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# Altered Thyroid Hormones in Chronic Kidney Disease: A Cross-Sectional Comparison of Hemodialysis and Conservative Treatment

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## ABSTRACT

**Background:** Chronic kidney disease is commonly associated with endocrine disturbances, particularly involving the hypothalamic-pituitary-thyroid axis. Thyroid dysfunction in chronic kidney disease (CKD) patients can complicate clinical management and affect overall prognosis; however, it often remains underdiagnosed, especially in resource-limited situations. This study aims to assess and compare alterations in thyroid hormone profiles—total triiodothyronine, total thyroxine, and thyroid-stimulating hormone—among CKD patients on haemodialysis and those under conservative medical management.

**Methods:** This hospital-based, cross-sectional study was conducted at a tertiary care centre in North India between April 2024 and March 2025. A total of 80 patients with stage 5 CKD were included: 40 on maintenance haemodialysis and 40 under conservative treatment. Serum TT3, TT4, and Thyroid Stimulating Hormone (TSH) levels were measured using chemiluminescent immunoassay and compared between the two groups. A control group of 50 age- and sex-matched healthy individuals was included for baseline comparison. Statistical analysis was performed using Student's t-test and the chi-square test.

**Results:** CKD patients exhibited significantly lower TT3 and TT4 levels and higher TSH levels compared to controls ($p<0.0001$). Among CKD subgroups, haemodialysis patients had significantly lower TT3 (mean 34.48 ng/dL) and TT4 (mean 5.24 µg/dL) levels and markedly elevated TSH (mean 26.85 µIU/mL) compared to conservatively managed patients (TT3: 46.76 ng/dL, TT4: 8.10 µg/dL, TSH: 6.19 µIU/mL), all with statistically significant differences ($p<0.005$).

**Conclusion:** The study concluded that there is a significant association between abnormalities in kidney function and thyroid hormone levels in patients with CKD.

**Key-words:** Chronic kidney disease, Thyroid dysfunction, Haemodialysis, Conservative management, TT3, TT4, TSH, Non-thyroidal illness syndrome, Endocrine abnormalities, Renal failure

## INTRODUCTION

Chronic kidney disease is a progressive and irreversible deterioration of renal function characterized by a reduction in glomerular filtration rate and changes in fluid, electrolyte, and hormonal balance [1].

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As CKD advances through its stages, systemic complications manifest due to the impaired excretion and metabolism of numerous endogenous substances, including thyroid hormones [2]. Cardiovascular disorders are common, with a considerable proportion of patients eventually requiring renal replacement therapy such as haemodialysis, and the occurrence of CKD is increasing worldwide due to the rise in hypertension and diabetes mellitus [3].

The complex interplay between kidney and thyroid function has been extensively recognized [4]. The kidneys contribute to the metabolism, degradation, and excretion of thyroid hormones and influence the

 

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peripheral conversion of thyroxine (T4) to the more active triiodothyronine (T3) [5]. Similarly, thyroid hormones have a regulatory role in renal development, renal hemodynamics, and electrolyte homeostasis [6]. Therefore, it is not surprising that CKD leads to a spectrum of thyroid dysfunction, often collectively termed "non-thyroidal illness syndrome" or "euthyroid sick syndrome," with changes in total and free T3 and T4 levels, and differences in thyroid-stimulating hormone concentrations [7].

Several studies have reported that patients with CKD, predominantly those undergoing maintenance haemodialysis, frequently exhibit low T3 levels with normal T4 and TSH, indicative of low T3 syndrome [8]. This syndrome is thought to arise from decreased peripheral conversion of T4 to T3, possibly due to reduced deiodinase activity, inflammation, and protein-energy wasting [9]. On the other hand, patients managed conservatively (without dialysis) may show different patterns of thyroid dysfunction, potentially influenced by the degree of renal insufficiency, nutritional status, and the presence of comorbid conditions [10].

