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ORIGINAL ARTICLE

Evaluation of Thyroid Function Tests in Elderly Patients with Chronic Kidney Disease

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ABSTRACT

Background: Thyroid dysfunction is prevalent among elderly patients who have chronic kidney disease (CKD) due to metabolic and hormonal disturbances. This study evaluated thyroid dysfunction among elderly CKD patients with declining glomerular filtration rate (GFR). **Methods**: This prospective case-control study was conducted at Zagazig General Hospital for one year. Sixty subjects (≥65 years) were divided into three groups: control (n=20), CKD stage III/IV (n=20), and hemodialysis (HD) (n=20). Clinical assessment, routine labs, eGFR calculation, and thyroid function tests (T3, T4, fT3, fT4, TSH) were performed via enzyme-linked immunoassay (ELISA).

Results: CKD and HD had significantly higher blood pressure than controls ($p<0.05$). Hemoglobin concentrations showed a decrease among CKD as well as HD groups ($p<0.001$). Fasting glucose and triglyceride concentrations were found to be raised in the CKD and HD groups ($p<0.05$). Liver function markers (total protein, albumin, AST, ALT) were considerably lower among the patient group ($p<0.05$). Serum creatinine and urea were found significantly higher, and a significant decline ($p<0.001$) in eGFR groups with CKD and HD. T3 and fT3 were considerably lower in CKD and HD groups ($p<0.001$); however, non-statistical significant differences were found for TSH, T4, and fT4. CKD and HD subjects had higher prevalence rates of subclinical hypothyroidism and NTIS, displaying correlation with eGFR and hemoglobin, creatinine, and urea levels. **Conclusion**: The study demonstrates a significant association between declining renal function and reduced T3/fT3 levels in elderly CKD patients, particularly those undergoing hemodialysis. The findings underscore a higher prevalence of subclinical hypothyroidism and non-thyroidal illness syndrome in advanced stages of CKD, emphasizing the clinical importance of regular thyroid function monitoring.

Keywords: Thyroid Function, Elderly Patients, Chronic Kidney Disease

INTRODUCTION

Chronic kidney disease (CKD) is a condition that compromises the structure and function of the kidneys. The heterogeneity in disease manifestation is influenced by factors such as etiology, pathological characteristics, severity, and progression rate [1].

Aging is an independent risk factor for renal function decline, as glomerular filtration rate (GFR) physiologically decreases with advancing age due to structural and functional changes in the kidneys, such as nephron loss, glomerulosclerosis, and vascular alterations. Epidemiological studies show that after age 40, GFR declines by approximately 1 mL/min/1.73 m² per year, even in healthy individuals, contributing to the increased prevalence of CKD among the elderly [2,3].

CKD poses a substantial challenge to the health of the public at large around the globe because of its rising incidence and prevalence, which, in turn, increase morbidity and mortality. CKD affects about 10% of the general population worldwide and causes millions of deaths every year due to a lack of access to affordable treatment. The principal sequelae of CKD, regardless of its underlying etiology, include the progression to end-stage renal disease, complications from varying degrees of kidney function, and heightened risk of cardiovascular events [2].

Chronic kidney disease includes renal impairment or a GFR that has remained less than 60 mL/minute/1.73 m² for at least 3 months, irrespective of the underlying etiology. GFR is traditionally calculated according to standardized serum creatinine measurements, using the Cockcroft-Gault formula and the Modification of Diet in Renal Disease (MDRD) equation [3].

The aging process also affects thyroid physiology. Age-related changes include hypothalamic-pituitary-thyroid (HPT) axis alterations, decreased thyroid hormone production, and reduced peripheral conversion of T4 to T3. Elderly individuals are at increased risk for both overt and subclinical thyroid dysfunction, with a higher prevalence of non-thyroidal illness syndrome (NTIS) and subclinical hypothyroidism reported in this age group. These changes can confound the interpretation of thyroid function tests in older adults and may have clinical consequences, particularly when coexisting with CKD [4,5].

However, this was established years back, and thyroid and kidney functional interplay has continued well into physiological and pathological states. Thyroid hormones (THs) may also be important in kidney organogenesis and function, and have effects that include the increase of renal blood flow and GFR. Hyperthyroidism would be associated with high values of GFR and activation of the renin-angiotensin-aldosterone system, while hypothyroidism would result in reduced GFR. Patients with CKD will also be known to have a generally high occurrence of both primary and subclinical hypothyroidism [4].

Alterations of the synthesis, secretion, metabolism, and elimination of thyroid hormones accompany kidney function decline. Hypothyroidism and hyperthyroidism affect the renal hemodynamics, glomerular filtration, electrolyte balance, tubular function, and the morphology of kidneys [5].

Many mechanisms lead to these rather complex and sometimes reversible aberrations. The hypothalamic-pituitary-thyroid axis disorders, serum protein binding of thyroid hormones, tissue uptake of the hormone changes, and alterations in thyroid hormone metabolism result in such conditions [6,7]. Diagnosing any thyroid hormone disorder will include assessing these abnormalities, as non-thyroidal illness syndrome also mimics an innocent or sometimes conflates biochemical patterns manifest in true thyroid diseases. The degree and kind of such abnormalities may also be prognostic markers of the underlying diseases [8].

