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ORIGINAL ARTICLE

Evaluation of Thyroid Hormones Level in Chronic Kidney Disease Patients

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Abstract

Background: **Chronic-kidney disease (CKD) is a global issue worldwide. The hypothalamus-pituitary-thyroid axis, peripheral-circulation, and production of thyroid-hormone release/or excretion are all affected by the etiology of thyroid dysfunction in CKD patients.**

Aim: **To measure the levels of thyroid stimulating hormone (TSH), triiodothyronine (T3), thyroxine (T4), in CKD patients.**

Patients and methods: **The control group in this study, which included 60 CKD patients from Egypt, consisted of 30 healthy volunteers (patients without CKD). There were three groups of participants: group (1) 30 patients with CKD in group (2) 30 CKD patients on dialysis regularly; group 3: 30 healthy people without CKD.**

Results: **There was no age-related difference between the studied groups that was statistically significant (*P* value greater than 0.05). Compared with female patients with CKD ESRD (8 patients, 26.7%) in the conservative group (60%) and control group (18 patients, 60%). There was a significant (*P* value 0.001) difference between the studied groups in Hb, urea, creatinine, thyroid stimulating hormone, free triiodothyronine (FT3), free triiodothyronine status, and free thyroxine (FT4) and free thyroxine status.**

Conclusion: **The prevalence of thyroid disorders was higher in the end-stage of CKD than in the early-stage, and it was higher in CKD patients on hemodynamic support than in healthy individuals.**

Keywords: Chronic kidney disease, Early-stage, End-stage, Hemodialysis, Thyroid stimulating hormone

1. Introduction

Abnormalities in the structure or function of the kidney that last longer than 3 months,[1] and albumin levels greater than 30 mg per A glomerular filtration rate (GFR) of less than 15 ml/min or the chronic kidney disease (CKD) stage are the two criteria for end-stage renal disease (ESRD).[2] Progressive CKD characterized by a high risk of primary and subclinical hypothyroidism due to decreased deiodinase activity, which prevents the conversion of thyroxine (T4) to triiodothyronine (T3) in the peripheral tissues.[3] Multiple uremic toxins, malnutrition, chronic metabolic acidosis, and the anticoagulant heparin used in hemodialysis all inhibit T4 protein binding in advanced CKD, which results in the accumulation of inorganic iodide.[4] In addition to chronic inflammation and hepatitis C infection, amiodarone, steroids, and -blockers have all been implicated.[5] This study aim to determine thyroid hormone function levels in CKD patients.

2. Patients and methods

The Internal Medicine Department's Outpatient Clinics, Inpatient Unit, and Hemodialysis Unit at Al-Azhar University's Assiut Faculty of Medicine were the patients of a case-control-study.

60 CKD patients were enrolled Clinic of the Internal medicine department at Al-Azhar University. 30 healthy volunteers served as the control patients.

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This study was carried out in 2021, from June to December. There were three groups of participants: group 1: 30 patients with CKD in group 2: 30 kidney-patient who are always on dialysis; group 3: 30 healthy individuals without CKD. Participants over the age of 18 with CKD were included. People under 18, people undergoing renal transplantation, people with concurrent cancers, people taking medications like Amiodarone, which can cause thyroid disorders, people who know they have thyroid disorders, and pregnant women were not included in the study.