Understanding the pattern of thyroid hormone alterations in CKD patients is clinically significant for several reasons. First, thyroid dysfunction in these patients may be subclinical and remain undiagnosed unless actively investigated, leading to unexplained fatigue, cold intolerance, weight gain, and worsening of cardiovascular risk [11]. Second, alterations in thyroid profile, particularly low T3 levels, have been associated with increased mortality, especially in dialysis populations [12]. Third, inappropriate initiation of thyroid hormone therapy in patients with non-thyroidal illness syndrome can lead to overtreatment and additional complications [13].

Despite the growing awareness of thyroid dysfunction in CKD, studies comparing the thyroid hormone profiles between patients undergoing haemodialysis and those managed conservatively remain limited and inconclusive. Factors such as the chronic inflammatory state, altered protein binding, reduced hormone clearance, and the dialysis process itself may contribute to differing thyroid hormone patterns in these subgroups. Haemodialysis, by removing uremic toxins and possibly affecting protein-bound hormone levels, may alter the hormonal milieu differently compared to conservative management, which depends on pharmacologic and dietary interventions to slow disease progression [1,3].

This cross-sectional study aims to assess and compare the thyroid hormone profiles, specifically TSH, free T3 (FT3), and free T4 (FT4), in patients with CKD receiving haemodialysis versus those on conventional management. By identifying the occurrence and pattern of thyroid abnormalities in these two patient populations, the study seeks to contribute to the growing body of evidence that advocates for routine thyroid function screening in CKD management protocols. In addition, it explores potential associations between thyroid hormone levels and clinical variables such as duration of CKD, serum creatinine, estimated glomerular filtration rate, and inflammatory markers [4,7].

A clearer understanding of thyroid profiles in CKD patients can help tailor treatment strategies and improve prognosis. It may aid in distinguishing adaptive hormonal changes from true thyroid disorders needing intervention. Additionally, it offers insight into how different renal management approaches may impact thyroid function and long-term outcomes.

**Table 1:** Pathophysiological Mechanisms of Thyroid Dysfunction in Chronic Kidney Disease [14]

| Mechanism | Effect on Thyroid Function |
|---|---|
| ↓ Deiodinase activity | ↓ T4 to T3 conversion → Low FT3 |
| ↓ Thyroid hormone clearance | Altered TSH feedback regulation |
| ↑ Inflammatory cytokines | Suppression of the hypothalamic-pituitary-thyroid axis |
| Dialysis-related loss | Removal of protein-bound or free thyroid hormones |
| Malnutrition/protein loss | ↓ Thyroid hormone binding proteins (e.g., TBG) |

## MATERIALS AND METHODS

**Research Design-** This hospital-based, cross-sectional, observational study was conducted at our tertiary care teaching hospital in North India. The study was carried out over a one-year period from April 2024 to March 2025. Before the beginning of the study, ethical clearance was obtained from the Institutional Ethics Committee of the hospital. A total of 80 participants were included in the study, comprising 40 patients diagnosed with stage 5 chronic kidney disease and 40

 

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age- and sex-matched healthy individuals serving as controls. CKD patients were further categorized based on their management: those on maintenance haemodialysis (HD group, n=20) and those managed conservatively without dialysis (CM group, n=20). Participants were recruited from both inpatient and outpatient departments of the Medicine and Nephrology units, including the dialysis unit.

**Inclusion Criteria**

- Age between 40 and 70 years
- Diagnosis of stage 5 CKD, defined by estimated glomerular filtration rate <15 ml/min/1.73 m², calculated using the 4-variable MDRD formula
- Serum creatinine >5.5 mg/dL and blood urea >55 mg/dL
- Presence of proteinuria on dipstick testing
- No history of thyroid disease
- Willingness to provide written informed consent

**Exclusion Criteria**

- Age below 40 years
- Known thyroid dysfunction or family history of goitre
- Ongoing treatment for any thyroid disorder
- Use of medications known to affect thyroid function, including glucocorticoids, salicylates, lithium, amiodarone, sulfonylureas, heparin, or phenobarbitone
- Acute illness or infection at the time of sampling

**Sample Collection and Biochemical Estimation-** All participants underwent fasting venous blood collection (2 mL) from the median cubital vein, using strict aseptic techniques. Blood samples were collected into clot activator vials and allowed to clot at room temperature for 30–45 minutes. The samples were then centrifuged at 3000 rpm for 5 minutes. The serum obtained was used immediately or stored in a clean vial if analysis could not be performed immediately. Hemolyzed samples were rejected.