Mechanisms by which chronic kidney disease affects thyroid function include decreased circulating levels of thyroid hormones, modifications in peripheral hormone metabolism, and deficient binding to carrier proteins. Numerous studies have identified an association between markers of cardiovascular risk and cardiac impairment and subclinical hypothyroidism. Even slight deviations from the normal serum level of thyroid-stimulating hormone may accelerate atherosclerosis development and negatively impact cardiovascular health in the general population [9,10].

Therefore, in elderly individuals, the interplay between age-related decline in renal function and age-associated changes in thyroid physiology results in a unique clinical scenario, necessitating careful evaluation of thyroid status in older patients with CKD. So, the present work aimed to assess the thyroid dysfunction in elderly chronic kidney disease patients with declining glomerular filtration rate.

METHODS

This prospective case-control study was carried out at the nephrology outpatient clinic and hemodialysis units of Zagazig General Hospital over one year. The study was conducted after obtaining approval from the Institutional Review Board (ZU-IRB#1385/25-5-2025) and written informed consent from all patients. The research was conducted under the World Medical Association's Code of Ethics (Helsinki Declaration) for human research.

Study Population

The study included 60 male and female participants aged 65 years and older. Participants were categorized into three groups: **Control Group:** 20 healthy subjects (10 males and 10 females, each comprising 50% of the group). **CKD Group (Stages III & IV):** 20 patients diagnosed with CKD stages III and IV were on conservative treatment (10 males and 10 females, each comprising 50% of the group). Conservative treatment included dietary modifications and pharmacological therapy. **Hemodialysis (HD) Group:** 20 patients with CKD stage V received regular hemodialysis for at least six months (10 males and 10 females, each comprising 50% of the group). Hemodialysis was performed three times/week, each session lasting at least four hours, using Fresenius 4008S dialysis machines. Low-flux polysulfone dialyzers and bicarbonate-based dialysate were standard for all patients.

Participants aged 65 years or older were included in the study. The patient groups consisted of individuals diagnosed with CKD stage III or IV receiving conservative treatment or those with stage V CKD undergoing regular hemodialysis for at least six months. All participants provided informed consent before enrollment.

Exclusion criteria involved cases younger than 65 years, those with a history of thyroid disease or ongoing thyroid treatment, incomplete medical records, or refusal to participate. Patients on medications known to affect thyroid hormone levels, like thyroxine, glucocorticoids, amiodarone, oral anticoagulants, interferon, and lithium, were also excluded. Additionally, individuals with active infections or inflammatory conditions unrelated to renal disease, hematological disorders, autoimmune diseases, or malignancies were excluded. Other exclusions comprised conditions associated with non-thyroidal illness syndrome, except renal failure, including prolonged fasting, liver disease, or known cardiac disease. Additionally, patients with a prior history of thyroidectomy or radioactive iodine therapy were excluded from the study.

Clinical Assessment

Each participant underwent a comprehensive medical evaluation, including demographic data collection such as name, age, sex, residence, and occupation. A detailed history was taken regarding previous and existing medical comorbidities, involving diabetes mellitus, hypertension, cardiovascular disease, dyslipidemia, liver disease, malignancy, thyroid disorders, and medication use. For hemodialysis patients, dialysis duration and history were also recorded.

A general physical examination was conducted for all participants. Blood pressure was evaluated using a mercury sphygmomanometer while the subject was recumbent with the arm supported at heart level. Pulse examination was performed to detect any abnormalities, along with body temperature measurements and respiratory rate.

Laboratory Investigations

Venous blood samples (10 mL) were collected under aseptic conditions following an overnight fast. In hemodialysis patients, blood was drawn just before dialysis initiation, directly from the arterial port or dialysis needle, before blood contact with the dialyzer and heparin administration, ensuring at least a 48-hour gap since the last heparin dose.

Complete blood count (CBC) was analyzed using an automated hematology analyzer (Mindray PC 2800 Hematology Analyzer). Liver function tests, including total protein, serum albumin, alanine aminotransferase (ALT), and aspartate aminotransferase (AST), were measured using a kinetic method. Renal function tests, including serum creatinine and blood urea nitrogen (BUN), were determined using a colorimetric method. Lipid profile assessments included fasting total cholesterol and triglyceride levels, while fasting blood glucose was measured via enzymatic assay.

We calculated the estimated glomerular filtration rate for all participants utilizing the MDRD equation and the eGFR mobile application recommended by the National Kidney Foundation. The calculation incorporated serum creatinine levels, age, gender, and ethnicity to estimate renal function. “GFR (mL/min/1.73 m2) = 175 × (Scr)-1.154 × (Age)-0.203 × (0.742 if female) × (1.212 if African American)”.