Each patient received the following treatments: history and clinical examination: obtaining a comprehensive medical history that includes information about diabetes, heart disease, and renal failure. The abdomen, heart, and chest were all examined for signs of renal disease. Observations made in the Lab: From each participant's 5 ml of venous blood, two samples were taken; after being centrifuged out of a typical traveler tube, the sample's first 3 ml were used for biochemical research. An anticoagulant-filled tube supplied the remaining 2 mL; EDTA and centrifuged right away; Serum creatinine was measured with a chemistry auto-analyzer from Roche Diagnostics, USA, called the COBAS 501. Two ml of blood were thoroughly mixed with EDTA (1 mg/ml blood) using the Erma Automated Blood Count Machine (Tokyo, Japan) for a comprehensive blood count. Thyroid hormone levels can be measured: TSH, Free T3 and Free T4 were assayed using ELSA technique. The National Academy of Clinical Biochemistry (NACB) guidelines for thyroid disease laboratory diagnosis and monitoring were used to classify thyroid abnormalities.[6] A decreased eGFR of 60 ml/min/1.73 m^2 for more than 3 months was considered to be CKD. The study was approved by Al-Azhar, the Faculty of Medicine, and the Internal Medicine Committees.[7] The university's committee on ethics. Statistical Program for the Social Sciences (SPSS) version 18.0 was used to analyze the data. The mean minus the standard deviation (SD) was utilized for the representation of quantitative data. Frequency and percentage were used to describe the qualitative data. The Chi-square (χ^2) test's significance was utilized to compare the proportions of two qualitative parameters. *P*-value: Probability A *P*-value that was less than 0.05 was deemed significant, a *P* value that was less than 0.001 was deemed highly significant, and a *P*-value that was greater than 0.05 was deemed insignificant. A *P* value that was greater than 0.05 was deemed insignificant.

3. Results

Female patients in CKD on conservative group (18 patients, 60%) and control group (18 patients, 60%) when compared with CKD ESRD (8 patients, 26.7%). Highly-statistical-significant difference (*P* value < 0.001) among included patients regards; Hb, urea, create, TSH, free triiodothyronine (FT3), FT3 status, free thyroxine (FT4) and FT4 status Tables 1–5.

4. Discussion

Any impairment in thyroid function can either cause or exacerbate kidney disorders.[8] TSH, T3, T4, TSH levels were the primary focus of our investigation among CKD patients. Our research demonstrated this; There was no age-related difference between the studied groups that was statistically significant (*P* value greater than 0.05). Out of 1000 ESRD patients in the Aswan governorate in Upper Egypt, 605 (60.5%) men and 395 (39.5%) women were receiving hemodialysis on a regular basis, according to Hall et al.[9] Elshimy et al.[10] included 641 men in the Menoufia governorate who were on regular HD for ESRD, or 32% of men on dialysis. 61.6% of the study's 641 participants were men and 38.4% were women. Patients receiving hemodialysis was 571 pmp (0.057%) in another recent study from our region. Eisenberg et al.[11] found that 60% of patients were male and 39% were female. In terms of FT3 status, there was a difference statistically significant (*P* value 0.001). Additionally, there was a FT4 status difference was statistically significant (*P* value 0.05). The evidence provided by Kamal et al.,[12]

Table 1. Post-Hoc test for multiple comparisons between demographic data.

	Groups						Stat. test	*P*-value
	CKD ESRD ($n = 30$)		CKD on conservative TTT ($n = 30$)		Control ($n = 30$)			
Age (y)								
Mean ± SD	57 ± 14.5		50.3 ± 20.5		49.2 ± 18.2		KW = 2.09	0.351 NS
Sex								
Male	22	73.3%	12	40%	12	40%	$\chi^2 = 8.9$	0.012 S
Female	8	26.7%	18	60%	18	60%		

Table 2. Post-Hoc test for multiple comparisons between laboratory data.

	Groups			KW	*P*-value
	CKD ESRD ($n = 30$)	CKD on Conservative TTT ($n = 30$)	Control ($n = 30$)		
Hb (g/dl)					
Mean ± SD	10.4 ± 2.1	9.3 ± 1.5	13.2 ± 1.2	48.01	<0.001
Urea (mg/dl)					
Mean ± SD	127.6 ± 16.3	88.3 ± 51.6	28.5 ± 7.1	62.2	<0.001
Creat (mg/dl)					
Mean ± SD	8.0 ± 2.0	2.9 ± 1.5	1.01 ± 0.16	183.5	<0.001

Table 3. Post-Hoc test for multiple comparisons between thyroid stimulating hormone.

