e-ISSN: 0976-822X, p-ISSN:2961-6042

Available online on http://www.ijcpr.com/

International Journal of Current Pharmaceutical Review and Research 2026; 18(1); 218-224

Original Research Article

A Study on Thyroid Profile in Chronic Kidney Disease

S. Deepika[1], S. Sujatha[2], M. Priyanka[3]

[1]Assistant Professor, Department of General Medicine, Government Medical College Hospital, Virudhunagar

[2]Assistant Professor, Department of General medicine, Government Mohan Kumaramangalam Medical College Hospital, Salem

[3]DNB General Medicine, Sri Palanimurugan Clinic, Panjukalipatty, Omalur (T.K.), Salem (D.T.)

Received: 01-10-2025 / Revised: 15-11-2025 / Accepted: 21-12-2025

Corresponding author: Dr. S. Deepika

Conflict of interest: Nil

Abstract

Background: Chronic kidney disease (CKD) is associated with significant alterations in thyroid hormone metabolism. Understanding these changes is crucial for comprehensive patient management. Aim of this study is to evaluate thyroid function abnormalities in patients with chronic kidney disease and correlate these changes with the severity of renal impairment.

Methods: A cross-sectional observational study was conducted on 100 CKD patients and 50 age and sex-matched healthy controls. Thyroid function tests including T3, T4, TSH, FT3, and FT4 were measured. Patients were categorized according to CKD stages based on estimated glomerular filtration rate (eGFR). Statistical analysis was performed using appropriate tests.

Results: The mean age of CKD patients was 52.3±12.4 years with male predominance (64%). Significantly lower levels of T3 and FT3 were observed in CKD patients compared to controls ($p < 0.001$). T4, FT4, and TSH levels showed no significant difference. The prevalence of low T3 syndrome increased with advancing CKD stages, being present in 78% of stage 5 CKD patients. A significant negative correlation was found between serum creatinine and T3 levels ($r = -0.542$, $p < 0.001$).

Conclusion: Thyroid dysfunction, particularly low T3 syndrome, is highly prevalent in CKD patients and correlates with disease severity. Regular thyroid function monitoring should be considered in CKD management.

Keywords: Chronic Kidney Disease, Thyroid Profile, Low T3 Syndrome, Thyroid Dysfunction.

DOI: 10.25258/ijcpr.18.1.35

Introduction

Chronic kidney disease (CKD) represents one of the most significant global health challenges of the 21st century, affecting approximately 10-15% of the world's population and imposing substantial burden on healthcare systems worldwide. This condition is characterized by progressive and irreversible loss of renal function, leading to accumulation of metabolic waste products, electrolyte disturbances, and widespread systemic complications.[1,2]

The rising prevalence of CKD is closely linked to the increasing incidence of diabetes mellitus and hypertension, which together account for more than two-thirds of all CKD cases globally. As the population ages and the prevalence of these risk factors continues to climb, CKD has emerged as a major public health concern requiring comprehensive multidisciplinary management approaches. The progressive deterioration of kidney function in CKD leads to a cascade of metabolic and endocrine disturbances that extend far beyond simple waste accumulation. Among these numerous complications, alterations in thyroid hormone metabolism and function represent a particularly important yet frequently underappreciated aspect of CKD management. Thyroid dysfunction in CKD patients is not merely an incidental finding but rather a complex manifestation of the profound metabolic derangements that characterize uremic syndrome. Understanding these thyroid abnormalities is crucial for clinicians, as they may significantly impact patient outcomes, quality of life, and overall disease management strategies.

