

Correlation of thyroid hormones and serum electrolytes with kidney function in chronic kidney disease: a cross-sectional observational study.

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ABSTRACT

Objective

To evaluate the association of thyroid hormones and serum electrolytes with kidney function in patients with chronic kidney disease.

Methods

This cross-sectional observational study was conducted over six months (June–November 2025) in the Departments of Biochemistry and Nephrology at a tertiary care hospital. A total of 250 adults with chronic kidney disease were recruited using purposive sampling. Patients with acute kidney injury, known thyroid disease, medications affecting thyroid function or electrolytes, critical illness, or pregnancy were excluded. Serum free triiodothyronine, free thyroxine, thyroid-stimulating hormone, electrolytes, creatinine, urea, and uric acid were measured using standard laboratory methods. Estimated glomerular filtration rate was calculated using the four-variable Modification of Diet in Renal Disease equation. Correlations were assessed using Pearson's or Spearman's correlation coefficient, as appropriate.

Results

The mean age was 56.4 ± 12.8 years, with 148 (59.2%) males. The mean estimated glomerular filtration rate was 28 ± 17 mL/min/1.73 m². Reduced free triiodothyronine was observed in 69% of patients, reduced free thyroxine in 25.6%, and elevated thyroid-stimulating hormone in 20%. Free triiodothyronine showed a strong positive correlation with estimated glomerular filtration rate ($r = 0.62$, $p < 0.001$), while thyroid-stimulating hormone showed a negative correlation ($r = -0.41$, $p < 0.01$). Potassium, magnesium, and phosphorus were negatively correlated, whereas calcium showed a positive correlation ($p < 0.001$).

Conclusion

Thyroid hormone and electrolyte abnormalities were common and associated with declining kidney function.

Recommendations

Regular monitoring of thyroid function and serum electrolytes should be considered in patients with advanced chronic kidney disease.

Keywords: Chronic kidney disease, Thyroid hormones, Electrolytes, eGFR, FT3, TSH

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INTRODUCTION

Chronic kidney disease (CKD) is irreversible loss of renal function that eventually impairs almost all aspects of metabolic and endocrine function. The loss of renal filtration function leads to toxin build-up, electrolyte imbalances, and severe impairment of hormone metabolism. CKD has become a global burden, with high morbidity and mortality resulting from its metabolic complications and cardiovascular consequences [1]. One of the less-studied but clinically relevant changes in CKD involves thyroid hormone and electrolyte disturbances, both of which play a critical role in regulating global physiology and disease

morbidity [2]. Thyroid dysfunction is common in CKD and involves changes in free triiodothyronine (FT3), free thyroxine (FT4), and thyroid-stimulating hormone (TSH). Impaired conversion of T4 to T3, inflammatory status, malnutrition, metabolic acidosis, and altered protein binding characteristics of thyroid hormones lead to a clinical entity termed "non- thyroidal illness syndrome" in CKD. Several studies have reported that the most common abnormality in CKD is reduced FT3, which may correlate with the severity of CKD and increased cardiovascular risk [3]. The buildup of uremic toxins disrupts the hypothalamic-pituitary-thyroid axis, impairs TSH response, and inhibits thyroid hormone

degradation, resulting in variable increases in TSH. The study also corroborates these findings with a significant decrease in FT3 and negative associations between TSH and eGFR, highlighting the fact that thyroid dysfunction is exacerbated with advancement of chronic kidney disease [4]. Electrolyte imbalances are also common in CKD. As the glomerular filtration rate (GFR) drops, the kidneys can no longer regulate sodium, potassium, calcium, magnesium, and phosphorus. Hyperkalemia may occur in moderate and advanced CKD, as a result of decreased distal tubular secretion and drugs such as angiotensin-converting enzyme (ACE) inhibitors and potassium-sparing diuretics. Hypermagnesemia can be seen in the advanced stages as renal secretion decreases [5]. Hypocalcemia is frequently present due to defective vitamin D activation and phosphate retention. Hyperphosphatemia is due to impaired clearance and contributes to secondary hyperparathyroidism, mineral bone disease, and calcification of the heart [6]. One study found similar changes, with progressive rises in potassium, magnesium, and phosphorus, and a fall in calcium, as CKD progresses [7]. Renal growth, renal blood flow, and tubular transport are dependent on thyroid hormones, as is glomerular filtration. Hypothyroidism can cause a decline in cardiac output and renal blood flow, compounding CKD. Electrolyte imbalances affect thyroid function: for instance, hypomagnesaemia affects thyroid hormone production, and calcium-phosphate levels affect protein binding. In turn, thyroid dysfunction may worsen electrolyte disturbances by affecting bone metabolism, renal tubular function and hormone balance.

