ISSN 2664-3987 (Print) & ISSN 2664-6722 (Online)
South Asian Research Journal of Medical Sciences
Abbreviated Key Title: South Asian Res J Med Sci

| Volume-8 | Issue-3 | May-Jun -2026 | **DOI: https://doi.org/10.36346/sarjms.2026.v08i03.005**

Original Research Article

An Examination of the Connection between Blood Indicators and the Thyroid Gland Function among Patients with Kidney Failure

Noor Mukhlis Hamid[1]*

[1]College of Nursing, Tikrit University, Tikrit, Iraq

***Corresponding Author:** Noor Mukhlis Hamid
College of Nursing, Tikrit University, Tikrit, Iraq

Article History
Received: 25.03.2026
Accepted: 18.05.2026
Published: 03.06.2026

Abstract: A diminished circulating thyroid hormonal concentrations, alterations to peripheral hormone metabolism, disrupted binding to carrier proteins, potential decrease in tissue thyroid thyroid hormone content, and an increased iodine store in the glands of the thyroid are some of the ways that chronic renal failure (CRF) impacts thyroid function. The goal of the research is to determine whether thyroid function and chronic renal failure are related. Furthermore, we attempted to investigate how CRF affected serum creatinine in relation to thyroid hormone levels (T3 and T4) with thyroid stimulated hormones (TSH). This study included 40 healthy individuals as a baseline group (20 male, 20 female) and 15 patients with chronic renal dysfunction (25 male, 25 female). The age range was between 20 and 60. Each including the two groups had measurements of T4, T3, TSH, urea, and creatinine. The findings showed a statistically significant decrease in T3 and T4, but an increase in TSH, urea, uric acid, as well as creatinine in the patient group relative to the control group.

Keywords: Chronic Renal Failure, Thyroid Gland, Urea, Creatinine.

INTRODUCTION

End-stage renal disease, also known as renal failure, is a medical condition marked by a marked deterioration in kidney function that makes it impossible for the kidneys to efficiently filter waste from the blood [1]. When the kidneys function at less than 15% of their normal capability, this disease develops. There are two types of kidney failure: chronic renal failure, which progresses gradually and is usually irreversible, and acute renal failure, a condition which happens unexpectedly, and may be repaired [2]. Leg edema, exhaustion, nausea, diminished appetite, and cognitive confusion are common signs of the illness. Uremia, hyperkalemia, and volume overload are among the consequences of both acute and chronic failure. Complications include anemia, hypertension, and cardiovascular disease might result from continuous failure [3].

In patients whose kidneys are no longer able to perform these functions normally, renal dialysis is a medical technique that involves eliminating excess water, solutes, and toxins from their blood [4]. Renal replacement therapy is the term used to describe this. 1943 was the first success in the field of dialysis. Acute renal damage, which is defined as an abrupt and rapid loss of kidney function, or chronic kidney failure that has advanced to stage 5 may require dialysis. When the glomerular filtration rate goes below 15% of the normal level and the creatinine clearance falls below 10 mL per minute, stage 5 chronic renal failure is diagnosed [5].

When a patient has symptomatic renal failure and their glomerular filtration rate (GFR) is less than 15 mL/min, chronic dialysis is advised. Starting dialysis at progressively higher estimated glomerular filtration rate (eGFR) values was a discernible trend between 1996 and 2008 [6]. All of the body's organ systems, including the heart, muscles, and brain, have been shown to be affected by thyroid disease. Renal function is also impacted by thyroid state. Thyroid hormone has an immediate impact on kidney function as well as systemic or local hemodynamic alterations [7]. Thyroid dysfunction affects renal the flow of blood, the glomerular filtration rate (GFR), tubular secretory and absorption capacity, electrolyte

pumps, and kidney shape. Both hyperthyroidism and significantly affect renal function [8]. Thyroid hyper- and hypofunction of the thyroid have been linked to a number of glomerulopathies [9]. However, there are few clinical research on thyroid gland disorders and its relationship to renal function, and little has been established about the impact of thyroid dysfunction on human renal function. The majority of research on thyroid hormone's effects on the kidney has been done on rats [10]. Furthermore, because changes in recorded renal function indicators are often within the normal range, the kidney's effects of thyroid hormones in humans might be mild and go unreported by clinicians. This study aimed to assess changes in renal function biochemical markers in individuals with thyroid gland disorders and to establish a correlation between these parameters and the patient's endocrine profile.