The relationship between kidney disease and thyroid function is intricate, multifaceted, and bidirectional in nature. The kidneys play several essential roles in thyroid hormone homeostasis, including the metabolism and degradation of thyroid hormones, renal clearance of iodine which influences thyroid hormone synthesis, and elimination of thyroid hormone metabolites from the circulation.[3,4] The kidneys are responsible for approximately 85% of the extrathyroidal conversion of thyroxine (T4) to triiodothyronine (T3), the metabolically active form of thyroid hormone. Additionally, renal tissue contains significant concentrations of deiodinase enzymes, which are critical for this conversion process. Conversely, thyroid hormones exert important regulatory effects on kidney function, influencing renal blood flow, glomerular filtration rate, tubular secretion and reabsorption mechanisms, and overall kidney development and maturation. This complex interplay between the two organ systems means that dysfunction in one invariably affects the other.

In patients with CKD, multiple mechanisms converge to disrupt normal thyroid hormone metabolism and function. The accumulation of uremic toxins, which are incompletely cleared due to reduced kidney function, directly inhibits the activity of deiodinase enzymes responsible for peripheral conversion of T4 to T3.[5,6] These uremic toxins also interfere with thyroid hormone binding to plasma proteins, alter thyroid hormone receptor expression and function at the cellular level, and may affect the hypothalamic-pituitary-thyroid axis itself. Metabolic acidosis, a common complication in advanced CKD, further impairs the peripheral conversion of thyroid hormones and reduces tissue responsiveness to these hormones. Protein-energy malnutrition, frequently encountered in CKD patients due to dietary restrictions, uremic anorexia, and increased protein catabolism, leads to decreased synthesis of thyroid hormone binding proteins and altered hormone distribution. Furthermore, chronic inflammation, which is nearly universal in CKD, produces cytokines that suppress thyroid function through multiple pathways. The various medications commonly prescribed to CKD patients, including heparin, furosemide, dopamine, and glucocorticoids, can also interfere with thyroid function tests and thyroid hormone metabolism, adding another layer of complexity to the clinical picture.

Several distinct patterns of thyroid dysfunction have been well-documented in the CKD population, each with unique characteristics and clinical implications.

The most prevalent abnormality is the low T3 syndrome, also referred to as euthyroid sick syndrome or non-thyroidal illness syndrome. This condition is characterized by decreased serum T3 levels in the presence of normal or low-normal T4 and normal or slightly elevated reverse T3 (rT3) levels, with thyroid-stimulating hormone (TSH) typically remaining within the normal range.[7,8] This pattern reflects impaired peripheral conversion of T4 to T3 and enhanced conversion to the inactive reverse T3. The term "euthyroid" in this context is somewhat misleading, as patients may exhibit clinical signs and symptoms suggestive of hypothyroidism despite having normal TSH levels. Subclinical hypothyroidism, defined by elevated TSH with normal free T4 levels, is also more prevalent in CKD patients compared to the general population. Overt hypothyroidism, characterized by elevated TSH and decreased free T4 levels, occurs less frequently but carries significant clinical implications when present. Interestingly, hyperthyroidism appears to be relatively uncommon in CKD patients, though when present, it may be masked by the general tendency toward low thyroid hormone levels in uremia.

Epidemiological studies have demonstrated that the prevalence and severity of thyroid abnormalities in CKD patients show a strong correlation with the degree of renal impairment.[9,10] Patients with early-stage CKD may exhibit minimal or no thyroid dysfunction, while those with advanced disease, particularly stages 4 and 5, demonstrate marked abnormalities in thyroid function tests. This stage-dependent relationship suggests that the accumulation of uremic toxins and the progressive metabolic derangements of advancing kidney disease directly contribute to thyroid dysfunction. Some studies have reported that up to 70-80% of patients with end-stage renal disease exhibit some form of thyroid abnormality, highlighting the magnitude of this problem in the dialysis population.

The clinical significance of thyroid dysfunction in CKD patients extends beyond simple laboratory abnormalities. Emerging evidence suggests that alterations in thyroid function may be associated with important clinical outcomes in this population. Low T3 levels have been linked to increased all-cause mortality, cardiovascular events, and accelerated progression of kidney disease in several observational studies.[11,12] Patients with low T3 syndrome may experience fatigue, decreased exercise capacity, impaired quality of life, altered lipid metabolism, and cardiovascular dysfunction. The question of whether low T3 syndrome represents an adaptive response designed to conserve energy and reduce protein catabolism in chronic illness, or a maladaptive state contributing to adverse outcomes, remains one of the most debated issues in this field. This distinction is crucial because it determines whether therapeutic intervention with thyroid hormone replacement is beneficial or potentially harmful.