The following biochemical parameters were analyzed:

- Serum total triiodothyronine (TT3)
- Serum total thyroxine (TT4)
- Thyroid-stimulating hormone (TSH)
- Serum creatinine
- Blood urea

Blood urea and serum creatinine levels were measured using the urease method and Jaffe alkaline picrate method, respectively, on the Beckman Coulter AU 480 autoanalyzer. Thyroid hormone assays were performed using the Access 2 Immunoassay System employing a chemiluminescent immunoassay method.

**Statistical Analysis-** All collected data were recorded in Microsoft Excel and statistically analysed using GraphPad InStat version 3.00 for Windows. Continuous variables were expressed as mean±standard deviation, and standard error of the mean where applicable. Confidence intervals were also calculated. Comparisons between groups were made using Student's t-test for continuous variables and the chi-square test for categorical variables. A $p<0.05$ was considered statistically significant. The thyroid hormone profiles were compared across the three groups: CKD patients on haemodialysis, those on conservative management, and the healthy control group.

**RESULTS**

The mean blood urea level in CKD patients was 153.94±72.50 mg/dL, significantly higher than 22.91±7.36 mg/dL observed in controls ($p<0.0001$). Similarly, serum creatinine levels in CKD cases averaged 8.76±3.48 mg/dL, in stark contrast to 0.75±0.31 mg/dL in the control group ($p<0.0001$) (Table 1).

**Table 1:** Comparison of Mean Blood Urea and Serum Creatinine Levels Between CKD Patients and Healthy Controls

| Variable | Group | Mean | SD | SEM | 95% CI | p-value |
|---|---|---|---|---|---|---|
| Blood Urea (mg/dL) | Cases | 153.94 | 72.5 | 10.25 | 133.32 – 174.57 | <0.0001 |
| | Controls | 22.91 | 7.36 | 1.04 | 20.81 – 25.06 | |
| Serum Creatinine (mg/dL) | Cases | 8.76 | 3.48 | 0.49 | 7.78 – 9.76 | <0.0001 |
| | Controls | 0.75 | 0.31 | 0.04 | 0.66 – 0.83 | |

 

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Thyroid function analysis in CKD patients revealed a high prevalence of hormonal abnormalities, most notably affecting TT3 and TSH levels. Notably, 100% of the CKD patients had low TT3 levels, suggesting a universal occurrence of low T3 syndrome, a common feature in chronic systemic illness such as end-stage renal disease. TT4 levels were within the normal range in the majority (62%) of patients; however, 36% exhibited low TT4, and 2% had elevated levels, indicating variable thyroxine alterations that may reflect differing stages or adaptations of the illness. TSH levels were abnormal in most patients: 76% showed elevated TSH, representing a hypothyroid pattern, while only 22% had normal TSH, and 2% had suppressed levels (Table 2).

**Table 2:** Prevalence of Thyroid Hormone Abnormalities in Chronic Kidney Disease Patients

| Thyroid Parameter (Normal Range) | Normal | Low | High |
|---|---|---|---|
| TT3 (81–171 ng/dL) | 0 (0%) | 50 (100%) | 0 (0%) |
| TT4 (4–12 µg/dL) | 31 (62%) | 18 (36%) | 1 (2%) |
| TSH (0.4–4.0 µIU/mL) | 11 (22%) | 1 (2%) | 38 (76%) |

The mean TT3 level in CKD patients (40.77±12.58 ng/dL) was markedly lower than that in healthy controls (109.27±22.26 ng/dL), with a $p<0.0001$, indicating a highly significant reduction in circulating triiodothyronine among patients with CKD. This reflects the classic "low T3 syndrome", often associated with chronic systemic illness and impaired peripheral conversion of T4 to T3 due to uraemia and inflammation. Similarly, TT4 levels were also significantly lower in CKD patients (6.61±3.06 µg/dL) compared to controls (8.94±1.91 µg/dL, $p<0.0001$). Although not as dramatically reduced as TT3, this decline in TT4 levels in CKD patients further reinforces the imbalance in thyroid hormone metabolism that accompanies advanced kidney disease (Table 3).