Thyroid Function Analysis

The determination of thyroid hormone levels, such as total triiodothyronine (T3), total thyroxine (T4), free T3 (fT3), free T4 (fT4), and TSH, was done through ELISA techniques. The technologies are based on antigen-antibody interaction to measure the hormone concentration. The normal reference

ranges of thyroid hormones comprise different elements, whereby the thyroid-stimulating hormone (TSH) was calculated from 0.25 to 5.0 µIU/mL. In contrast, the total thyroxine (T4) ranged between 60 and 120 nmol/L, and finally normal ranges of free thyroxine (fT4) between 10.8 and 19.4 pmol/L. Furthermore, total triiodothyronine (T3) was below 0.9 -2.3 nmol/L, and free triiodothyronine (fT3) was 2.5-8.3 pmol/L. Thus, these values were used as the study's standard reference to evaluate thyroid function among the participants.

Subclinical hypothyroidism was diagnosed as an elevated TSH (>5.0 µIU/mL) with normal free T4 (10.8–19.4 pmol/L) and normal free T3 (2.5–8.3 pmol/L) levels, in the absence of clinical symptoms of hypothyroidism. Non-thyroidal illness syndrome (NTIS) was diagnosed as a reduced total T3 (<0.9 nmol/L) and/or free T3 (<2.5 pmol/L), with normal or low TSH and normal or low free T4, in the absence of primary thyroid disease or use of thyroid medications [5].

Imaging Studies

All study participants underwent renal ultrasound to assess kidney morphology, size, cortical echogenicity, and to exclude obstructive uropathy or structural abnormalities. This imaging was performed using a standard B-mode ultrasonography with a convex probe (3.5–5 MHz). The findings were used to support clinical CKD staging and differentiate chronic parenchymal changes from acute causes of renal dysfunction. Additionally, thyroid ultrasound was conducted for subjects with abnormal thyroid function tests to evaluate thyroid size, nodularity, and parenchymal echotexture, aiding in the exclusion of structural thyroid disease. All scans were interpreted by experienced radiologists blinded to laboratory data.

Statistical analysis

All data were analyzed using SPSS 22.0 and MedCalc 13. Categorical data were shown as frequencies, whereas continuous variables were shown as means ± SD and median (range). Regarding group differences, parametric data with normally distributed distributions were analyzed using one-way ANOVA, while categorical data were analyzed using Chi-square (χ^2). According to Spearman's correlation, this study's parameters were related to CRP and Neopterin. The diagnostic performance was assessed using AUROC, with a range of 0.90-1 showing excellent, 0.80-0.90 good, and <0.60 failing. Statistical significance was found when $p < 0.05$.

RESULTS

The age, BMI, sex, and smoking habits of participants were similar across the three groups ($p > 0.05$). However, SBP and DBP were significantly higher in the CKD and HD groups than in the control group ($p = 0.002$ and $p = 0.037$). For SBP, there was a marked difference when comparing controls to HD ($p < 0.001$) and a lesser difference when comparing to CKD ($p = 0.035$). For DBP, there were significantly higher levels in CKD and HD vs. controls ($p < 0.001$, $p = 0.001$). A statistically significant variance in DBP was present between CKD and HD ($p = 0.014$) (**Table 1**).

Although the CKD and HD groups did not vary substantially ($p = 0.21$), both groups had significantly lower hemoglobin levels compared to the control group ($p < 0.001$). Fasting blood glucose and serum triglyceride levels were significantly higher in CKD and HD groups when compared to controls ($p < 0.05$ for all comparisons). Still, there was no significant difference between the CKD and HD groups ($p = 0.811$ for FBS, $p = 0.762$ for TG) (**Table 2**).

Compared to the control group, the CKD and HD groups had significantly lower levels of total protein, albumin, ALT, and AST ($p < 0.001$ for total protein and albumin, $p = 0.034$ and $p = 0.043$ for ALT and AST, respectively). In contrast, albumin and ALT were slightly lower in HD than CKD ($p = 0.032$ and $p = 0.699$). Kidney function tests showed significantly higher serum creatinine and urea in CKD and HD groups than controls ($p < 0.001$), with HD values being the highest. eGFR was significantly lower in CKD and HD groups, with the lowest levels in HD patients ($p < 0.001$) (**Table 3**).

About TSH, T4, or free T4, thyroid functions were all similar across groups at $p > 0.05$. On the contrary, total T3 showed lower levels than controls, with its accompanying free T3 levels among CKD and HD patients ($p=0.001$; $p=0.005$). CKD and HD groups did not differ significantly regarding these parameters: $p=0.501$ and $p=0.97$ (**Table 4**).

The prevalence of thyroid abnormalities was significantly greater in the CKD and HD groups relative to the controls ($p = 0.011$). At

the same time, subclinical hypothyroidism and NTIS were found more frequently among HD than CKD patients. However, the distribution of thyroid dysfunctions across CKD stages did not influence their prevalence (p = 0.27). The most prevalent type of dysfunction was euthyroidism, followed by low T3 syndrome and subclinical hypothyroidism. This correlates with a greater incidence of thyroid dysfunction in CKD and HD at advanced stages of disease (**Table 5**).

The majority of these individuals demonstrated normal thyroid gland size and echotexture. Minor abnormalities were observed in a small subset: three patients had diffuse mild hypoechogenicity without nodularity, and two patients showed a single benign-appearing thyroid nodule (<1 cm) with regular margins and no suspicious features. There was no evidence of significant thyroid enlargement, multinodularity, or features suggestive of thyroiditis or malignancy in any patient; structural thyroid abnormalities were not commonly detected in this elderly CKD cohort, and most thyroid dysfunction appeared to be functional rather than structural.