Despite the well-recognized association between CKD and thyroid dysfunction, several important clinical questions remain unanswered or controversial. First, there is ongoing debate regarding the appropriate reference ranges for thyroid function tests in CKD patients. Standard reference ranges derived from healthy populations may not be applicable to uremic patients, potentially leading to misdiagnosis and inappropriate treatment.

Second, the distinction between low T3 syndrome and true hypothyroidism requiring treatment is not always clear-cut, particularly in patients with mixed patterns of thyroid abnormalities. Third, the role of thyroid hormone replacement therapy in CKD patients with low T3 syndrome remains uncertain, as randomized controlled trials investigating this intervention are limited and have yielded conflicting results.

Fourth, the impact of renal replacement therapy, including hemodialysis, peritoneal dialysis, and kidney transplantation, on thyroid function abnormalities is incompletely understood. Some evidence suggests that successful kidney transplantation may partially reverse thyroid dysfunction, while dialysis appears to have limited effect on normalizing thyroid hormone levels.

The management of CKD patients requires a comprehensive approach that addresses not only renal function but also the myriad complications arising from uremia. Given the high prevalence of thyroid dysfunction in this population and its potential impact on clinical outcomes, systematic evaluation of thyroid function should be an integral component of CKD care. However, current clinical practice guidelines provide limited specific recommendations regarding thyroid function screening and management in CKD patients, reflecting the gaps in our understanding of this complex issue. There is a clear need for well-designed studies to establish evidence-based approaches to thyroid assessment and treatment in the CKD population.

Furthermore, the interaction between thyroid dysfunction and other comorbidities common in CKD patients, such as cardiovascular disease, anemia, mineral bone disorder, and malnutrition, adds additional layers of complexity to patient management.

Thyroid hormones influence cardiac function, vascular tone, lipid metabolism, and bone turnover, all of which are already compromised in CKD. T

he potential synergistic effects of thyroid dysfunction and CKD-related complications on cardiovascular outcomes and mortality require further investigation to optimize therapeutic strategies.

Aim of this study is to evaluate thyroid function abnormalities in patients with chronic kidney disease and correlate these changes with the severity of renal impairment.

Methodology

This cross-sectional observational study was conducted in the Department of Medicine at a tertiary care hospital from January 2021 to June 2022. The study protocol received approval from the Institutional Ethics Committee, and written informed consent was obtained from all participants.

The study enrolled 100 patients with chronic kidney disease and 50 age and sex-matched healthy controls. CKD was diagnosed based on KDIGO criteria. Patients aged 18-75 years with confirmed CKD stages 2-5 were included. Exclusion criteria comprised patients with pre-existing thyroid disorders, those on thyroid medications, acute kidney injury, dialysis duration less than 3 months, pregnant or lactating women, severe systemic illnesses, and patients on medications affecting thyroid function (amiodarone, lithium, corticosteroids).

Patients were classified into CKD stages based on estimated glomerular filtration rate (eGFR) calculated using the MDRD equation: Stage 2 (eGFR 60-89 mL/min/1.73m²), Stage 3 (eGFR 30-59 mL/min/1.73m²), Stage 4 (eGFR 15-29 mL/min/1.73m²), and Stage 5 (eGFR <15 mL/min/1.73m²). Detailed clinical history and physical examination were performed for all participants, with emphasis on symptoms of thyroid dysfunction including fatigue, weight changes, cold intolerance, and bowel habit alterations.

Blood samples were collected after 8-12 hours overnight fasting. Thyroid function tests including total T3, T4, free T3, free T4, and TSH were measured using chemiluminescence immunoassay.