**Table 3:** Comparison of Mean Thyroid Hormone Levels Between CKD Patients and Healthy Controls

| Thyroid Hormone | Group | Mean | SD | SEM | 95% CI | p-value |
|---|---|---|---|---|---|---|
| TT3 (ng/dL) | Cases | 40.77 | 12.58 | 1.77 | 37.20 – 44.35 | <0.0001 |
| | Controls | 109.27 | 22.26 | 3.14 | 102.94 – 115.60 | |
| TT4 (µg/dL) | Cases | 6.61 | 3.06 | 0.43 | 5.74 – 7.48 | <0.0001 |
| | Controls | 8.94 | 1.91 | 0.27 | 8.39 – 9.48 | |
| TSH (µIU/mL) | Cases | 16.92 | 26.97 | 3.81 | 9.25 – 24.60 | 0.0002 |
| | Controls | 2.29 | 1.24 | 0.17 | 1.93 – 2.64 | |

TT3 levels were significantly lower in patients on haemodialysis (34.48±11.21 ng/dL) compared to those on conservative therapy (46.76±11.84 ng/dL, $p=0.0005$), suggesting a greater suppression of peripheral triiodothyronine in those with more advanced or treated renal failure. Similarly, TT4 levels were also reduced in the haemodialysis group (5.24±3.02 µg/dL) compared to the conservative group (8.10±2.38 µg/dL, $p=0.0006$), reflecting potential loss of binding proteins, altered clearance, or dialysis-related endocrine disruption. Notably, TSH levels were markedly elevated in the haemodialysis group (26.85±34.59 µIU/mL) versus the conservative group (6.19±4.07 µIU/mL, $p=0.0055$), though with a wide standard deviation and confidence interval, representing a heterogeneous thyroid response among dialysis patients (Table 4).

 

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**Table 4:** Comparison of Thyroid Hormone Levels in CKD Patients Based on Treatment Modality

| Hormone | Group | Mean | SD | SEM | 95% CI | p-value |
|---|---|---|---|---|---|---|
| TT3 (ng/dL) | Conservative | 46.76 | 11.84 | 2.49 | 42.02 – 51.49 | 0.0005 |
| | Haemodialysis | 34.48 | 11.21 | 2.24 | 30.17 – 38.78 | |
| TT4 (µg/dL) | Conservative | 8.1 | 2.38 | 0.49 | 7.10 – 9.11 | 0.0006 |
| | Haemodialysis | 5.24 | 3.02 | 0.59 | 4.05 – 6.46 | |
| TSH (µIU/mL) | Conservative | 6.19 | 4.07 | 0.83 | 4.45 – 7.89 | 0.0055 |
| | Haemodialysis | 26.85 | 34.59 | 6.79 | 12.88 – 40.83 | |

## DISCUSSION

Chronic Kidney Disease relates to numerous systemic problems, and one of the less obvious but clinically important consequences is the disturbance of thyroid hormone homeostasis. The interaction between kidney function and thyroid regulation is complex, involving changes in hormone production, metabolism, transport, and excretion [15]. The current cross-sectional study intended to assess the thyroid hormone profile in CKD patients, with a specific comparison between those on haemodialysis and those under conservative management. Our results exposed important modifications in TT3, TT4, and TSH levels in CKD patients compared to healthy controls, and additionally established that these abnormalities were more pronounced in patients experiencing haemodialysis [16].

Our data showed an important decrease in serum total triiodothyronine levels in CKD patients. The mean TT3 in cases was decidedly lower than in controls (40.77±12.58 ng/dL vs. 109.27±22.26 ng/dL, $p$<0.0001). This makes even with the well-documented "low T3 syndrome", a non-thyroidal illness condition frequently seen in CKD. The syndrome is supposed to be an adaptive response to chronic illness, where the peripheral conversion of thyroxine to the biologically active triiodothyronine is diminished. This reduction is primarily attributed to downregulation of type I and type II deiodinases in the kidney and liver under uremic and inflammatory conditions [17,18].