Total and free T3 levels showed strong correlations with hemoglobin (p = 0.013 and p = 0.047, respectively), creatinine (p < 0.001), urea (p < 0.001), and eGFR (p < 0.001) (**Table 6 and Figure 1**)

Table (1): Comparison of demographic data, mean values ± SD of SBP & DBP, and LSD for systolic and Diastolic Blood Pressure of the three studied groups:

Variable	Control Group (n=20)		CKD Group (n=20)		*HD Group (n=20)*		F	p
Age (years):								
- Mean ± SD	**67.65 ± 2.46**		**68 ± 2.81**		**67.9 ± 2.47**		**0.09**	**0.91 (NS)**
- Range	**65 - 75**		**65 - 74**		**65 - 73**			
BMI (kg/m^2):								
- Mean ± SD	**22.39 ± 1.79**		**22.1 ± 1.86**		**21.89 ± 1.74**		**0.39**	**0.68 (NS)**
- Range	**19.3 – 24.4**		**18.9 - 25**		**19.5 – 24.7**			
	No	**%**	**No**	**%**	**No**	**%**	**χ^2**	**p**
Sex:								
- Male	**10**	**50**	**10**	**50**	**10**	**50**	**0**	**1 (NS)**
- Female	**10**	**50**	**10**	**50**	**10**	**50**		
Smoking:								
- No	**16**	**80**	**15**	**75**	**16**	**80**	**0.20**	**0.91 (NS)**
- Yes	**4**	**20**	**5**	**25**	**4**	**20**		

Variable	Control Group (n=20)	CKD Group (n=20)	HD Group (n=20)	F	P
SBP: (mmHg)					
- Mean ± SD	**129.35 ± 10.29**	**137.75±13.72**	**143.5± 12.68**	**6.779**	**0.002 (HS)**
- Range	**110 – 145**	**115 - 165**	**120 - 170**		
DBP: (mmHg)					
- Mean ± SD	**81.5±6.09**	**86.25± 12.34**	**90.79±13.49**	**3.487**	**0.037 (S)**
- Range	**70-90**	**70-105**	**70-115**		

LSD for systolic and Diastolic Blood Pressure

	SBP	DBP
Control vs CKD	0.035 (S)	<0.001 (HS)
Control vs HD	<0.001 (HS)	0.001 (HS)
CKD vs HD	0.177 (NS)	0.014 (S)

SBP: Systolic Blood Pressure, DBP: Diastolic Blood Pressure, BMI: Body Mass Index, CKD: Chronic Kidney Disease, HD: Hemodialysis, LSD: Least Significant Difference, mmHg: Millimeters of Mercury, SD: Standard Deviation, NS: Non-Significant, S: Significant, HS: Highly Significant, χ^2: Chi-Square Test, F: F-statistic from ANOVA Test. SD: Stander deviation, F: ANOVA test, χ2: Chi-square test

Table (2): Comparison of the mean values ± SD of CBC results, LSD for Hemoglobin, Lipid profile and FBS, and LSD for Fasting Blood Glucose and Serum Triglycerides of the three studied groups:

Variable	Control Group (n=20)	CKD Group (n=20)	HD Group (n=20)	F	P
WBCs: ($\times10^3$/ul) - **Mean ± SD** - **Range**	**6.04 ± 2.1** **3.2 – 10**	**6.24 ± 1.9** **3.3 - 10**	**6.35 ± 1.96** **3.8 - 10**	**0.14**	**0.93 (NS)**
Hb: (gm/dl) - **Mean ± SD** - **Range**	**13.5 ± 0.84** **12.2 – 15.2**	**11.19 ± 0.94** **9.3 – 12.6**	**10.83 ± 0.89** **9.3 – 12.5**	**53.17**	**<0.001 (HS)**
Platelets: ($\times10^3$/ul) - **Mean ± SD** - **Range**	**290.4 ± 53.24** **180 – 370**	**265.55±62.74** **185 - 320**	**261.75±74.39** **175 - 290**	**3.657**	**0.314 (NS)**

LSD for Hemoglobin	
	Hemoglobin
Control vs CKD	<0.001 (HS)
Control vs HD	<0.001 (HS)
CKD vs HD	0.21 (NS)

Variable	Control Group (n=20)	CKD Group (n=20)	HD Group (n=20)	F	P
FBS: (mg/dL) - **Mean ± SD** - **Range**	86.2 ± 10.87 70 – 102	123.85± 52.87 70 - 230	119.75± 54.95 74 - 255	K 4.315	0.018 (S)
Fasting triglyceride:(mg/dL) - **Mean ± SD** - **Range**	115.45 ± 14.2 110 – 157	142.9 ± 44.86 92 - 234	147.31± 46.71 87 - 225	F 4.067	0.022 (S)
Fasting Cholesterol: (mg/dL) - **Mean ± SD** - **Range**	175.95± 20.11 105 – 170	181.65± 20.07 95 - 168	173.65± 21.95 110 - 190	F 0.790	0.459 (NS)