Normal reference ranges were T3: 80-200 ng/dL, T4: 5-12 μg/dL, FT3: 2.3-4.2 pg/mL, FT4: 0.8-2.0 ng/dL, and TSH: 0.5-5.0 μIU/mL. Low T3 syndrome was defined as T3 or FT3 below lower limit with normal T4 and TSH. Subclinical hypothyroidism was defined as elevated TSH (>5.0 μIU/mL) with normal FT4, while overt hypothyroidism as elevated TSH with decreased FT4.

Renal function parameters including serum creatinine and blood urea nitrogen were measured using standard enzymatic methods. Additional investigations included complete blood count, serum electrolytes, lipid profile, and blood glucose levels.

Statistical analysis was performed using SPSS version 23.0. Continuous variables were expressed as mean ± standard deviation and categorical variables as frequencies and percentages. Student's t-test was used for comparing means between two groups, while one-way ANOVA with post-hoc Tukey's test was employed for multiple group comparisons. Pearson's correlation coefficient assessed relationships between thyroid parameters and renal function markers.

Chi-square or Fisher's exact test was used for categorical variables. A p-value <0.05 was considered statistically significant.

Results

Demographic and Clinical Characteristics: A total of 100 CKD patients and 50 healthy controls were enrolled in the study.

The demographic profile of the study population is presented in Table 1.

Table 1: Demographic and Clinical Characteristics of Study Population

Parameter	CKD Patients (n=100)	Controls (n=50)	p-value
Age (years), mean ± SD	52.3 ± 12.4	50.8 ± 11.2	0.485
Male gender, n (%)	64 (64%)	31 (62%)	0.802
BMI (kg/m²), mean ± SD	23.8 ± 3.6	24.2 ± 3.2	0.512
Diabetes mellitus, n (%)	48 (48%)	0 (0%)	<0.001
Hypertension, n (%)	72 (72%)	0 (0%)	<0.001
Duration of CKD (months)	28.6 ± 18.4	-	-

The mean age of CKD patients was 52.3 ± 12.4 years with a male predominance (64%). The most common etiologies of CKD were diabetic nephropathy (48%) and hypertensive nephrosclerosis (36%), followed by chronic glomerulonephritis (10%) and other causes (6%).

Distribution of CKD Stages: The distribution of patients across different CKD stages is shown in Table 2.

Table 2: Distribution of Patients According to CKD Stages

CKD Stage	eGFR (mL/min/1.73m²)	Number of Patients	Percentage
Stage 2	60-89	12	12%
Stage 3	30-59	28	28%
Stage 4	15-29	32	32%
Stage 5	<15	28	28%

The majority of patients were in stages 4 and 5 (60%), reflecting the advanced nature of kidney disease in our study population.

Thyroid Function Parameters: Comparison of thyroid function parameters between CKD patients and healthy controls revealed significant differences (Table 3).

Table 3: Comparison of Thyroid Function Parameters between CKD Patients and Controls

Parameter	CKD Patients (n=100) Mean ± SD	Controls (n=50) Mean ± SD	p-value
T3 (ng/dL)	68.4 ± 22.8	118.6 ± 18.4	<0.001*
T4 (μg/dL)	7.8 ± 2.1	8.2 ± 1.6	0.248
FT3 (pg/mL)	2.1 ± 0.6	3.2 ± 0.5	<0.001*
FT4 (ng/dL)	1.2 ± 0.4	1.3 ± 0.3	0.156
TSH (μIU/mL)	3.8 ± 2.4	3.2 ± 1.8	0.128

CKD patients demonstrated significantly lower levels of T3 and FT3 compared to controls ($p < 0.001$), while T4, FT4, and TSH levels showed no statistically significant differences. The mean T3 level in CKD patients was 68.4 ± 22.8 ng/dL compared to 118.6 ± 18.4 ng/dL in controls.

