

Prevalence of Thyroid Dysfunction in Patients with Chronic Kidney Disease

Original Article

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ABSTRACT

Background: Chronic renal failure can affect thyroid function through various mechanisms, including reduced levels of circulating thyroid hormones, increased iodine retention within the thyroid glands, altered peripheral hormone metabolism, impaired transport protein binding, and possibly decreased thyroid hormone concentrations in tissue.

Aim of the Study: The study aimed to assess the prevalence of thyroid dysfunction at various stages of chronic kidney disease (CKD) and to highlight the significance of screening for thyroid dysfunction in patients with CKD.

Methods: This cross-sectional study involved 202 patients with clinical criteria of CKD (estimated glomerular filtration rate <60 ml/min/1.73m²). Patients were categorized into two groups: a dialysis-dependent group of patients ($n = 92$) and a non-dialysis-dependent group of patients ($n = 110$). A thyroid ultrasound examination was performed on each patient.

Results: There was a negative correlation between blood urea nitrogen (BUN) and thyroid stimulating hormone (TSH) in the non-dialysis group. At the same time, there was no correlation between [BUN and TSH], [creatinine and (TSH, free triiodothyronine (T3) and thyroxine (T4)] in the dialysis group. In the dialysis group, there was a positive association between [parathyroid hormone (PTH) and BUN] and [hemoglobin level and Free T4]. In both the dialysis and non-dialysis groups, there was a negative connection between PTH and calcium levels ($P \leq 0.05$).

Conclusions: The development of CKD is strongly associated with thyroid dysfunction, especially hypothyroidism.

Key Words: Chronic kidney disease, thyroid dysfunction, uremia, thyroid function.

Received: 26 February 2025, **Accepted:** 03 June 2025.

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ISSN: 2735-3540, Vol. 76, No. 3, Sep. 2025.

INTRODUCTION

The prevalence of chronic kidney disease (CKD) in Egypt is on the rise. Deterioration of kidney function over an extended period is the hallmark of chronic renal disease^[1].

The kidney plays a crucial role in facilitating the correct metabolism, breakdown, and excretion of various thyroid hormones. Hormone production, distribution, and excretion, as well as other hypothalamic-pituitary-thyroid axis levels, could be affected. Consequently, uremia is often associated with abnormal thyroid function testing^[2,3].

Patients with end-stage renal disease (ESRD) have affected hypothalamic-pituitary-thyroid hormone axis relationships, which affect peripheral thyroid hormone concentrations. Free triiodothyronine (FT3) levels are typically lower in ESRD patients because peripheral tissues convert less thyroxine (T4) into triiodothyronine

(T3). In these individuals, the thyroid gland produces normal amounts of T3, and clearance rates of T3 are either standard or lowered^[4].

Reducing FT3 levels in plasma is a hallmark of ESRD and is indicative of impaired peripheral T4 to T3 conversion^[5]. This abnormality is unrelated to the increased conversion of T4 to the metabolically inactive reverse T3 (rT3) since plasma rT3 levels are often within the normal range. This discovery distinguishes uremic patients from chronically sick patients, who also have a decreased conversion of T4 to T3 but an augmented generation of rT3 from T4.

The kidneys help the body excrete iodide normally. As renal failure progresses, iodide excretion decreases, which in turn causes an increase in thyroidal iodide absorption and a subsequent rise in plasma inorganic iodide concentration. Patients with uremic symptoms have reduced thyroid absorption of radiolabelled iodide due to the dramatic

increase in the intrathyroidal iodide pool that follows. The Wolff-Chaikoff effect describes the potential suppression of thyroid hormone synthesis triggered by an increase in total body inorganic iodide levels^[6, 7].

The thyroid glands' ability to synthesize and secrete thyroid hormone, as well as its ability to bind to carrier proteins, peripheral hormone metabolism, tissue thyroid hormone content, and iodine storage, are all negatively impacted by chronic renal failure^[8].

AIM OF THE WORK

The work aimed to identify the prevalence of thyroid dysfunction in CKD patients and establish a relationship between serum TSH, FT3, and FT4 and the different stages of CKD.

METHODS

This cross-sectional study involved 202 patients, aged > 18 years old, both sexes, with CKD patients (eGFR less than 60 ml/min/1.73m²).