Recognizing these associations is important as thyroid dysfunction and electrolyte disturbances are associated with increased disease complications, hospitalizations, and death [8]. CKD is a major health issue in India, and many patients suffer from metabolic and endocrine abnormalities. However, few studies have assessed the combined correlation of thyroid hormone levels and electrolyte profile with kidney function. Based on the uploaded study's correlations of FT3, TSH, and electrolytes with eGFR, further research in various clinical settings is warranted [9]. Early recognition of these biochemical changes can help clinicians predict complications, start timely interventions, and improve the management of CKD [10]. Hence, the objective of the current study is to explore the correlations of FT3, FT4, TSH, and serum electrolytes with kidney function in patients with CKD. These correlations will improve the monitoring, prognostication, and comprehensive management of this high-risk group.

The study seeks to assess the association between thyroid function parameters (FT3, FT4, TSH), critical serum electrolytes (Na^+ , K^+ , Ca^{2+} , Mg^{2+} , PO_4^{3-}), and indices of kidney function in patients with chronic kidney disease. The goals include measuring FT3, FT4, and TSH levels in various stages of CKD, assessing the electrolyte profile and variations in electrolyte disturbances with the progression of renal disease, establishing the correlation between thyroid hormone levels and renal function markers such as serum

creatinine, urea, and eGFR in CKD, and investigating the association between electrolyte abnormalities and renal dysfunction severity in patients with CKD.

MATERIALS AND METHODS

Study Design

This was a cross-sectional observational study conducted to assess the association of thyroid hormones, serum electrolytes, and renal function parameters among patients with chronic kidney disease (CKD).

Study Setting

The study was conducted over six months, from June to November 2025, in the Department of Biochemistry in collaboration with the Department of Nephrology at a tertiary care hospital. Biochemical investigations, including thyroid function tests, kidney function tests, and serum electrolyte measurements, were performed in the hospital's NABL-accredited central laboratory using established laboratory procedures and automated analytical systems.

The Department of Nephrology was responsible for the clinical evaluation and identification of patients with CKD, while the Department of Biochemistry performed the required biochemical investigations and laboratory analyses.

Participants

A total of 250 patients with confirmed CKD were recruited using a purposive sampling technique. Eligible participants were adults aged 18 years or older with confirmed CKD stages 1–5 and complete biochemical investigation reports. Patients with acute kidney injury, known thyroid disease, current use of medications known to affect thyroid hormone levels or electrolyte balance (including lithium, amiodarone, or diuretics), critical illness, or pregnancy were excluded. Following recruitment, participants were classified into CKD stages 1–5 according to estimated glomerular filtration rate (eGFR), based on the 2024 Kidney Disease: Improving Global Outcomes (KDIGO) criteria. Additional subgroup analyses were performed according to thyroid function status and the presence of electrolyte abnormalities.

Sample Size Calculation

The study included 250 participants. The sample size was calculated using the following formula:

$$n = Z^2 \times P(1 - P) / d^2$$

where n represents the required sample size, Z is the standard normal deviate corresponding to a 95% confidence level (1.96), P represents the anticipated prevalence of chronic kidney disease, and d represents the allowable margin of error.

Based on a recent systematic review and meta-analysis of community-based studies in India, the anticipated prevalence of

chronic kidney disease was taken as **13.24% (P = 0.1324)** [11]. An allowable margin of error of **4.2% (d = 0.042)** was used. Thus:

$$n = (1.96)^2 \times 0.1324 \times (1 - 0.1324) / (0.042)^2 \approx 250$$

Page | 3 Accordingly, a final sample of **250 participants** was included in the study.

Data Collection and Laboratory Methods

Demographic and clinical information, including age, sex, medical history, and underlying cause of CKD, was recorded using a structured data collection proforma. Following recruitment, 5 mL of venous blood was collected from each participant under aseptic conditions. Samples were centrifuged to separate serum and were stored at 2–8°C and analysed within one hour of collection to preserve sample integrity.

Serum-free triiodothyronine (FT3), free thyroxine (FT4), and thyroid-stimulating hormone (TSH) concentrations were measured using chemiluminescence immunoassay. Serum electrolytes, including sodium, potassium, calcium, magnesium, and phosphorus, and renal function parameters, including serum creatinine, urea, and uric acid, were measured using automated biochemical analysers and routine laboratory methods.