Thyroid Parameters Across CKD Stages: Analysis of thyroid function parameters across different stages of CKD revealed a progressive decline in T3 and FT3 levels with advancing disease severity (Table 4).

Table 4: Thyroid Function Parameters According to CKD Stages

Parameter	Stage 2 (n=12)	Stage 3 (n=28)	Stage 4 (n=32)	Stage 5 (n=28)	p-value
T3 (ng/dL)	92.4 ± 18.6	78.2 ± 16.4	64.8 ± 18.2	48.6 ± 20.4	<0.001*
T4 (μg/dL)	8.4 ± 1.8	7.9 ± 2.0	7.6 ± 2.2	7.4 ± 2.4	0.412
FT3 (pg/mL)	2.8 ± 0.5	2.4 ± 0.5	1.9 ± 0.6	1.6 ± 0.5	<0.001*
FT4 (ng/dL)	1.4 ± 0.3	1.3 ± 0.4	1.2 ± 0.4	1.1 ± 0.5	0.168
TSH (μIU/mL)	3.2 ± 1.8	3.6 ± 2.2	3.9 ± 2.4	4.2 ± 2.8	0.524
Serum Creatinine (mg/dL)	1.8 ± 0.3	2.8 ± 0.6	4.6 ± 1.2	8.4 ± 2.6	<0.001*

There was a statistically significant progressive decrease in T3 and FT3 levels from stage 2 to stage 5 CKD ($p < 0.001$). Post-hoc analysis revealed significant differences between stage 5 and stages 2 and 3 for both T3 and FT3 levels.

Prevalence of Thyroid Dysfunction: The prevalence of various thyroid dysfunction patterns in the CKD population is presented in Table 5.

Table 5: Prevalence of Thyroid Dysfunction in CKD Patients

Type of Dysfunction	Overall (n=100)	Stage 2 (n=12)	Stage 3 (n=28)	Stage 4 (n=32)	Stage 5 (n=28)
Low T3 Syndrome	62 (62%)	2 (17%)	12 (43%)	26 (81%)	22 (78%)
Subclinical Hypothyroidism	8 (8%)	0 (0%)	2 (7%)	3 (9%)	3 (11%)
Primary Hypothyroidism	4 (4%)	0 (0%)	1 (4%)	1 (3%)	2 (7%)
Normal Thyroid Function	26 (26%)	10 (83%)	13 (46%)	2 (6%)	1 (4%)

Low T3 syndrome was the most common thyroid abnormality, present in 62% of CKD patients overall. The prevalence increased significantly with advancing CKD stages, affecting 78% of stage 5 patients compared to only 17% of stage 2 patients. Subclinical hypothyroidism was present in 8% and overt hypothyroidism in 4% of patients.

Correlation Analysis: Pearson's correlation analysis revealed significant relationships between thyroid parameters and markers of renal function. A strong negative correlation was observed between serum creatinine and T3 levels ($r = -0.542$, $p < 0.001$) and between serum creatinine and FT3 levels ($r = -0.486$, $p < 0.001$). Similarly, eGFR showed positive correlation with T3 ($r = 0.524$, $p < 0.001$) and FT3 levels ($r = 0.468$, $p < 0.001$). No significant correlation was found between TSH and markers of renal function.

Discussion

This study provides comprehensive data on thyroid function abnormalities in chronic kidney disease patients and demonstrates a clear relationship between the severity of renal impairment and the degree of thyroid dysfunction. Our findings are consistent with previous studies documenting significant alterations in thyroid hormone metabolism in the CKD population.[13,14]

Thyroid Dysfunction in CKD: The most striking finding in our study was the high prevalence of low T3 syndrome, affecting 62% of CKD patients overall and increasing to 78% in stage 5 CKD. This pattern, characterized by decreased T3 levels with relatively preserved T4 and TSH levels, represents the most common thyroid abnormality in CKD and reflects impaired peripheral conversion of T4 to T3.[15,16]