MATERIALS AND METHODS

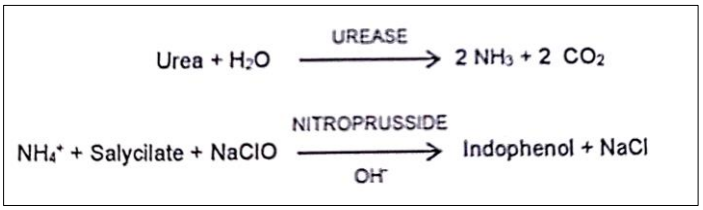
Sample Collection

The research population, which spanned in age from 20 to 60, began in January 2024 and ended in August 2025. There were 50 subjects in all, 40 of whom were in the control group. Five milliliters of blood were taken in a plane tube, allowed to clot for twenty to thirty minutes, and then centrifuged using a macro centrifuge for five to fifteen minutes at 3000 rpm. Fresh, non-hemolysis serum was then collected and stored in a deep freezer (-20 Co). The obtained serum was split into two tubes in order to measure changes in thyroid hormone levels and kidney function tests, which include: Those individuals underwent a thorough clinical evaluation that included physical tests and a history of illness. This assessment showed that all individuals with different levels of chronic renal failure and no prior history of thyroid dysfunction were included in the trial. Thyroid hormones are measured using radioimmune test methods using a hormonal kit from BIOMERIEYX in France [11].

Blood urea [12, 13]

Principle

Urease hydrolyzes urea to produce carbon dioxide and ammonia. When sodium nitroprusside is present as a coupling agent, the ammonia produced combines with alkali hypochlorite and salicylate of sodium to produce a green chromophore molecule [14]. The amount of urea in the specimen is directly correlated with the color's intensity.



Serum Creatinine [15]

Principle

Without any pre-treatment, the colorimetric reactions (Jaffe reaction) of ammonia with a standard alkaline picrate was evaluated kinetically at 490 nm (490-510). The invention of an initial-rate approach has increased this reaction's specificity, speed, and adaptability.

Analysis of Statistics

The statistical program (SPSS) was used for the statistical analysis, and one-way analysis of variance, or ANOVA for short, was used to compare the groups. The level of the statistical significance was set at ($P<0.05$).

RESULTS

Initial Anthropological and Biographical Features of Research Participant

All study groups had similar respondent mean ages. Patients with chronic kidney disease (CKD) shared an age range of 20 to 60 years, with a mean age of 38.12 ± 8.35 years and 40.43 ± 9.54 years for healthy controls. The three groups did not differ statistically significantly, according to a one-way ANOVA ($p = 0.2$).

Each of the three groups had a fairly equal distribution of sexes (Table 1). Males and females made up 25 (50.0%) of the CKD group. There were 33 males (51.6%) and 31 females (48.4%) in the control group, compared to 20 (50.0%) in the DKD group. There was no statistically significant difference in the sex distribution (χ^2 $p = 0.6$).

The groups' body mass index levels were likewise very similar. CKD patients had a mean BMI of 28.13 ± 3.54 kg/m², while controls had a mean BMI of 27.08 ± 3.95 kg/m². A one-way ANOVA showed no significant difference in BMI between the three groups ($p = 0.3$).

Table 1: Initial anthropometric and Demographic Features of Research Participants

Parameter	CKD (n=50)	Control (n=40)	P-value
Age (years)			
Mean ± SD	38.12 ± 8.35	40.43 ± 9.54	0.2*
Range	20-60	20-60	
Sex, n (%)			
Male	25 (50.0%)	20 (50.0%)	0.6**
Female	25 (50.0%)	20 (50.0%)	
BMI (kg/m²)			
Mean ± SD	28.13 ± 3.54	27.08 ± 3.95	0.3*
Range	20.76-39.65	20.23-39.06	

Data presented as Mean ± Standard Deviation. *One-way ANOVA **Chi-square test

The T3, T4, TSH, urea, and creatinine concentrations (mean ± SD) in the sera of patients with chronic renal failure and the control group are displayed in Table (2), where $P < 0.05$ was deemed significant.