LSD for Fasting Blood Glucose and Serum Triglycerides		
	FBS	**TG3**
Control vs CKD	0.003 (HS)	0.013 (S)
Control vs HD	0.011 (S)	0.006 (HS)
CKD vs HD	0.811 (NS)	0.762 (NS)

WBCs: White Blood Cells, Hb: Hemoglobin, CKD: Chronic Kidney Disease, HD: Hemodialysis, FBS: Fasting Blood Sugar, LSD: Least Significant Difference, SD: Standard Deviation, NS: Non-Significant, S: Significant, HS: Highly Significant, K: Kruskal-Wallis Test, F: F-statistic from ANOVA Test, mg/dL: Milligrams per Deciliter, µL: Microliter, Fasting Triglyceride: Fasting Triglyceride Level, Fasting Cholesterol: Fasting Cholesterol Level. SD: Stander deviation, F: ANOVA test.

Table (3): Comparison of the mean values ± SD of Liver function, kidney functions test results, LSD for Total plasma proteins, Serum Albumin, ALT and AST, LSD for Blood Urea, Serum creatinine and eGFR of the three studied groups:

Variable	Control Group (n=20)	CKD Group (n=20)	HD Group (n=20)	F	P
T. protein: (g/dL)					
- **Mean ± SD**	7.39 ± 0.40	6.81 ± 0.35	6.72 ± 0.37	18.42	<0.001
- **Range**	6.8 – 8	6.4 – 7.5	6 – 7.5		(HS)
Albumin: (g/dL)					
- **Mean ± SD**	3.9 ± 0.18	3.75 ± 0.14	3.59 ± 0.29	10.59	<0.001
- **Range**	3.5 – 4.5	3.5 - 4	3.2 – 3.5		(HS)
ALT: (u/L)					
- **Mean ± SD**	26.34 ± 6.41	21.30± 8.64	20.29± 7.71	3.308	0.034
- **Range**	19 – 37	10 - 35	9 - 34		(S)
AST: (u/L)					
- **Mean ± SD**	21.54± 4.18	18.53± 4.73	17.84± 5.48	3.324	0.043
- **Range**	15 – 28	12 - 25	10 - 24		(S)

LSD for Total plasma proteins, Serum Albumin, ALT and AST

	T. Protein	S. ALbumin	ALT	AST
Control vs CKD	<0.001 (HS)	0.006 (HS)	(S)0.043	(S)0.039
Control vs HD	<0.001 (HS)	<0.001 (HS)	(S)0.010	(S)0.021
CKD vs HD	0.434 (NS)	0.032 (S)	0.699 (NS)	(NS)672.0

Variable	Control Group (n=20)	CKD Group (n=20)	HD Group (n=20)	Test	P
S Creatinine: (mg/dL)					
- **Mean ± SD**	0.67±0.11	2.26±0.91	9.27±1.92	K	<0.001
- **Range**	0.05 – 0.8	1.1 – 4.1	6.1 – 13.1	52.62	(HS)
Urea: (mg/dl)					
- **Mean ± SD**	30.95±6.11	104.6±26.09	129.15±24.15	F	<0.001
- **Range**	21 - 39	60 - 147	85 - 163	120.4	(HS)
eGFR: (ml/min/1.73m2)					
- **Mean ± SD**	106±10.52	30.2±12.05	5.05±1.5	K	<0.001
- **Range**	95 - 123	15 - 50	3 – 9	52.71	(HS)

LSD for Blood Urea, Serum creatinine and eGFR

	Urea	Creatinine	EGFR
Control vs CKD	<0.001 (HS)	<0.001 (HS)	<0.001 (HS)
Control vs HD	<0.001 (HS)	<0.001 (HS)	<0.001 (HS)
CKD vs HD	0.004 (HS)	<0.001 (HS)	<0.001 (HS)

T. Protein: Total Protein, S. Albumin: Serum Albumin, ALT: Alanine Aminotransferase, AST: Aspartate Aminotransferase, CKD: Chronic Kidney Disease, HD: Hemodialysis, LSD: Least Significant Difference, SD: Standard Deviation, NS: Non-Significant, S: Significant, HS: Highly Significant, K: Kruskal-Wallis Test, F: F-statistic from ANOVA Test, mg/dL: Milligrams per Deciliter, eGFR: Estimated Glomerular Filtration Rate, ml/min/1.73m²: Milliliters per Minute per 1.73 Square Meters of Body Surface Area, S. Creatinine: Serum Creatinine, Urea: Blood Urea Nitrogen (BUN). SD: Stander deviation, F: ANOVA test.