ETHICAL APPROVAL

Following approval from Tanta University's Ethical Committee in Tanta, Egypt (Approval code: 34798/7/21). The research took place from September 2021 to October 2022. Written informed consent was obtained from the patients.

Patients who had a history of thyroid illness in their family, cancer, pregnancy, or were taking particular drugs, such as lithium, tricyclic antidepressants, antiepileptics, selective serotonin reuptake inhibitors, rifampicin, metformin, or amiodarone, and patients who had undergone radioactive iodine therapy were not included in the study.

Two groups of patients were established: the dialysis-dependent group ($n = 92$) and the non-dialysis-dependent group ($n = 110$).

Comprehensive medical assessments involved a thorough medical background check, a general physical examination, and a sequence of laboratory tests, including a complete Blood Count, serum ferritin, transferrin saturation, blood tests for kidney function [Blood Urea Nitrogen (BUN) and eGFR], creatinine levels, a urinary creatinine/albumin ratio (UACR), a lipid profile consisting of total cholesterol, high-density lipoprotein (HDL), low-density lipoprotein (LDL), cholesterol, and triglycerides, plus serum levels of calcium, phosphorus, sodium, and potassium using the Diestro RS232 Electrolytes device, as well as levels of HbA1c, serum TSH, free T3, free T4, and parathyroid Hormones (PTH) analyzed via chemiluminescent Immunoassay. Typical ranges for TSH, free T3, and free T4 are 0.5–5.5 mIU/L, 1.5–4.4 ng/dL, and 0.8–1.4 ng/dL, respectively. Imaging studies, including ultrasound of the thyroid, were conducted.

Thyroid ultrasound examination

In all instances, high-resolution B-mode thyroid ultrasounds were performed using an 18-5 MHz linear transducer, with patients lying in a supine position while maintaining regular breathing patterns and applying sufficient ultrasound gel.

If a patient's free T3 (4.0-8.3 pmol/L), free T4 (9.0-20.0 pmol/L), or TSH (0.25-5 mIU/L) levels were significantly higher or lower than the reference range, thyroid dysfunction was evaluated.

Kidney disease improving global outcomes (KDIGO) criteria were used to identify the CKD stages. If the levels of thyroid hormone are within the normal range, it was thought that the patient was euthyroid. If the TSH level is greater than 5 mIU/L and the free T3 and free T4 levels are less than or equal to 4.0 and 9.0 pmol/L, respectively, it was deemed that overt hypothyroidism was present. With free T3 and free T4 levels within the normal range and a TSH level higher than 5 mIU/L, subclinical hypothyroidism was diagnosed. Subclinical hyperthyroidism was defined as a TSH level of 0.25 mIU/L or below, with free T3 and free T4 values within the normal range^[9].

Estimated GFR (MDRD equation):

According to the National Kidney Foundation, chronic kidney disease (CKD) is characterized by an eGFR below 60 ml/min/1.73 m² for more than three months.

The estimated GFR was calculated by formulating the following: $GFR = 175 \times \text{standardized } S_{cr} - 1.154 \times \text{age} - 0.203 \times 1.212 \text{ (if black)} \times 0.742 \text{ (if female)}$, where S_{cr} is serum creatinine. Isotope dilution mass spectroscopy-standardized serum creatinine measurements were used to re-express this formula.

Statistical analysis

SPSS v26 (IBM Inc., Chicago, IL, USA) was employed to conduct the statistical analysis. To determine if the data was normally distributed, the Shapiro-Wilks test and histograms were employed. We used an unpaired Student's T-test to compare the quantitative parametric variables of the two groups, which were presented as means and standard deviations (SD). For quantitative non-parametric data, the Mann-Whitney U test was used to determine the median and interquartile range (IQR). The qualitative variables were examined using the Chi-square test and were expressed as percentages and frequencies. Measurements of TSH, FreeT3, and Free T4, as well as BUN and creatinine, were correlated in both groups. Statistical significance was determined by a two-tailed P value that was lower than 0.05.

RESULTS

The two groups did not differ significantly in terms of age, sex, hypertension (HTN), diabetes mellitus (DM),

systolic blood pressure (SBP), and diastolic blood pressure (DBP). (Table1)

Table 1: Sociodemographic characteristics, HTN and DM disorders, SBP and DBP of both groups.