	Groups						Stat. test	*P*-value
	CKD ESRD ($n = 30$)		CKD on Conservative TTT ($n = 30$)		Control ($n = 30$)			
TSH (uIU/ml)								
Mean ± SD	1.22 ± 0.56		2.5 ± 0.88		2.2 ± 0.9		KW = 29.8	<0.001
TSH status								
Low	3	13.3%	0	0%	0	0%	$\chi^2 = 8.3$	0.015
Normal	27	86.7%	30	100%	30	100%		

who conducted a cross-sectional study on 360 CKD patients, supports the current study's findings. The participants in the study were, on average, 44.1 and 16.4 years old. 144 CKD patients (40, 43.3, and 16.6%, respectively) were in stages 3, 4, and 5. 38.6% of CKD patients had thyroid-disturbance. Thyroid dysfunction was significantly more common in stage 4 and 5 CKD patients than it was in stage 3 patients. Our findings are comparable to those of Song et al.,[13] who found that as the stage of CKD advanced, the prevalence of low T3 patients increased. According to our findings. Thirty healthy, age- and sex-matched controls and 37 CKD patients on or off of hemodialysis were included in the Stremke and Gallant[14] case control study. Hemodialysis was used by 10 of the 37 CKD patients in this study, while nondialysis treatment was used by 27. Three (11%) of the 27 CKD patients who did not receive hemodialysis and three (30%) of the 10 CKD patients who did received it both had hypothyroidism. CKD was present in 26 men and 29 women for less than 5 years. 45 healthy individuals of equal age and gender served as controls. The study patients had significantly lower total T3 and total T4 serum levels than the controls. Rajeev et al.[15] found that TSH levels in CKD patients were significantly higher than those in controls. Rhee et al.[16] conducted a case–control study with 30 patients with ESRD on maintenance HD. The HD group had significantly lower serum FT3 levels (*P* 0.001) than the control group and the CKD group. Serum levels of TSH also did not differ significantly between any of the groups ($P = 0.765$). According to Pogoryelova *et al.*[17] they measured serum levels of urea, creatinine, total T4, total T3, FT4, FT3, and TSH in 40 healthy individuals and 80 patients with various degrees of chronic renal failure. They concluded that uremia is associated with thyroid disorders due to impaired hormone degradation. On the other hand, Benvenga et al.[18] looked at 905 nondialysis participants and found that eGFR increased the prevalence of FT3 or T3, with the lowest level

Table 4. Post-Hoc test for multiple comparisons between free triiodothyronine.

	Groups						Stat. test	*P*-value
	CKD ESRD ($n = 30$)		CKD on Conservative TTT ($n = 30$)		Control ($n = 30$)			
FT3 (pg/ml)								
Mean ± SD	2.07 ± 0.64		2.29 ± 0.65		2.8 ± 0.41		KW = 26.1	<0.001
FT3 status								
Low	14	46.7%	6	20%	0	0%	$\chi^2 = 19.02$	<0.001
Normal	16	53.3%	24	80%	30	100%		

Table 5. Post-Hoc test for multiple comparisons between free thyroxine.

	Groups						Stat. test	*P*-value
	CKD ESRD ($n = 30$)		CKD on Conservative TTT ($n = 30$)		Control ($n = 30$)			
FT4 (pg/ml)								
Mean ± SD	1.06 ± 0.17		1.11 ± 0.3		1.22 ± 0.23		KW = 26.1	<0.001 HS
FT4 status								
Low	0	0%	6	20%	0	0%	$\chi^2 = 12.8$	0.002 HS
Normal	30	100%	24	80%	30	100%		

occurring in CKD5 (*P* 0.01). In terms of FT4, T4, or TSH, there were no groups that were significantly different ($P > 0.05$). According to Kamal et al.,[19] patients with CKD who have ESS, most frequently low T3 syndrome, have a high morbidity rate.

4.1. Conclusion

The prevalence of thyroid disorders was higher in the end stage of CKD than in the early stage, and it was higher in CKD patients on hemodynamic support than in healthy individuals.

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Conflicts of interest

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