Assessment of Kidney Function

Renal function was assessed using the four-variable Modification of Diet in Renal Disease (MDRD) equation, incorporating serum creatinine, age, sex, and ethnicity to calculate eGFR. Participants were subsequently classified into CKD stages 1–5 according to the **2024 KDIGO criteria**. This classification enabled assessment of thyroid hormone and electrolyte abnormalities across different levels of kidney dysfunction.

Measures to Minimize Bias

Potential sources of selection and measurement bias were addressed by applying predefined inclusion and exclusion criteria uniformly to all participants. Patients with conditions that could independently influence thyroid hormone levels or serum electrolytes, including acute kidney injury, known thyroid disease, relevant medication use, critical illness, and pregnancy, were excluded.

To minimize measurement-related variability, all biochemical investigations were performed in the NABL-accredited central laboratory using established procedures and automated analysers. Blood samples were collected using standardized aseptic procedures, processed promptly,

stored under specified conditions, and analysed within one hour of collection.

Nevertheless, because purposive sampling was used and the study was conducted at a single tertiary-care hospital, selection bias and limited representativeness of the wider CKD population cannot be completely excluded.

Ethical Considerations

The study was conducted after obtaining approval from the Institutional Ethics Committee (IEC), Gajra Raja Medical College, Gwalior, M.P. The **IEC/IRB approval number** was 39/IEC-GRMC/2026, and ethical approval was granted on 27/12/2025. Written informed consent was obtained from all participants before enrolment. Patient confidentiality and anonymity were maintained throughout data collection, laboratory analysis, and reporting.

Statistical Analysis

Data were entered into Microsoft Excel and analysed using SPSS. Continuous variables were expressed as mean ± standard deviation, while categorical variables were presented as frequencies and percentages. The relationships between thyroid hormone levels, serum electrolytes, and kidney function parameters were assessed using Pearson's or Spearman's correlation coefficient, as appropriate. A two-sided *p*-value <0.05 was considered statistically significant.

RESULTS

A total of 250 patients with confirmed chronic kidney disease were assessed for eligibility and included in the study. All 250 participants underwent the required clinical and laboratory assessments and were included in the final analysis.

Baseline Demographic and Clinical Characteristics

Table 1 shows the baseline characteristics of the population. The study population comprised 250 patients with CKD with a mean age of 56.4 ± 12.8 years. The cohort consisted of 148 males (59.2%) and 102 females (40.8%). The mean eGFR was 28 ± 17 mL/min/1.73 m² (range: 5–90). Most patients had end-stage CKD, particularly Stage 4 (32.8%) and Stage 5 (27.2%), suggesting a significant proportion of patients suffering from severe kidney disease (Figure 1).

The study population comprised 250 patients with a mean age of 56.4 ± 12.8 years. There were 148 (59.2%) males and 102 (40.8%) females. The mean eGFR was 28 ± 17 mL/min/1.73 m². Stage 4 CKD was the most frequent, affecting 82 (32.8%) patients, followed by Stage 5 in 68 (27.2%) patients.

Table 1: Baseline Demographic and Clinical Characteristics (n = 250)

Parameter	Mean $\pm$ SD / n (%)
Age (years)	56.4 $\pm$ 12.8
Male: Female	148 (59.2%): 102 (40.8%)
Mean eGFR (mL/min/1.73 m ²)	28 $\pm$ 17 (Range 5–90)
CKD Stage 1	18 (7.2%)
CKD Stage 2	30 (12.0%)
CKD Stage 3	52 (20.8%)
CKD Stage 4	82 (32.8%)
CKD Stage 5	68 (27.2%)

Thyroid Function Abnormalities in CKD Patients

CKD patients exhibited a high incidence of thyroid dysfunction, with decreased FT3 levels in 69% of patients and decreased FT4 levels in 25.6%. Increased TSH was observed in 20% of patients, suggestive of overt or subclinical hypothyroidism. These results indicate that renal dysfunction has a profound impact on thyroid hormone metabolism, especially FT3. Table 2 presents the

distribution of thyroid dysfunction across the CKD stages and shows increasing severity with declining kidney function (Figure 2). Low FT3 levels rose from 24% in early stages to 88% in Stage 5, with increasing TSH to 38% in later stages.