The significantly lower mean T3 (68.4 ± 22.8 ng/dL) and FT3 (2.1 ± 0.6 pg/mL) levels in CKD patients compared to controls ($p < 0.001$) underscore the magnitude of this dysfunction. The pathophysiology of low T3 syndrome in CKD is multifactorial. Uremic toxins accumulate in CKD and inhibit the activity of type 1 deiodinase, the enzyme responsible for peripheral conversion of T4 to T3.[17] Additionally, metabolic acidosis, commonly present in advanced CKD, further impairs this conversion. Protein-energy malnutrition, frequently observed in CKD patients, contributes to decreased thyroid hormone binding proteins and altered hormone metabolism.[18]

Stage-wise Progression: Our study demonstrated a clear progressive decline in T3 and FT3 levels correlating with worsening renal function, with the most pronounced changes observed in stages 4 and 5 CKD. This finding is clinically significant as it suggests that thyroid dysfunction is not merely an epiphenomenon but is intrinsically linked to the severity of uremia. The strong negative correlation between serum creatinine and T3 levels ($r = -0.542$, $p < 0.001$) and the positive correlation between eGFR and T3 levels ($r = 0.524$, $p < 0.001$) support this relationship.

These correlations align with studies showing that accumulation of uremic toxins and the degree of renal impairment directly influence thyroid hormone metabolism.[19] The progressive nature of these changes suggests that regular monitoring of thyroid function should be integrated into the routine follow-up of CKD patients, particularly those with advancing disease.

Clinical Implications of Low T3 Syndrome: The clinical significance of low T3 syndrome in CKD remains a subject of debate. While some researchers consider it an adaptive response to conserve energy in chronic illness, emerging evidence suggests that low T3 levels may be associated with adverse outcomes in CKD patients.[20] Studies have reported associations between low T3 syndrome and increased mortality, cardiovascular events, and accelerated CKD progression. However, whether thyroid hormone replacement therapy in these patients improves outcomes remains controversial and requires further investigation through randomized controlled trials.

In our study, 8% of patients had subclinical hypothyroidism and 4% had overt hypothyroidism. These figures are slightly higher than the general population, suggesting that CKD patients may be at increased risk for developing true thyroid disease in addition to non-thyroidal illness syndrome. The distinction between low T3 syndrome and true hypothyroidism is clinically important as the latter requires thyroid hormone replacement therapy.

Preserved TSH Levels: An interesting finding in our study was the relatively preserved TSH levels across all CKD stages, with no significant difference between patients and controls. This observation is consistent with the concept of low T3 syndrome, where the hypothalamic-pituitary axis remains intact and TSH levels stay within the normal range despite peripheral thyroid hormone abnormalities.[21] The preservation of TSH levels helps distinguish low T3 syndrome from primary hypothyroidism and suggests that the thyroid gland function per se is not primarily affected in most CKD patients.

Comparison with Previous Studies: Our findings are concordant with several previous investigations. A study by Kaptein documented similar patterns of thyroid dysfunction in CKD patients, with low T3 syndrome being the predominant abnormality.[22] Similarly, research by Carrero demonstrated that thyroid dysfunction prevalence increases with declining renal function, supporting our stage-wise analysis.[23]

However, some studies have reported higher prevalences of subclinical and overt hypothyroidism in CKD populations than we observed. These differences may be attributed to variations in study populations, geographic factors, iodine nutritional status, and inclusion criteria. Our strict exclusion of patients with pre-existing thyroid disorders may have resulted in lower prevalence rates of overt thyroid disease.

Mechanisms and Pathophysiology: Multiple mechanisms contribute to thyroid dysfunction in CKD beyond impaired peripheral conversion of T4 to T3. These include alterations in thyroid hormone binding proteins, decreased renal clearance of iodine leading to changes in intrathyroidal iodine content, reduced thyroid hormone receptor expression, and the effects of uremic toxins on the hypothalamic-pituitary-thyroid axis.[24,25]

Furthermore, medications commonly used in CKD management may influence thyroid function. Phosphate binders, erythropoietin, and various antihypertensive agents can interfere with thyroid hormone metabolism and should be considered when interpreting thyroid function tests in this population.