Table 2: T3, T4, TSH, urea, and creatinine amounts in the serum of renal failure patients and the control group

Subjects	CKD (n=50)	Control (n=40)	P-value
T3(ng/ml)	0.76±0.03	2.23 ± 0.43	$P \leq 0.05$
T4(µg/ml)	4.12±1.67	9.21±1.76	$P \leq 0.05$
TSH (µIU/ml)	7.32±2.65	3.54± 0.43	$P \leq 0.05$
Urea (mg/dl)	124.76±23.76	36.65±6.23	$P \leq 0.05$
Creatinine (mg/dl)	8.21 ±1.87	0.87 ± 0.21	$P \leq 0.05$

Correlation Pattern in the Chronic Kidney Disease Group

Table 3 demonstrates the correlation profile among the investigated variables in the chronic kidney disease group. Serum T3 (ng/ml) displayed a significant positive correlation with T4(µg/ml) ($r = 0.28$, $p = 0.19$) . Additionally, T3 (ng/ml) showed a weak but significant positive correlation with serum TSH(µIU/ml) ($r = 0.28$, $p = 0.19$), as well as Serum T3 (ng/ml) displayed a significant positive correlation with Urea ($r = 0.25$, $p = 0.17$), finally Serum T3 (ng/ml) displayed a significant positive correlation with Creatinine ($r = 0.24$, $p = 0.19$).

Table 3: Correlation Analysis of DKK1, Galectin-3, and Clinical Parameters in CKD group

		T3 (ng/ml)	T4 (µg/ml)	TSH (µIU/ml)	Urea (mg/dl)	Creatinine (mg/dl)
T3 (ng/ml)	r	1.00	0.28	0.28	0.25	0.24
	p		0.19	0.19	0.17	0.19
T4 (µg/ml)	r	0.12	1.00		0.28	0.17
	p	0.53		1.00	0.19	0.20
TSH (µIU/ml)	r	0.02	0.14	1.00	0.28	0.66
	p	0.90	0.44		0.19	0.30
Urea (mg/dl)	r	-0.08	-0.36	0.20	1.00	0.22
	p	0.66	0.04	0.28		0.46
Creatinine (mg/dl)	r	0.40	0.13	0.23	0.10	1.00
	p	0.02	0.48	0.36	0.58	

DISCUSSION

Sex and gender differences in CKD epidemiology, progression rates, and outcomes are well documented; women tend to have a higher prevalence of early-stage CKD, whereas men often experience faster progression [16].

Broader work on sex and kidney health has further emphasized that hormonal factors, body composition, and differential exposure to risk factors can alter both disease course and biomarker expression [17].

By achieving a sex-matched cohort, the present study follows best-practice recommendations that demographic characteristics should be carefully controlled when evaluating inflammatory and metabolic markers in CKD populations, thereby improving the interpretability of biomarker–disease associations [18].

This confirms that the cohorts were effectively age-matched, thereby minimizing the influence of age on subsequent biochemical and clinical comparisons. Age is a well-recognized determinant of renal function, with glomerular

filtration rate (GFR) declining progressively with advancing age and older individuals exhibiting a higher burden of CKD and comorbidities [19]**.** Age also modifies biomarker profiles and inflammatory status, which is why controlling for it is crucial in CKD biomarker research. Recent methodological reviews have similarly stressed that inadequate adjustment or matching for age can mask true disease-related signals in biomarker studies [20].

The age profile of 20–65 years reflects a typical working-age adult population, where early identification of kidney dysfunction has important implications for long-term outcomes and prevention of progression to end-stage kidney disease (ESKD). However, this also means that the findings may not be directly generalizable to very elderly patients (>75 years), in whom CKD often coexists with frailty and a higher multi-morbidity burden [21].

According to this study, the concentration of T3 and T4 in the serum of patients with CRF was significantly lower than that of the control group ($P \leq 0.05$). Renal insufficiency has been shown to have a variety of effects on thyroid function, including altered peripheral hormone metabolism, disrupted protein binding, decreased tissue thyroid hormone levels, and iodine buildup in the thyroid gland [22]. Thyroxine binding-globin (TBG) insufficiency is one of the possible explanations for the decline in T4 [23]. Serum TSH levels in the CRF and control groups were assessed in this investigation. TSH was higher than normal in the CRF group. Despite the distortion of TSH in certain euthyroid patients with NTI who have considerable elevation of TSH due to underlying primary hypothyroidism, other writers interpret this rise as an indication of recovery from a hypothyroid condition [24]. Serum levels of urea, uric acid, and creatinine were significantly higher in CRF patients than in the control group. Due to a significant decrease in glomerular filtration rate (GRF), the ability to eliminate proteinaceous catabolites is compromised, which results in an increase in urea in renal failure. Reduced renal excretion also contributes to elevated serum creatinine levels [25].