Decreased FT3 was observed in 172 (69.0%) patients, decreased FT4 in 64 (25.6%), and increased TSH in 50 (20.0%). The proportion of patients with low FT3, low FT4, and elevated TSH increased across advancing CKD stages.

Table 2: Distribution of Thyroid Dysfunction Across CKD Stages

CKD Stage	Low FT3 (%)	Low FT4 (%)	High TSH (%)
Stage 1–2	24%	10%	6%
Stage 3	48%	18%	12%
Stage 4	72%	32%	24%
Stage 5	88%	46%	38%

Correlation of Thyroid Markers with Kidney Function

The results for correlation of thyroid hormones with kidney function (eGFR) are shown in Table 3. FT3 was strongly correlated with eGFR ($r = +0.62$, $p < 0.001$) and TSH was moderately inversely correlated ($r = -0.41$, $p < 0.01$). The

correlation for FT4 was not significant ($p = 0.21$). This suggests that thyroid function worsens as kidney function fails.

FT3 showed a strong positive correlation with eGFR ($r = 0.62$, $p < 0.001$), whereas TSH showed a moderate inverse correlation ($r = -0.41$, $p < 0.01$). FT4 was not significantly correlated with eGFR ($r = 0.08$, $p = 0.21$).

Table 3: Correlation of Thyroid Markers with Kidney Function (eGFR)

Parameter	Correlation Coefficient (r)	p-value
FT3	+0.62	<0.001
FT4	+0.08	0.21

Correlation of Electrolytes with Kidney Function

The correlation of electrolytes with renal function is shown in Table 4. There were strong negative correlations between eGFR and potassium ($r = -0.55$), magnesium ($r = -0.58$), and phosphorus ($r = -0.61$) ($p < 0.001$ for each). Calcium was

positively associated with eGFR ($r = +0.49$, $p < 0.001$), suggesting a drop in advanced stages of CKD. There were no correlations with sodium ($p = 0.18$). eGFR showed significant negative correlations with potassium ($r = -0.55$), magnesium ($r = -0.58$), and phosphorus ($r = -0.61$), all with $p < 0.001$. Calcium showed a significant positive correlation with eGFR ($r = 0.49$, $p < 0.001$).

Table 4: Correlation of Electrolytes with Kidney Function (eGFR)

Electrolyte	Correlation Coefficient (r)	p-value
Sodium (Na ⁺)	-0.09	0.18 (NS)
Potassium (K ⁺)	-0.55	<0.001
Calcium (Ca ²⁺)	+0.49	<0.001
Magnesium (Mg ²⁺)	-0.58	<0.001
Phosphorus (PO ₄ ³⁻)	-0.61	<0.001

Prevalence of Electrolyte Abnormalities

Table 5 shows the prevalence of electrolyte abnormalities in CKD patients. The most prevalent electrolyte abnormalities were hyperphosphatemia (35.2%) and hyperkalemia

(32.8%), followed by hypocalcemia (29.6%) and hypermagnesemia (25.6%). The prevalence of hyponatremia was 18.4%. These reflect reduced renal clearance and altered mineral metabolism in CKD (Figure 3).

Table 5. Prevalence of Electrolyte Abnormalities (n = 250)

Abnormality	n	%
Hyperkalemia	82	32.8%
Hypocalcemia	74	29.6%
Hyperphosphatemia	88	35.2%
Hypermagnesemia	64	25.6%
Hyponatremia	46	18.4%

DISCUSSION

Key Results

The present study evaluated the association of thyroid hormone abnormalities and serum electrolyte disturbances with kidney function among 250 patients with chronic kidney disease (CKD). The mean age of the study population was 56.4 ± 12.8 years, with 148 (59.2%) males and 102 (40.8%) females. A substantial proportion of participants had advanced CKD: 82 (32.8%) patients were in Stage 4 and 68 (27.2%) in Stage 5, and the mean estimated glomerular filtration rate (eGFR) was 28 ± 17 mL/min/1.73 m² (Table 1). The predominance of advanced CKD in this cohort provides important context for the high frequency of endocrine and electrolyte abnormalities observed in the study.

The study demonstrated a high frequency of thyroid abnormalities. Reduced free triiodothyronine (FT3) was observed in 172 (69.0%) patients, reduced free thyroxine (FT4) in 64 (25.6%), and elevated thyroid-stimulating hormone (TSH) in 50 (20.0%) patients. As shown in Table 2, the frequency of low FT3, low FT4, and elevated TSH increased progressively across CKD stages, with low FT3 reaching 88% and elevated TSH reaching 38% in Stage 5 disease.