Table (4): Comparison of the mean values ± SD of thyroid function, LSD for T3 and free T3 of the three studied groups:

Variable	Control Group (n=20)	CKD Group Stage III &IV (n=20)	HD Group (n=20)	F	P
TSH: (µIU/ml)					
- **Mean ± SD**	3.8 ± 0.51	4.18 ± 1.11	4.12 ± 0.80	1.16	0.32
- **Range**	2.8 – 4.6	2.6 – 7.6	3.4 – 6.2		(NS)
T4: (nmol/L)					
- **Mean ± SD**	94.4 ± 11.86	94.15 ± 9.74	90.4 ± 10.20	0.89	0.42
- **Range**	74 – 111	77 - 110	75 - 108		(NS)
Free T4: (pmol/L)					
- **Mean ± SD**	13.46 ± 1.32	12.81 ± 1.52	12.76 ± 1.62	1.32	0.28
- **Range**	11.2 – 16.2	10.6 – 15.4	10.7 – 16.2		NS
T3: (nmol/L)					
- **Mean ± SD**	1.65 ± 0.28	1.27 ± 0.46	1.17 ± 0.47	7.532	0.001
- **Range**	1.2 – 2.2	0.6 – 2.0	0.4 – 1.8		(HS)
Free T_3: (pmol/L)					
- **Mean ± SD**	5.5 ± 0.98	3.95 ± 1.32	3.97 ± 2.34	5.800	0.005
- **Range**	3.3 – 7.2	1.4 – 6.5	1 – 7.2		(HS)

LSD for T3 and free T3		
	Total T3	**Free T3**
Control vs CKD	0.003 (HS)	0.004 (HS)
Control vs HD	<0.001 (HS)	0.005 (HS)
CKD vs HD	0.501 (NS)	0.97 (NS)

TSH: Thyroid-Stimulating Hormone, T4: Total Thyroxine, Free T4: Free Thyroxine (fT4), T3: Total Triiodothyronine, Free T3: Free Triiodothyronine (fT3), CKD: Chronic Kidney Disease, HD: Hemodialysis, LSD: Least Significant Difference, SD: Standard Deviation, NS: Non-Significant, S: Significant, HS: Highly Significant, F: F-statistic from ANOVA Test, µIU/mL: Micro-International Units per Milliliter, nmol/L: Nanomoles per Liter, pmol/L: Picomoles per Liter. SD: Stander deviation, F: ANOVA test.

Table (5): Distribution of thyroid function abnormalities among the patients groups, and Relation between thyroid function abnormality and CKD stage among the two cases groups::

Thyroid Status	Control Group (N=20)		CKD Group (N=20)		HD Group (N=20)		X^2	p-value (Sig.)
	No	%	No	%	No	%		
*Normal thyroid	20	100%	14	70%	10	50%	13.05	0.011 (S)
*NTIS	0	0%	4	20%	6	30%		
*Subclinical hypothyroidism	0	0%	2	10%	4	20%		
*Subclinical hyperthyroidism	0	0%	0	0%	0	0%		
Variable	**Subclinical hypothyroidism (n=6)**		**NTI (n=10)**		**Euthyroid (n=24)**		χ^2	**P**

	No	%	No	%	No	%		
Stage:								
- **Stage III**	0	0%	1	10%	9	90%	5.2	0.27 NS
- **Stage IV**	2	20%	3	30%	5	50%		
- **Stage V**	4	20%	6	30%	10	50%		

NTIS: Non-Thyroidal Illness Syndrome, CKD: Chronic Kidney Disease, HD: Hemodialysis, χ^2: Chi-Square Test, S: Significant, NS: Non-Significant, p-value: Probability Value, Subclinical Hypothyroidism: A mild form of hypothyroidism with normal T4 but elevated TSH, Euthyroid: Normal Thyroid Function, Stage III, IV, V: Stages of Chronic Kidney Disease Progression, %: Percentage. X^2: Chi-square

Table (6): Correlation between Thyroid functions and demographic data, BP and laboratory Investigation of the cases groups:-

Correlations						
		TSH	T4	fT4	T3	fT3
TSH (µIU/ml)	r		0.267	0.041	0.095	0.066
	p		0.096 (NS)	0.800 (NS)	0.561 (NS)	0.686 (NS)
T4 (nmol/L)	r			0.169	-0.106	-0.073
	p			0.296 (NS)	0.513 (NS)	0.654 (NS)
Free T4 (pmol/L)	r				-0.043	0.349*
	p				0.790 (NS)	0.027 (S)
T3 (nmol/L)	r					0.732**
	p					0.000 (HS)
Free T3 (pmol/L)	r					
	p					
Sex (M/F)	r	-0.104	0.088	-0.070	-0.177	-0.154
	p	0.525 (NS)	0.588 (NS)	0.667 (NS)	0.274 (NS)	0.343 (NS)
Age (years)	r	-0.265	-0.004	0.118	-0.202	-0.072
	p	0.099 (NS)	0.979 (NS)	0.467 (NS)	0.212 (NS)	0.658 (NS)
Smoking	r	0.049	0.082	-0.056	-0.224	-0.001
	p	0.763 (NS)	0.616 (NS)	0.732 (NS)	0.166 (NS)	0.994 (NS)
BMI (kg/m^2)	r	0.104	0.166	0.042	0.268	0.198
	p	0.524 (NS)	0.305 (NS)	0.797 (NS)	0.094 (NS)	0.220 (NS)
SBP (mmHg)	r	0.206	-0.178	-0.064	0.122	-0.081
	p	0.203 (NS)	0.289 (NS)	0.694 (NS)	0.452 (NS)	0.619 (NS)
DBP (mmHg)	r	0.105	-0.013	-0.054	0.13	0.128
	P	0.518 (NS)	0.936 (NS)	0.717 (NS)	0.936 (NS)	0.430 (NS)
Hemoglobin (gm/dl)	r	0.080	-0.147	-0.044	0.389*	0.316*
	p	0.625 (NS)	0.365 (NS)	0.790 (NS)	0.013 (S)	0.047 (S)
WBCs (x10^3/mm^3)	r	-0.013	-0.163	-0.118	-0.087	-0.058
	p	0.937 (NS)	0.314 (NS)	0.467 (NS)	0.595 (NS)	0.724 (NS)
Platelets (x10^3/mm^3)	r	0.028	-0.099	0.215	0.063	0.254
	p	0.865 (NS)	0.544 (NS)	0.182 (NS)	0.700 (NS)	0.114 (NS)
Fasting blood glucose (mg/dl)	r	0.022	-0.060	-0.082	0.172	0.125
	p	0.893 (NS)	0.714 (NS)	0.614 (NS)	0.289 (NS)	0.441 (NS)
T.Proteins (mg/dl)	r	0.161	-0.073	-0.162	0.265	0.233
	p	0.321 (NS)	0.656 (NS)	0.318 (NS)	0.099 (NS)	0.147 (NS)
Albumin (mg/dl)	r	0.078	-0.030	0.310	0.277	0.301
	p	0.634 (NS)	0.856 (NS)	0.052 (NS)	0.083	0.092 (NS)
ALT (U/L)	r	-0.064	-0.051	0.039	-0.112	-0.052
	p	0.696 (NS)	0.753 (NS)	0.811 (NS)	0.490 (NS)	0.750 (NS)