Limitations: Several limitations of our study should be acknowledged. The cross-sectional design precludes establishment of causality and does not allow assessment of temporal changes in thyroid function as CKD progresses. Longitudinal studies following patients through different CKD stages would provide more definitive data on the evolution of thyroid dysfunction.

We did not measure reverse T3 (rT3), which is typically elevated in low T3 syndrome and would have provided additional confirmation of this diagnosis. Additionally, we did not assess the impact of thyroid dysfunction on clinical outcomes such as cardiovascular events, hospitalization rates, or mortality, which would be valuable for determining the clinical significance of these findings.

The relatively small sample size in stage 2 CKD (n=12) may limit the power to detect subtle differences in this subgroup. Future studies with larger cohorts across all CKD stages would strengthen these observations.

Clinical Recommendations: Based on our findings, we recommend regular screening of thyroid function in CKD patients, particularly those with stage 4 and 5 disease. While routine thyroid hormone replacement for low T3 syndrome is not currently recommended due to lack of evidence for benefit, identification of patients with true hypothyroidism requiring treatment is essential.

Clinicians should be aware that standard reference ranges for thyroid hormones may not be appropriate for CKD patients, and interpretation of thyroid function tests should consider the stage of kidney disease. The diagnosis of hypothyroidism in CKD patients should not be based solely on low T3 levels but should require elevation of TSH levels.

Conclusion

This study demonstrates that thyroid dysfunction, particularly low T3 syndrome, is highly prevalent in chronic kidney disease patients and shows a strong correlation with the severity of renal impairment. The progressive decline in T3 and FT3 levels across advancing CKD stages, coupled with relatively preserved TSH levels, characterizes the typical pattern of thyroid dysfunction in this population. Low T3 syndrome affected 62% of CKD patients overall, increasing to 78% in stage 5 disease, making it the predominant thyroid abnormality in this population. The significant negative correlation between markers of renal function and T3 levels underscores the relationship between uremia and thyroid hormone metabolism. Regular monitoring of thyroid function should be considered an integral component of comprehensive CKD management. While the clinical significance of low T3 syndrome and the role of thyroid hormone replacement remain topics requiring further investigation, identification of patients with true hypothyroidism is essential for appropriate treatment. These findings emphasize the complex interplay between renal and thyroid function and highlight the need for a multidisciplinary approach to managing CKD patients. Future longitudinal studies and randomized controlled trials are necessary to establish the prognostic significance of thyroid dysfunction in CKD and to determine optimal management strategies for improving patient outcomes.