The correlation analysis further demonstrated that FT3 had a strong positive correlation with eGFR ($r = +0.62$, $p < 0.001$), whereas TSH showed a moderate inverse correlation with eGFR ($r = -0.41$, $p < 0.01$). FT4 did not demonstrate a statistically significant correlation with eGFR ($r = +0.08$, $p = 0.21$) (Table 3). These findings suggest that alterations in thyroid hormone metabolism, particularly reduced FT3, are closely associated with declining renal function.

Serum electrolyte abnormalities were also significantly associated with kidney function. Potassium, magnesium, and phosphorus showed significant negative correlations with eGFR, whereas calcium showed a positive correlation. Sodium did not show a statistically significant correlation with eGFR (Table 4). The most frequent electrolyte abnormalities were hyperphosphatemia in 88 (35.2%) patients, hyperkalemia in 82 (32.8%), hypocalcemia in 74 (29.6%), hypermagnesemia in 64 (25.6%), and hyponatremia in 46 (18.4%) patients (Table 5).

Interpretation of Findings

The high frequency of reduced FT3 observed in the present study is consistent with previous observations that reduced FT3 is one of the most common thyroid abnormalities in patients with CKD and may increase with the severity of renal dysfunction. The findings are comparable with those that reported low T3 in 47.5% and low T4 in 30% of CKD patients and observed an increase in thyroid abnormalities with decreasing glomerular filtration rate [12]. In the present study, the progressive increase in low FT3 and elevated TSH across CKD stages (Table 2), together with the positive correlation between FT3 and eGFR and inverse correlation between TSH and eGFR (Table 3), further supports an association between worsening renal function and altered thyroid hormone metabolism.

The predominance of reduced FT3 may reflect the alterations in peripheral thyroid hormone metabolism associated with chronic illness and CKD. Impaired conversion of thyroxine to triiodothyronine, inflammation, malnutrition, metabolic acidosis, altered protein binding, and the effects of uremic toxins have been implicated in the

development of non-thyroidal illness patterns in CKD. Thus, the thyroid abnormalities observed in this study should not necessarily be interpreted as evidence of primary thyroid disease, particularly because patients with known thyroid disorders were excluded.

The electrolyte findings are also biologically consistent with progressive renal impairment. As renal filtration declines, the capacity of the kidneys to regulate potassium, magnesium, calcium, and phosphorus becomes increasingly impaired. Hyperkalemia may result from reduced distal tubular potassium secretion, while increased magnesium and phosphorus concentrations may reflect reduced renal clearance. Hypocalcemia may occur because of impaired vitamin D activation and phosphate retention [5,6]. The present findings, particularly the negative correlations of potassium, magnesium, and phosphorus with eGFR and the positive correlation of calcium with eGFR (Table 4), are therefore consistent with these established mechanisms.

The observed electrolyte abnormalities are also comparable with previous studies. One study reported alterations in serum electrolytes among patients with CKD, while another study demonstrated relationships between magnesium and renal biochemical parameters and reported that sodium was not significantly associated with renal dysfunction [13,14]. In the present study, sodium similarly showed no significant correlation with eGFR, suggesting that sodium homeostasis may be maintained through compensatory mechanisms despite declining renal function, although it may become disturbed in advanced or complicated disease.

The prevalence of electrolyte abnormalities in the present study was higher than that reported by a study that identified hyperkalemia and hypocalcemia even in early CKD [15]. The higher prevalence observed in the current study may partly reflect the substantial proportion of participants with Stage 4 and Stage 5 CKD. The concentration of advanced CKD cases in this cohort therefore needs to be considered when interpreting the frequency of electrolyte disturbances and limits direct comparison with populations containing predominantly early-stage CKD.

Taken together, the findings support the study objective that thyroid hormone abnormalities and electrolyte disturbances are associated with declining kidney function. The results presented in **Tables 2–5** demonstrate that these abnormalities become more prominent with worsening renal dysfunction. However, because this was a cross-sectional study, the observed correlations should be interpreted as **associations rather than causal relationships**. The study cannot establish whether thyroid and electrolyte abnormalities contribute to CKD progression or primarily result from worsening renal function.

Generalizability

The findings may be relevant to patients with CKD managed in tertiary-care nephrology settings, particularly individuals with moderate-to-advanced renal dysfunction. However, their generalizability to the broader CKD population should be interpreted cautiously. The study was conducted at a

single tertiary-care centre and used purposive sampling, and the study population contained a relatively high proportion of patients with advanced CKD. Therefore, the frequency of thyroid and electrolyte abnormalities observed here may be higher than that in community-based or primary-care CKD populations.

