid states across CKD stages 3, 4, and 5.

MATERIALS AND METHODS

Study Design and Setting: This observational cross-sectional study was conducted in the Department of General Medicine at Kalpana Chawla Government Medical College and Hospital, Karnal. The total study duration was 18 months, from March 2024 to September 2025, including one year of data collection followed by six months for data compilation and analysis. The study population comprised patients admitted to the General Medicine inpatient department with chronic kidney disease (CKD).

Diagnosis and Classification of CKD: CKD was diagnosed according to Kidney Disease: Improving Global Outcomes (KDIGO) criteria. Diagnosis was confirmed within 72 hours of hospital admission using renal function tests and ultrasonography of the kidneys, ureters, and bladder (USG KUB). Thyroid hormone levels were assessed within 24 hours after confirmation of CKD.

The estimated glomerular filtration rate (eGFR) was calculated using the Modification of Diet in Renal Disease (MDRD) equation:

eGFR (mL/min/1.73 m^2) = 1.86 × (serum creatinine)^−1.154 × (age)^−0.203

The calculated value was multiplied by 0.742 for female patients. Participants were categorized as stage 3 (eGFR 30–59 mL/min/1.73 m^2), stage 4 (15–29 mL/min/1.73 m^2), or stage 5 (<15 mL/min/1.73 m^2).

Sample Size and Participant Selection: A total of 150 patients were included. The minimum sample size was calculated using Cochran's formula at a 95% confidence level. A prevalence of thyroid dysfunction in CKD of 38.6% from a previous study and a maximum allowable error of 8% were used for the calculation.

Patients aged ≥18 years with CKD of at least three months' duration, uremic symptoms, elevated blood urea and serum creatinine, and ultrasonographic evidence of chronic renal failure were eligible. Patients with acute kidney injury, known thyroid disease, hypothalamic or pituitary disorders, malignancy, pregnancy, acute medical illness requiring intensive care, or use of drugs known to alter thyroid function were excluded.

Clinical and Laboratory Assessment: After obtaining written informed consent, demographic and clinical information was recorded using a predesigned structured proforma. Detailed history and physical examination were performed, with particular attention to renal and thyroid-related manifestations. Laboratory investigations included complete blood count, blood urea, serum creatinine, serum sodium and potassium, fasting blood glucose, urine routine examination, and thyroid profile. USG KUB was performed to evaluate kidney size and corticomedullary differentiation.

Serum creatinine was measured using a Cobas 6000 analyzer by the Jaffe end-point method. Fasting venous blood samples were collected for thyroid assessment, and serum T3, T4, and TSH concentrations were measured using the enzyme-linked immunosorbent assay method.

The reference ranges were 0.6–2.1 ng/mL for total T3, 5–13 µg/dL for total T4, and 0.4–4.5 µIU/mL for TSH. Subclinical hypothyroidism was defined as TSH 4.6–9.9 mIU/L with normal T3 and T4 levels, whereas overt hypothyroidism was defined as TSH ≥10 mIU/L.

Statistical Analysis: Data were entered into Microsoft Excel and analyzed using SPSS version 29.0.0 (IBM Corp., USA). Normality of continuous variables was evaluated using the Kolmogorov–Smirnov test. Quantitative variables were expressed as mean ± standard deviation, and categorical variables as frequencies and percentages. The Chi-square test was used to evaluate associations between categorical variables. Pearson's or Spearman's correlation coefficient was specified for assessment of relationships between thyroid and renal parameters, as appropriate. A p-value <0.05 was considered statistically significant, while p<0.01 was considered highly significant.

RESULTS

Table 1: Distribution of participants according to CKD stage (N=150)

CKD stage	eGFR (mL/min/1.73 m²)	n	%
Stage 3	30–59	47	31.3
Stage 4	15–29	46	30.7
Stage 5	<15	57	38.0
Total	—	150	100.0

Table 2: Thyroid function status among CKD patients (N=150)

Thyroid status	n	%
Euthyroid	58	38.7
Low T3 syndrome	46	30.7
Subclinical hypothyroidism	32	21.3
Overt hypothyroidism	10	6.7
Subclinical hyperthyroidism	3	2.0
Overt hyperthyroidism	1	0.6
Total	150	100.0

Table 3: Mean thyroid hormone levels among CKD patients (N=150)

Parameter	Mean ± SD
T3 (ng/mL)	0.71 ± 0.22
T4 (µg/dL)	7.9 ± 1.8
TSH (µIU/mL)	6.42 ± 3.9

Table 4: Association between CKD stage and thyroid dysfunction (N=150)

CKD stage	Euthyroid n (%)	Low T3 n (%)	Subclinical hypothyroidism n (%)	Overt hypothyroidism n (%)	χ^2	p-value
Stage 3 (n=47)	28 (59.6)	10 (21.3)	7 (14.9)	2 (4.2)	18.942	0.003
Stage 4 (n=46)	16 (34.8)	14 (30.4)	12 (26.1)	4 (8.7)		
Stage 5 (n=57)	14 (24.6)	22 (38.6)	13 (22.8)	4 (7.0)		