AST (U/L)	r	0.171	-0.057	0.041	-0.172	-0.022
	p	0.292 (NS)	0.725 (NS)	0.803 (NS)	0.289 (NS)	0.892 (NS)
S.Creatinine (mg/dl)	r	0.045	-0.032	-0.133	-0.595**	-0.935**
	p	0.781 (NS)	0.846 (NS)	0.412	0.000 (HS)	0.000 (HS)
eGFR (ml/min)	r	0.102	-0.104	0.301	0.644**	0.926**
	p	0.533 (NS)	0.522 (NS)	0.059 (NS)	0.000 (HS)	0.000 (HS)
Urea (mg/dl)	r	-0.158	0.195	0.100	-0.664**	-0.653**
	p	0.332 (NS)	0.228 (NS)	0.540 (NS)	0.000 (HS)	0.000 (HS)
Fasting triglycerides (mg/dl)	r	-0.078	-0.159	-0.030	0.112	0.000
	p	0.633 (NS)	0.328 (NS)	0.853 (NS)	0.493 (NS)	0.998 (NS)
Fasting cholesterol (mg/dl)	r	-0.120	0.155	0.101	-0.055	-0.076
	p	0.462 (NS)	0.340 (NS)	0.537 (NS)	0.737 (NS)	0.641 (NS)
**. Correlation is significant at the 0.01 level (2-tailed).						
*. Correlation is significant at the 0.05 level (2-tailed).						

TSH: Thyroid-Stimulating Hormone, T4: Total Thyroxine, fT4: Free Thyroxine, T3: Total Triiodothyronine, fT3: Free Triiodothyronine, r: Spearman's Correlation Coefficient, p: p-value (Probability Value), NS: Non-Significant, S: Significant, HS: Highly Significant, BMI: Body Mass Index, SBP: Systolic Blood Pressure, DBP: Diastolic Blood Pressure, Hb: Hemoglobin, WBCs: White Blood Cells, ALT: Alanine Aminotransferase, AST: Aspartate Aminotransferase, S. Creatinine: Serum Creatinine, eGFR: Estimated Glomerular Filtration Rate, Urea: Blood Urea Nitrogen (BUN), Fasting Blood Glucose: Fasting Blood Sugar, Fasting Triglycerides: Fasting Triglyceride Level, Fasting Cholesterol: Fasting Cholesterol Level, T. Proteins: Total Proteins, Albumin: Serum Albumin.r Spearman rank correleation coefficient. P< 0.05 is significant.