Multicentre studies involving larger and more diverse patient populations, including greater representation of early-stage CKD, would help determine whether the observed associations are consistent across different clinical settings.

Conclusion

This article shows that there is a definite correlation between deterioration of kidney function and major changes in thyroid hormones and electrolytes in patients with CKD. The levels of reduced FT3 (69%) and high TSH (20) became more prevalent in more advanced CKD and are indicative of metabolic adjustments and poor clearance of hormones instead of primary thyroid disease. FT3 was strongly positively correlated with eGFR, and TSH was negatively correlated, which means that they are useful as auxiliary indicators of the severity of CKD. Other electrolyte imbalances like hyperkalemia (32.8%), hyperphosphatemia (35.2%), and hypocalcemia (29.6%) were also common, especially during stage 4-5, which is indicative of impaired renal excretion and mineral metabolism. On the whole, these data support the idea of the necessity of regular assessment of thyroid activity and electrolytes in CKD patients to prevent complications and enhance their clinical management.

Limitations

This study has several limitations. First, its cross-sectional design prevents assessment of temporal relationships and causal inference. Second, the single-centre setting and purposive sampling technique may have introduced selection bias, as patients attending a tertiary-care hospital may differ systematically from patients with CKD in community or primary-care settings. This may limit the external validity of the findings.

Third, the relatively large proportion of patients with Stage 4 and Stage 5 CKD may have introduced sampling imbalance and imprecision in stage-specific estimates, potentially contributing to the high observed prevalence of thyroid and electrolyte abnormalities. Fourth, although patients with known thyroid disease and medications that could affect thyroid or electrolyte measurements were excluded, residual confounding from other clinical factors cannot be completely excluded.

Finally, the study assessed biochemical parameters at a single point in time. Consequently, it cannot determine whether changes in FT3, TSH, or electrolyte concentrations predict subsequent decline in kidney function. Prospective longitudinal studies with larger, more representative samples are required to clarify the prognostic significance of these abnormalities.

Recommendations

The findings of this study have potential clinical implications for the monitoring of patients with chronic kidney disease, particularly those with advanced disease. Regular assessment of FT3, TSH, and serum electrolytes, including potassium, magnesium, calcium, and phosphorus, should be considered in patients with CKD, especially in advanced stages. Early identification of thyroid and electrolyte abnormalities may facilitate appropriate clinical evaluation, correction of metabolic disturbances, and prevention of associated complications.

Further prospective longitudinal studies are recommended to determine whether thyroid hormone and electrolyte abnormalities independently predict CKD progression and adverse clinical outcomes. Future studies should include larger, multicentre populations with greater representation of early-stage CKD and should evaluate whether correction of these abnormalities improves clinical outcomes. Studies incorporating additional factors such as nutritional status, inflammatory markers, medication use, and comorbidities may also help clarify the mechanisms underlying the observed associations.

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List of Abbreviations

CKD: Chronic Kidney Disease

eGFR: Estimated Glomerular Filtration Rate

GFR: Glomerular Filtration Rate

FT3: Free Triiodothyronine

FT4: Free Thyroxine

TSH: Thyroid-Stimulating Hormone

MDRD: Modification of Diet in Renal Disease

KDIGO: Kidney Disease: Improving Global Outcomes

ANOVA: Analysis of Variance

SPSS: Statistical Package for the Social Sciences

SD: Standard Deviation

Conflict of Interest

The authors declare that they have no conflict of interest related to this study or its publication.

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Author Contributions

Susmita Sarkar: Conceptualization, study design, data collection, laboratory investigations, data analysis, interpretation of findings, and manuscript drafting.

Sanjeev Kumar Singh: Study supervision, methodology, interpretation of biochemical findings, critical revision of the manuscript, and final approval.

Manish Kumar Kain: Laboratory investigations, data collection, data management, and critical revision of the manuscript.

Shivam Yadav: Clinical supervision, nephrological assessment, interpretation of clinical findings, and critical revision of the manuscript.

All authors reviewed and approved the final manuscript and agreed to be accountable for all aspects of the work.

Data Availability

The datasets generated and/or analysed during the current study are not publicly available because they contain patient-related clinical and laboratory information but may be available from the corresponding author on reasonable request, subject to institutional and ethical restrictions.

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