	Non-dialysis group (n=110)	Dialysis group (n=92)	Test of sig.	P
Age (years)	51.33 ± 14.95	46.67±15.12	t=2.458	0.627
Sex				
Male	28(25.5%)	38(41.3%)	$\chi^2=2.861$	0.091
Female	82(74.5%)	54(58.7%)		
HTN	34(30.9%)	36(39.1%)	$\chi^2=1.154$	0.282
DM	76(69.1%)	56(60.9%)	$\chi^2=1.150$	0.283
SBP (mmHg)	120(110 – 140)	120(107 – 132)	U=1131.50	0.357
DBP (mmHg)	80 (70 – 90)	80 (70 – 90)	U=1170.50	0.509

The data are displayed as frequency (%) or mean (IQR) ± SD; U: Mann-Whitney test; t-test: independent sample t-test, significant *p* value < 0.05; and ~2: Chi-square test; DM stands for diabetes mellitus; and HTN for hypertension; Systolic blood pressure is known as SBP; while diastolic blood pressure is known as DBP.

The dialysis group had substantially lower levels of BUN, creatinine, HB, and cholesterol than the non-dialysis group. Both groups showed no significant differences in WBCs, Platelets, HbA1C, ferritin, transferrin saturation, calcium, potassium and parathyroid hormone, frequency of calcium disorders (hypocalcemia, normal calcium levels,

and hypercalcemia, TSH, Free T3, Free T4 and frequency of thyroid dysfunctions. Phosphorus, triglycerides, low-density lipoprotein, and HDL were all considerably higher in the dialysis group than in the non-dialysis group (*P* < 0.05). (Table 2)

Table 2: Comparison between the non-dialysis group and dialysis group regarding laboratory investigations.

	Non-dialysis group (n=110)	Dialysis group (n=92)	U	P
BUN (mg/dL)	121.00 (94 – 155)	78.00(59.75 - 110)	508.00	0.000*
Creatinine(mg/dL)	4.60 (3.20 – 5.80)	9.50(7.14 - 10.40)	404.00	0.000*
UACR (mg/g)	452(321.5 - 735.5)	-----	-----	-----
Hb(gm/dl)	9.5 (8.6 -10.08)	9 (8.08 – 12.6)	4225.5	0.044*
WBCs (× 10 ⁹ /L)	5.5(4.6 - 6.3)	6.45(4.5 - 9.03)	1456.5	0.192
Platelets (× 10 ⁹ /L)	190(154 - 250)	193.5 (135.25 - 312)	1350.00	0.564
HbA1C (%)	5.6(5.20 – 8.10)	5.5 (5.17 – 6.82)	1179.00	0.559
Ferritin(ug/L)	33(15.5 - 145)	26(19.25 - 74)	1303.0	0.798
Transferrin saturation (%)	22(19 - 35)	20.5(17.25 - 29.75)	1461.5	0.181
Cholesterol level(mg/dL)	190 (178 – 211.25)	185 (174 – 198)	4230.5	0.045*
LDL (mg/dL)	99 (86 - 132)	110 (99 - 141)	4110	0.022*
HDL (mg/dL)	40 (32.25 - 45)	42 (36 – 45.25)	4204.5	0.038*
Triglycerides (mg/dL)	160(119.5 - 228)	194.5(160 - 263.5)	1690.5	0.003*
Calcium(mg/dL)	7.80(7.10 -8.20)	7.65(7.20 – 8.12)	1260.50	0.975
Potassium(mg/dL)	4.30(3.80 – 4.90)	4.30(3.87 – 5.00)	1233.50	0.830
Phosphorus(mg/dL)	4.60(3.20 – 5.30)	6.20(5.27 – 9.05)	423.00	0.000*
Para-thyroid hormone (pg/mL)	315.00 (152.50–537.00)	316.50(136.25–449.62)	626.50	0.977
TSH (mIU/L)	2.01 (1.07-2.84)	2.38(1.20 – 3.52)	1105.50	0.277
Free T3(pg/mL)	2.2			

Neither group differed significantly from the other in terms of the frequency of calcium disorder or thyroid dysfunctions. (Table 3)

Table 3: Frequency of calcium disorders and thyroid dysfunctions in both groups.