Table 5: Association of selected sociodemographic factors with CKD stage

Variable	Key distribution	χ^2	p-value
Age group	Stage 5 increased to 57.2% among >60 years	12.84	0.012
Gender	Stage 5: males 39.5%; females 35.2%	0.82	0.660
Residence	Stage 5: rural 45.5%; urban 27.4%	6.94	0.031
Educational status	Stage 5: illiterate 55.5%	14.62	0.006
Socioeconomic status	Stage 5: lower class 50.0%	13.91	0.008

DISCUSSION

The present study demonstrated a significant association between the severity of chronic kidney disease and thyroid dysfunction. As renal impairment progressed from stage 3 to stage 5, the proportion of euthyroid patients decreased from 59.6% to 24.6%, whereas low T3 syndrome increased from 21.3% to 38.6%. Subclinical and overt hypothyroid patterns were also more frequent in advanced CKD than in stage 3 disease. The overall association between CKD stage and thyroid dysfunction was statistically significant (χ^2=18.942, p=0.003).

The progressive increase in low T3 syndrome with worsening CKD is consistent with previous evidence. Pan et al. demonstrated that T3 and FT3 concentrations declined significantly with advancing CKD and were lowest in stage 5 disease. They reported euthyroid sick syndrome in 69.1% of stage 5 patients and identified a positive relationship between eGFR and T3/FT3.[8] These observations support the pattern identified in the present study, in which low T3 increased progressively across CKD stages.

Khatiwada et al. similarly found that patients with stages 4 and 5 CKD had a significantly greater risk of thyroid dysfunction than those with stage 3 disease.[9] Thyroid dysfunction was identified in 38.6% of their cohort, with subclinical hypothyroidism being the most frequent abnormality. The progressive endocrine disturbance associated with worsening renal function may reflect reduced peripheral conversion of T4 to T3, altered hormone metabolism and clearance, chronic inflammation, and the effects of the uremic milieu.

The hypothyroid pattern observed with advanced CKD is also supported by Sinjari et al., who reported thyroid abnormalities in 32.7% of CKD patients. Subclinical hypothyroidism was the most frequent abnormality (21.2%), followed by overt hypothyroidism (6.7%). Importantly, hypothyroidism was significantly associated with low eGFR and hemodialysis status.[10] These observations are consistent with the greater burden of hypothyroid states seen in stages 4 and 5 in the present study.

Raj et al. reported low FT3 in 87.9% of CKD patients and demonstrated a significant association between low FT3 and reduced eGFR (p=0.014).[11] They also observed significant associations of urea with low FT3, low FT4, and subclinical hypothyroidism. Although the absolute prevalence differed from our study, the direction of association supports the concept that deterioration of renal function is accompanied by increasingly abnormal thyroid hormone profiles.

The clinical relevance of this relationship has also been demonstrated in longitudinal studies. Schultheiss et al. found that low FT3 syndrome was significantly associated with reduced eGFR. Lower FT3 was subsequently associated with adverse renal outcomes and mortality, while patients with low FT3 syndrome and hypothyroidism had 2.2-fold and 1.6-fold greater risks of adverse renal events, respectively.[12] These findings suggest that thyroid abnormalities in CKD may represent more than isolated biochemical changes, although the cross-sectional design of the present study does not allow assessment of prognosis or causality.

Echterdiek et al. reported that hypothyroidism occurs approximately two to five times more frequently among CKD patients than in the general population and reviewed evidence linking thyroid abnormalities with mortality and renal deterioration.[13] More recently, Cheng et al. found that hypothyroidism was independently associated with renal failure (adjusted HR 1.29), while both hypothyroidism and hyperthyroidism were associated with increased all-cause mortality.[14] These findings further emphasize the potential clinical importance of the thyroid–kidney interaction.

The present study also found significant associations between CKD stage and age, residence, educational status, and socioeconomic status, whereas gender was not significantly associated with CKD severity. Older patients were increasingly represented in stage 5 disease, and advanced CKD was more frequent among rural and socioeconomically disadvantaged participants. These observations may reflect differences in disease burden, access to healthcare, awareness, and timing of presentation. However, because these relationships were assessed cross-sectionally, causal interpretations should be avoided.

This study has several limitations. It was conducted at a single tertiary-care center and included hospitalized CKD patients, which may limit generalizability. Its cross-sectional design precluded assessment of temporal relationships, CKD progression, mortality, or the effect of treatment of thyroid dysfunction. Furthermore, the available thesis results do not provide stage-specific mean T3, T4, and TSH values or multivariable effect estimates; therefore, these were not inferred for the present manuscript. Nevertheless, the significant stage-wise variation in thyroid status provides evidence of an increasing burden of thyroid abnormalities with worsening renal impairment.

CONCLUSION

Thyroid dysfunction showed a significant association with CKD severity. Euthyroid status progressively decreased from 59.6% in stage 3 to 24.6% in stage 5 CKD, while low T3 syndrome increased from 21.3% to 38.6%. The overall association between CKD stage and thyroid dysfunction was statistically significant (p=0.003).

These findings indicate that worsening renal impairment is accompanied by an increasing burden of thyroid abnormalities, particularly low T3 and hypothyroid patterns. Thyroid function assessment may therefore be clinically relevant in patients with advanced CKD. Longitudinal and multicenter studies are required to determine whether these abnormalities independently influence CKD progression and whether their treatment modifies renal or cardiovascular outcomes.

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