Total thyroxine levels were also significantly decreased in CKD patients compared to controls (6.61±3.06 µg/dL vs. 8.94±1.91 µg/dL, $p$<0.0001). However, the reduction in TT4 was less drastic than that seen with TT3, indicating that the primary defect lies in peripheral hormone conversion rather than T4 synthesis itself.

Reduced protein binding due to hypoalbuminemia or uraemia-induced changes in thyroxine-binding globulin may also contribute to changed TT4 levels [19].

The most interesting result in this study of thyroid-stimulating hormone in CKD patients (mean: 16.92±26.97 µIU/mL vs. 2.29±1.24 µIU/mL in controls, $p$=0.0002) was the important promotion. TSH promotion suggests that the hypothalamic-pituitary-thyroid axis may be responding to perceived thyroid hormone deficiency. However, the wide variation and high standard deviation in TSH levels among CKD patients point to heterogeneous responses, ranging from subclinical hypothyroidism to obvious primary hypothyroidism or central dysregulation due to uremic toxin effects on pituitary function [20].

When comparing treatment modalities, patients on haemodialysis had significantly lower TT3 (34.48±11.21 ng/dL) and TT4 (5.24±3.02 µg/dL) levels than those managed conventionally (TT3: 46.76±11.84 ng/dL; TT4: 8.10±2.38 µg/dL). This may be due to the more advanced disease state in the haemodialysis group, chronic inflammation, protein-energy wasting, and potential losses of protein-bound hormones during dialysis [21,22]. In addition, TSH levels were substantially higher in the haemodialysis group (26.85±34.59 µIU/mL) compared to the conservative group (6.19±4.07 µIU/mL), with important differences ($p$=0.0055). The exaggerated TSH elevation in haemodialysis patients suggests impaired renal metabolism and clearance of TSH and a potentially greater degree of subclinical or overt hypothyroidism in this subgroup [23].

Our results document previous studies by Chonchol *et al.* [24] and Lo *et al.* [25], who reported a higher occurrence of hypothyroidism in CKD populations, especially among those experiencing dialysis. CKD relates to increased

 

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cardiovascular risk, dyslipidaemia, and higher death rates; the need for early identification and management emphasises that even subclinical hypothyroidism.

The clinical implication of changed thyroid hormone levels in CKD should not be underestimated. Thyroid hormones play a critical part in basal metabolic rate, erythropoiesis, cardiovascular function, and overall nutritional status, all areas that are compromised in CKD. Moreover, thyroid dysfunction in CKD may, in addition, impair renal function by affecting glomerular hemodynamics and sodium handling [26].

There are numerous limitations to this study. Firstly, the sample size was modest, and although sufficient to detect statistical differences, larger multicentre studies are needed for generalization. Then, we assessed only total T3 and T4 rather than free hormone levels, which could provide a more accurate understanding of the bioavailable hormone status. Thirdly, this study did not assess reverse T3, which could help differentiate between euthyroid sick syndrome and true hypothyroidism.

However, the strong point of this study includes its well-defined inclusion and exclusion criteria, biochemical assessment with automated chemiluminescent immunoassay, and clear stratification based on treatment modality. The data propose that routine thyroid function monitoring in CKD patients, particularly those on dialysis, should be considered essential in clinical practice.

## CONCLUSIONS

The study concludes that thyroid hormone disturbances are significantly associated with impaired renal function in patients with CKD. Elevated blood urea and serum creatinine levels confirm declining kidney function, while thyroid profile assessment revealed that all CKD patients exhibited low total T3 (TT3) levels, characteristic of non-thyroidal illness syndrome. Total T4 (TT4) levels were also reduced in a substantial number of patients. Notably, 76% had elevated TSH, indicating a trend toward hypothyroidism. Patients undergoing haemodialysis showed more severe hormonal disturbances than those under conservative treatment, including markedly lower TT3 and TT4 levels and significantly higher TSH concentrations. This suggests that the progression of kidney disease and dialysis further impacts thyroid regulation. The findings underscore the importance of routine thyroid function monitoring in CKD patients, especially those on dialysis, to enable early detection and appropriate management of metabolic and endocrine complications that may otherwise go unnoticed but contribute to increased morbidity.

## CONTRIBUTION OF AUTHORS

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