	Non-dialysis group (n=110)	Dialysis group (n=92)	U	P
Frequency of calcium disorders				
Hypocalcemia	68(61.8%)	56(60.9%)	MC= 0.225	0.798
Normal calcium levels	38(34.5%)	34(37.0%)		
Hypercalcemia	4(3.6%)	2(2.2%)		
Frequency of thyroid dysfunctions				
Hypothyroidism	8(7.3%)	8(8.7%)	1.836	0.597
Isolated low T3	36(32.7%)	40(43.5%)		
Normal	52(47.2%)	38(41.3%)		
NTI	6(5.5%)	4(4.3%)		
Subclinical hypothyroidism	6(5.5%)	2(2.2%)		
Subclinical hyperthyroidism	2(1.8%)	0(0.0%)		

Data are presented as frequency (%); *: Significant *p-value* (≤ 0.05); U: Mann-Whitney test; MC: Monte Carlo test; NTI: Non-thyroidal illness.

In the non-dialysis group, BUN and TSH were negatively correlated with each other. In contrast, the dialysis group did not show any link between [BUN and TSH], [creatinine and (TSH, Free T3 and Free T4)]. In both groups, there was no correlation between [BUN and (Free T3 and Free T4)], [PTH and creatinine and BUN], and the other variables. In the dialysis group, PTH and BUN were positively correlated. (Table 4)

Table 4: Correlation between [TSH, FreeT3, Free T4, and (BUN and creatinine)] and between [PTH, BUN, and creatinine] between both groups.

	Non-dialysis group		Dialysis group	
	r_s	P	r_s	P
BUN				
TSH (mIU/L)	-0.263	0.043*	-0.034	0.823
Free T3(pg/mL)	-0.060	0.663	-0.230	0.125
Free T4(ng/dL)	0.105	0.451	-0.150	0.319
Creatinine (mg/dL)				
TSH (mIU/L)	0.015	0.915	-0.098	0.515
Free T3(pg/mL)	0.084	0.544	-0.168	0.264
Free T4(ng/dL)	-0.022	0.876	-0.093	0.537
Para-thyroid hormone				
BUN (mg/dL)	0.178	0.291	0.382	0.026*
Creatinine (mg/dL)	-0.037	0.829	0.150	0.398

rs: Spearman correlation test; *Significant *p-value* ≤ 0.05 ; TSH: thyroid stimulating hormone; T3: triiodothyronine; T4: thyroxine; BUN: blood urea nitrogen.

Neither the dialysis nor the non-dialysis groups showed any correlation between [Hb level and (TSH and free T3)], [calcium level and (TSH, free T3, and free T4)], or [Hb level and free T4] in the non-dialysis group. Hb level and free T4 were positively correlated in dialysis groups. Both groups showed a negative correlation between PTH and calcium levels ($P \leq 0.05$). (Table 5)

Table 5: Correlation between [TSH, FreeT3, Free T4, and hemoglobin level] and [TSH, FreeT3, Free T4, PTH and calcium level] between both groups

	Non-dialysis group		Dialysis group	
	r_s	P	r_s	P
Hemoglobin level (gm/dl)				
TSH (mIU/L)	-0.149	0.341	-0.019	0.915
Free T3(pg/mL)	0.175	0.261	0.078	0.660
Free T4(ng/dL)	-0.134	0.396	0.348	0.044*
Calcium level (mg/dL)				
TSH (mIU/L)	-0.164	0.231	-0.091	0.549
Free T3(pg/mL)	-0.089	0.520	0.115	0.44

DISCUSSION

Because CKD is a more enduring form of kidney disease than acute renal injury, it can be distinguished by the fact that the decline in kidney function persists for a period exceeding three months. A globally acknowledged public health issue, CKD affects 5–10% of people worldwide^[10].

Our study reveals that the dialysis group exhibited substantially lower levels of BUN and creatinine than the non-dialysis group. UACR ranged from 9 to 980 mg/g with a mean value ($\pm$ SD) of 481.09 ($\pm$ 272.92) and with a median (IQR) of 452 (321.5 - 735.5) mg/g in a non-dialysis group. This is supported by *Canaud et al.*^[11], who noted lower creatinine, BUN, and UACR levels after dialysis. The explanation for the lower creatinine levels in the dialysis group is that creatinine is a low-molecular-weight substance that passes through the dialysis membrane and is washed away from the blood^[11].