GFR and T3 and T4 levels were found to be significantly correlated in two investigations conducted in Germany (2021) and Southern India (2022) [26, 27]. Our results, which demonstrate a decrease in GFR along with decreases in T3 and T4 levels, corroborate this association. According to a comparable cross-sectional study, hypothyroidism became more common in CKD patients as estimated GFR declined [7].

Impaired T4 interaction to serum carrier proteins may be the main cause of the low total T4 readings in patients with chronic renal failure. Numerous antagonists of T4 binding to serum carrier proteins have been found in CRF patients, which may be a factor in their lower T4 levels. According to this study, individuals with CRF had significantly lower FT3 and TT3 concentrations in their serum compared to the control group ($p<0.001$). These findings are consistent with those of earlier investigations [28-30].

According to other earlier studies, the increased excretion of bound and free T4 in the urine of chronic renal failure is responsible for the lower total T3 levels. Additionally, a reduction in the peripheral synthesis of T3 from T4 could be the cause [31].

CONCLUSION

Clinical practice frequently encounters chronic kidney disease (CKD), and people with CKD are more likely to acquire a number of illnesses, especially thyroid abnormalities. Examining the physiological connection between thyroid gland disorders and renal illness is essential given the high frequency of both conditions. Significant changes in function of the thyroid are frequently observed in CKD, especially in variations of FT3 as well as TSH levels. Notably, as the severity of renal disease increases, so does the chance of thyroid dysfunction. Patients with concurrent thyroid and renal disorders can significantly improve their overall care and survival with immediate interventions and constant surveillance.

Funding: No particular support from any financing organizations—governmental, private, or nonprofit—was available for this inquiry.

Authors' Contributions: All the aspects of this study were fully the responsibility of each author and they were involved in data analysis, writing and editing of the article.

REFERENCES

1. Mousa D, Alharbi A, Helal I, et al. Prevalence and associated factors of chronic kidney disease among relatives of hemodialysis patients in Saudi Arabia. *Kidney Int Rep.* 2021; 6(3): 817-820.
2. Yuasa R, Ohashi Y, Saito A, et al. Prevalence of hypothyroidism in Japanese chronic kidney disease patients. *Ren Fail.* 2020; 42(1): 572-579.
3. Srivastava S, Rajput J, Shrivastava M, et al. Correlation of thyroid hormone profile with biochemical markers of renal function in patients with undialyzed chronic kidney disease. *Indian J Endocrinol Metab.* 2018; 22(3): 316-320.