References

1. Mohamedali M, Reddy Maddika S, Vyas A, Iyer V, Cheriyath P. Thyroid disorders and chronic kidney disease. Int J Nephrol. 2014; 2014:520281.
2. Dousdampanis P, Trigka K, Fourtounas C. Diagnosis and management of chronic kidney disease in the elderly: a field of ongoing debate. Aging Dis. 2012;3(5):360-372.
3. Mariani LH, Berns JS. The renal manifestations of thyroid disease. J Am Soc Nephrol. 2012;23(1):22-26.
4. Rhee CM, Kalantar-Zadeh K, Streja E, et al. The relationship between thyroid function and estimated glomerular filtration rate in patients with chronic kidney disease. Nephrol Dial Transplant. 2015;30(2):282-287.
5. Iglesias P, Díez JJ. Thyroid dysfunction and kidney disease. Eur J Endocrinol. 2009; 160(4): 503-515.
6. Kutlay S, Atli T, Koseogullari O, Nergizoglu G, Duman N, Gullu S. Thyroid disorders in hemodialysis patients in an iodine-sufficient area. Artif Organs. 2005;29(4):329-332.
7. Wartofsky L, Burman KD. Alterations in thyroid function in patients with systemic illness: the "euthyroid sick syndrome". Endocr Rev. 1982;3(2):164-217.
8. Chopra IJ. Clinical review 86: Euthyroid sick syndrome: is it a misnomer? J Clin Endocrinol Metab. 1997;82(2):329-334.
9. Kaptein EM, Quion-Verde H, Chooljian CJ, et al. The thyroid in end-stage renal disease. Medicine (Baltimore). 1988;67(3):187-197.
10. Zoccali C, Mallamaci F, Tripepi G, et al. Low triiodothyronine and survival in end-stage renal disease. Kidney Int. 2006;70(3):523-528.
11. Meuwese CL, Gussekloo J, de Craen AJ, Dekker FW, den Elzen WP. Thyroid status and renal function in older persons in the general population. J Clin Endocrinol Metab. 2014; 99(8): 2689-2696.
12. Rhee CM, Alexander EK, Bhan I, Brunelli SM. Hypothyroidism and mortality among dialysis patients. Clin J Am Soc Nephrol. 2013; 8(4):593-601.
13. Kaptein EM. Thyroid hormone metabolism and thyroid diseases in chronic renal failure. Endocr Rev. 1996;17(1):45-63.
14. Lo JC, Chertow GM, Go AS, Hsu CY. Increased prevalence of subclinical and clinical hypothyroidism in persons with chronic kidney disease. Kidney Int. 2005;67(3):1047-1052.
15. Lim VS. Thyroid function in patients with chronic renal failure. Am J Kidney Dis. 2001;38(4 Suppl 1):S80-84.
16. Wiederkehr MR, Kalogiros J, Krapf R. Correction of metabolic acidosis improves thyroid and growth hormone axes in haemodialysis patients. Nephrol Dial Transplant. 2004;19(5):1190-1197.
17. Carrero JJ, Qureshi AR, Axelsson J, et al. Clinical and biochemical implications of low thyroid hormone levels (total and free forms) in euthyroid patients with chronic kidney disease. J Intern Med. 2007;262(6):690-701.
18. Ramirez G, Jubiz W, Gutch CF, et al. Thyroid abnormalities in renal failure: a study of 53 patients on chronic hemodialysis. Ann Intern Med. 1973;79(4):500-504.
19. Song SH, Kwak IS, Lee DW, Kang YH, Seong EY, Park JS. The prevalence of low triiodothyronine according to the stage of chronic kidney disease in subjects with a normal thyroid-stimulating hormone. Nephrol Dial Transplant. 2009;24(5):1534-1538.
20. Meuwese CL, Dekker FW, Lindholm B, Qureshi AR, Heimburger O, Barany P, et al. Baseline levels and trimestral variation of triiodothyronine and thyroxine and their association with mortality in maintenance hemodialysis patients. Clin J Am Soc Nephrol. 2012;7(1):131-138.
21. Lim VS, Fang VS, Katz AI, Refetoff S. Thyroid dysfunction in chronic renal failure: a study of the pituitary-thyroid axis and peripheral turnover kinetics of thyroxine and triiodothyronine. J Clin Invest. 1977; 60(3): 522-534.
22. Kaptein EM, Feinstein EI, Massry SG. Thyroid hormone metabolism in renal diseases. Contrib Nephrol. 1986; 50:11-23.
23. Carrero JJ, Qureshi AR, Axelsson J, et al. Comparison of nutritional and inflammatory markers in dialysis patients with reduced appetite. Am J Clin Nutr. 2007;85(3):695-701.
24. Spector DA, Davis PJ, Helderman JH, Bell B, Utiger RD. Thyroid function and metabolic state in chronic renal failure. Ann Intern Med. 1976;85(6):724-730.
25. Lim VS, Flanigan MJ, Zavala DC, Freeman RM. Protective adaptation of low serum triiodothyronine in patients with chronic renal failure. Kidney Int. 1985;28(3):541-549.