Our study showed that the Dialysis group exhibited substantially higher levels of triglycerides and phosphorus than the non-dialysis group. *Huang et al.*^[12] reported similar results, noting that 40% of dialysis patients had high phosphorus levels. Additionally, *Yin et al.*^[13] revealed that dialysis patients had higher phosphorus levels compared to those without dialysis. Conversely, *Palmer et al.*^[14] discovered that following four months of hemodialysis with a polyamide membrane, lipid parameters improved. Cholesterol and serum triglyceride levels fell, but HDL cholesterol rose substantially.

Regarding our results, neither group showed a significant difference from the other in terms of the frequency of calcium disorders (hypocalcemia, normal calcium levels, and hypercalcemia). *Almutary et al.*^[15] discovered that calcium levels did not differ significantly between dialysis patients and those who did not need dialysis.

The results showed that there was no substantial difference between both groups concerning TSH, Free T3, and Free T4. Additionally, there was no considerable difference in the frequency of thyroid dysfunctions between both groups. *Khatiwada et al.*^[9] discovered that CKD patients undergoing dialysis had a higher prevalence of thyroid dysfunction compared to CKD patients who were not on dialysis. However, *Mohamedali et al.*^[16] observed that patients on hemodialysis were euthyroid compared to CKD patients without dialysis, who were mostly subclinical hypothyroidism. Low total T4 and high FT4 levels are standard in hemodialysis patients with euthyroid because heparin inhibits T4 protein binding, which increases the FT4 fraction.

Regarding the correlation between TSH, FT3, FT4, and BUN among non-dialysis and dialysis groups, there was a negative correlation between BUN and TSH in

the non-dialysis group. At the same time, there was no correlation between BUN and TSH in the dialysis group. In both groups, BUN was not correlated with (FT3 and FT4). *Shamsadini et al.*^[17] found that TSH had a negative correlation with BUN in non-dialysis patients. Still, in the dialysis group, there was no association between BUN and TSH, possibly because hemodialysis has a positive feedback influence on thyroid gland function. As a feedback mechanism, the release of thyroid hormones is inhibited when the levels of creatinine and BUN rise due to an increase in catabolic processes caused by thyroid gland hormones. A hypothalamic-pituitary axis depression, which in turn reduces TSH receptor expression, is one possible mechanism by which the thyroid becomes dysfunctional in chronic kidney disease (CKD). Contrary to our results, *Salih et al.*^[18] found that CKD patients were hypothyroid and showed that BUN had a negative correlation with TSH and a positive correlation with free T3 and T4.

Our study found no correlation between creatinine and TSH, FT3, and FT4 in both non-dialysis and dialysis groups. *Inaba et al.*^[19] found that creatinine had no correlation with TSH and FT4 in dialysis patients, although they obtained different results from ours, as they noted that creatinine positively correlated with FT3. Moreover, *Sadaria & Vasava al.*^[20] concluded that serum creatinine and free thyroid hormones were inversely correlated.

Regarding the results, PTH and BUN showed a positive correlation in the dialysis group but a negative correlation in the non-dialysis group. In their study of dialysis patients, *Suhail et al.*^[21] found a favorable connection between parathyroid hormone and BUN. Even though *Bover et al.*^[22] noted there was a positive correlation of PTH with BUN in non-dialysis dependent CKD.

Our study reveals that there was no correlation between hemoglobin level and TSH and FT3 in both non-dialysis and dialysis groups. Dialysis groups showed a favorable association between HB levels and FT4, whereas non-dialysis groups showed no such correlation. These outcomes follow *Kamal et al.*^[23], who demonstrated that hemoglobin level and TSH, FT3, and FT4 did not correlate significantly in non-dialysis CKD patients. Additionally, *Kim et al.*^[24] discovered a positive correlation between serum hemoglobin and FT4 and an inverse relationship with eGFR. This suggests that thyroid hormones regulate red blood cell production through multiple pathways, including erythropoietin production and responsiveness, as well as iron transport and utilization.

Our results demonstrated that calcium levels were unrelated to (TSH, FT3, and FT4) in both groups. Both the non-dialysis and dialysis groups showed a negative connection between PTH and calcium levels. *Carneiro Dias et al.*^[25] revealed that PTH was inversely correlated with serum calcium in CKD non-dialysis patients. Moreover, hemodialysis patients' serum calcium levels

were found to be negatively correlated with PTH, as noted by *Jean et al.*^[26].