4. Al Hussaini HA, Al Sahlawi MA, Alhussain F, et al. Prevalence of hypothyroidism among dialysis patients in Eastern region, Saudi Arabia. *Cureus.* 2023; 15(1): e33807.
5. Cotoi L, Borcan F, Sporea I, et al. Thyroid pathology in end-stage renal disease patients on hemodialysis. *Diagnostics (Basel).* 2020; 10(4): 245.
6. Elzakil M, Gareeballah A, Gameraddin M. Sonographic assessment of thyroid gland in patients with chronic kidney disease undergoing hemodialysis. *Int J Diagn Imaging.* 2017; 5(1).
7. Alshammari F, Alhazaa S, Althemery A, et al. Prevalence of hypothyroidism among chronic kidney disease patients in security force hospital (SFH) in Saudi Arabia. *J Family Med Prim Care.* 2019; 8(10): 3313-3317.
8. Kashif M, Hussain MS, Anis M, Shah PK. Thyroid dysfunction and chronic kidney disease: a study among the northeastern population of India. *Cureus.* 2023; 15(5): e38700.
9. Ansari I, Kumar S, Acharya S, et al. Thyroid dysfunction at different stages of chronic kidney disease: a cross-sectional study at a rural teaching college in central India. *Cureus.* 2023; 15(8): e42130.
10. Matsuoka-Uchiyama N, Tsuji K, Sang Y, et al. The association between hypothyroidism and proteinuria in patients with chronic kidney disease: a cross-sectional study. *Sci Rep.* 2022; 12(1): 14999.
11. BioMerieux. *Vidas TSH Reagent Pack Insert (REF 30 404).* Marcy l'Etoile, France: BioMerieux; 2010.
12. Patton CJ, Crouch SR. Spectrophotometric and kinetics investigation of Berthelot reaction for the determination of ammonia. *Anal Chem.* 1977; 49(3): 464-469.
13. Fawcett JK, Scott JE. A rapid and precise method for the determination of urea. *J Clin Pathol.* 1960; 13(2): 156-159.
14. Chaney AL, Marbach EP. Modified reagents for determination of urea and ammonia. *Clin Chem.* 1962; 8(2): 130-132.
15. Henry RJ. *Clinical Chemistry: Principles and Technics.* 2nd ed. New York: Harper and Row; 1974. p. 525.
16. Hödlmoser S. *Sex-Specific Differences in Chronic Kidney Disease* [Dissertation]. Vienna: Medical University of Vienna; 2022.
17. Swartling O. *Progression of chronic kidney disease: risk factors, sex differences and intervention* [Dissertation]. Stockholm: Karolinska Institutet; 2025.
18. Ye B, et al. Interpretable machine learning model based on routine metabolic laboratory indices to identify advanced chronic kidney disease. *Front Endocrinol.* 2026; 17: 1776419.
19. Kushner PR, Mende CW. Estimated Glomerular Filtration Rate Slope, Chronic Kidney Disease Progression, and Pillars of Care in Patients With Diabetic Kidney Disease. *Diabetes Obes Cardiometab CARE.* 2026; 1(2): 278-287.
20. Ruperto M, Barril G. Nutritional status, body composition, and inflammation profile in older patients with advanced chronic kidney disease stage 4–5: a case-control study. *Nutrients.* 2022; 14(17): 3650.
21. Ebert T, et al. Inflammation and oxidative stress in chronic kidney disease and dialysis patients. *Antioxid Redox Signal.* 2021; 35(17): 1426-1448.
22. Inaba M, et al. Association of reduced free T3 to free T4 ratio with lower serum creatinine in Japanese hemodialysis patients. *Nutrients.* 2021; 13(12): 4537.
23. Liu X, et al. Partial thyroid hormone-binding globulin deficiency: a case report and literature review. *Diabetes Metab Syndr Obes.* 2023; 16: 2225-2232.
24. Jansen HI, et al. Hypothyroidism: the difficulty in attributing symptoms to their underlying cause. *Front Endocrinol.* 2023; 14: 1130661.
25. Wang X, et al. Challenges of serum creatinine level in GFR assessment and drug dosing decisions in kidney injury. *Adv Pharm Bull.* 2024; 14(4): 745.
26. Galeti EH, Reddy S, Conjeevaram J. Thyroid and lipid profile in chronic kidney disease in Southern India. *Int J Adv Med.* 2022; 9(3): 294-299.
27. Schultheiss UT, Steinbrenner I, Nauck M, et al. Thyroid function, renal events and mortality in chronic kidney disease patients: the German Chronic Kidney Disease study. *Clin Kidney J.* 2021; 14(3): 959-968.
28. Ali LK. The Effect of Chronic Renal Failure on Thyroid Hormone. *Iraqi J Pharm Sci.* 2010; 19(1).
29. Rotondi M, Netti GS, Rosti A, et al. Pretransplant serum FT3 levels in kidney graft recipients are useful for identifying patients with higher risk for graft failure. *Clin Endocrinol (Oxf).* 2008; 68(2): 220-225.
30. Jusufovic S, Hodzic E, Halilcevic A. Role of Renal Anemia in the Functional, Morphological and Autoimmune Thyroid Disorders in patient on Chronic Hemodialysis. *Med Arh.* 2011; 65(4): 228-232.
31. Alsaran K, Sabry A, Alshahhat H, et al. Free Thyroxine, Free Triiodothyronine and Thyroid-Stimulating Hormone Before and After Hemodialysis in Saudi Patient with End Stage Renal Disease. *Saudi J Kidney Dis Transpl.* 2011; 22(5): 917-921.