One of the study's limitations was that it was conducted in a single location, which may have introduced crowd bias. Since excessive iodine intake or iodine shortage may exacerbate thyroid conditions, particularly subclinical hypothyroidism, the participants' iodine status was not examined. It is challenging to rule out all confounding variables and prove a causal association, as is the case with all observational studies.

CONCLUSIONS

Thyroid dysfunction, especially hypothyroidism, is closely linked to CKD progression. In CKD patients, those on dialysis showed higher phosphorus and TG levels than non-dialysis patients. A negative correlation between TSH and BUN was observed in the non-dialysis group. In dialysis patients, Hb and free T4 were positively correlated, as were PTH and BUN. In both groups, Ca and PTH levels were negatively correlated.

ACKNOWLEDGEMENT

Nil

CONFLICT OF INTERESTS

The authors have no financial or proprietary interests in any material discussed in this article.

AUTHORS' CONTRIBUTION

All authors contributed to the study conception and design. Material preparation, data collection and analysis were performed by [ARH] and [SAK]. The first draft of the manuscript was written by [GAS] and [AAS] and all authors commented on previous versions of the manuscript. All authors read and approved the final manuscript.

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انتشار اختلال وظائف الغدة الدرقية بين مرضى الفشل الكلوي المزمن

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المقدمة: يمكن أن يؤثر الفشل الكلوي المزمن على وظيفة الغدة الدرقية من خلال آليات مختلفة، مثل انخفاض مستويات هرمونات الغدة الدرقية المنتشرة، وزيادة احتباس اليود داخل الغدة الدرقية، وتغيير استقلاب الهرمونات المحيطة، وضعف ربط بروتين النقل، وربما انخفاض تركيزات هرمون الغدة الدرقية في الأنسجة.

الهدف: تهدف الدراسة إلى تقييم مدى انتشار خلل الغدة الدرقية في مراحل مختلفة من مرض الكلى المزمن توضيح مدى أهمية فحص خلل الغدة الدرقية لدى المرضى الذين يعانون من مرض الكلى المزمن.

أجريت هذه الدراسة المقطعية علي ٢٠٢ مريض يعانون من المعايير السريرية لمرض الكلى المزمن (معدل الترشيح الكبيبي المقدر > ٦٠ مل / دقيقة / ١,٧٣ م^٢). تم تصنيف المرضى إلى مجموعتين: مجموعة من المرضى المعتمدين على غسيل الكلى (ن = ٩٢) ومجموعة من المرضى غير المعتمدين على غسيل الكلى (ن = ١١٠). تم إجراء فحص الغدة الدرقية بالموجات فوق الصوتية على كل مريض.

النتائج: كان هناك ارتباط سلبي بين نيتروجين اليوريا في الدم وهرمون الغدة الدرقية في المجموعة التي لا تعتمد علي غسيل الكلى بينما لم يكن هناك ارتباط بين [نيتروجين و هرمون لغدة الدرقية]، [الكرياتينين و (هرمون الغدة الدرقية، ثلاثي يودوثيرونين حر وهرمون الثيروكسين) في مجموعة غسيل الكلى. كان هناك ارتباط إيجابي بين [هرمون الغدة الدرقية و اليوريا نيتروجين في الدم] و [مستوى الهيموجلوبين و هرمون الثيروكسين الحر] في مجموعة غسيل الكلى. في كل من مجموعات غسيل الكلى وغير غسيل الكلى، كان هناك اتصال سلبي بين مستويات هرمون الغدة الجار درقية والكالسيوم.

النتيجة: يرتبط تطور مرض الكلى المزمن ارتباطاً وثيقاً بخلل الغدة الدرقية، وخاصة قصور الغدة الدرقية. بالنسبة للمرضى الذين يعانون من مرض الكلى المزمن، نقترح إجراء اختبار روتيني لوظيفة الغدة الدرقية للمساعدة في التشخيص المبكر لمشاكل الغدة الدرقية وبالتالي تحسين رعاية المرضى. ويرتبط تطور مرض الكلى المزمن بقوة بخلل الغدة الدرقية، وخاصة قصور الغدة الدرقية. بالنسبة للمرضى الذين يعانون من مرض الكلى المزمن، نقترح إجراء اختبار روتيني لوظيفة الغدة الدرقية للمساعدة في التشخيص المبكر لمشاكل الغدة الدرقية وبالتالي تحسين رعاية المرضى.