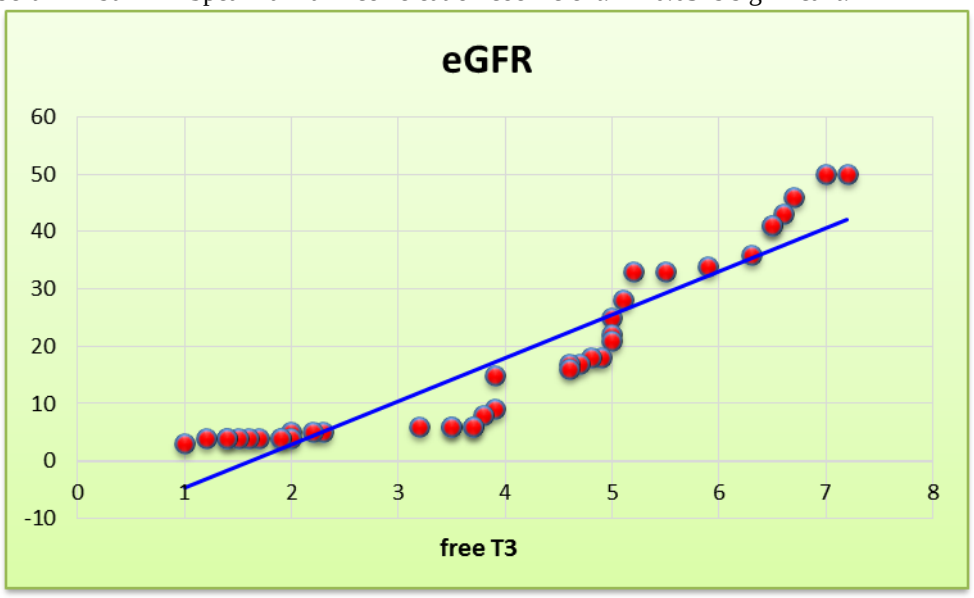

Figure (1): Correlation between eGFR and Total and free T3

DISCUSSION

The thyroid gland plays a crucial role in metabolism, growth, protein synthesis, and hormone regulation by producing triiodothyronine (T3) and thyroxine (T4). Alterations in thyroid function can disrupt hormone production, leading to various diseases [11,12]. Aging affects thyroid function and test parameters [13], while CKD impacts thyroid function and morphology through hypothalamic-pituitary-thyroid (HPT) axis disturbances and altered peripheral thyroid hormone metabolism. These disruptions may contribute to the complex interplay between thyroid dysfunction and CKD-related metabolic derangements [14]. Most of the information clinicians may find is Low T3 within the laboratory findings, and subclinical hypothyroidism is the most commonly found thyroid disorder in CKD patients [15].

The thyroid and kidneys are closely connected. Thyroid hormones (T3 and T4) affect kidney development, blood flow, and glomerular filtration rate. Hypothyroidism decreases GFR, while hyperthyroidism increases GFR and stimulates renin-angiotensin-aldosterone system activity. Chronic kidney disease also shows a higher incidence of hypothyroidism [15].

This prospective, case-control study examined thyroid dysfunction in elderly CKD patients with declining GFR. Sixty participants (equal male and female) aged 65–75 years (median 67.5) were recruited. Most patients had hypertension, diabetes, or both, reflecting the higher prevalence of these conditions in this age group [16]. Other causes of CKD in the patient groups included obstructive uropathy, drug-induced nephropathy, and chronic glomerulonephritis. The following classification of CKD into different stages for this study was based on National Kidney Foundation guidelines [12], estimating GFR using the 4-variable MDRD formula.

The current study found no significant differences in age, sex, smoking, and body mass index (BMI) between the control and CKD groups. This conclusion is in line with previous research [17–20], and it is explained by the fact that the individuals were selected via cross-matching based on age and sex.

In line with Ridao et al. [21], the hemodialysis and chronic kidney disease groups had substantially higher systolic and diastolic blood pressures than the control group. Excess volume and sodium, activation of the renin-angiotensin-aldosterone system, sympathetic overactivity, and decreased vasodepressor chemicals are some of the contributing causes. Structural arterial changes, renovascular disease, natriuretic peptides, and pre-existing hypertension also play roles. Additionally, anemia, hypoxia, vasopressin, serotonin, and thyroid dysfunction further exacerbate hypertension, as noted by Campese [22].

Regarding CBC parameters, WBC levels showed no significant difference between the control and CKD groups. However, hemoglobin is significantly lower in CKD patients, consistent with Jingxian et al. [23], who attributed CKD-related anemia to multiple factors. These included erythropoietin deficiency, reduced erythroblast sensitivity, vitamin B12/folic acid deficiencies, impaired iron utilization, gastrointestinal bleeding, hyperparathyroidism, systemic inflammation, dialysis, platelet dysfunction from uremia, and treatment-related drug effects.

A review of platelet counts revealed that the platelet level was significantly reduced in CKD groups compared to the control; this observation supports the findings of Ahmed [24] but contradicts those of Eneman et al. [25]. Our results supported that mild thrombocytopenia frequently occurs in these patients due to insufficient production of thrombopoietin.

The study presents findings concerning the lipid profile, indicating that serum triglycerides were significantly raised in both non-dialysis and hemodialysis groups of CKD patients compared to the control group. This finding corroborates the study by Lakshmi and Anil [26], who also described hypercholesterolemia in CKD patients, posing an opinion of causation due to heavy proteinuria.

Serum total protein and albumin levels were significantly lower in CKD groups compared to control levels, corroborating Jingxian et al.'s findings [23]. These reductions are likely due to multiple factors, primarily related to uremia and dialysis, such as depression and anorexia, insufficient dietary recommendations and supplementation, chronic inflammation, and metabolic acidosis. Furthermore, amino acid and protein losses associated with dialysis exacerbate declines in protein and albumin levels in these patients.

In the current study, ALT and AST levels were significantly lower in CKD and dialysis groups than in the control group, consistent with Ray et al. [27]. The exact cause remains unclear, but Liberato et al. [28] suggested water retention and hemodilution as contributing factors. Other possible mechanisms include reduced pyridoxal-5-phosphate (a key coenzyme), high uremic toxin levels, decreased synthesis, inhibited hepatocyte release, or accelerated clearance of aminotransferases.

Concerning kidney function tests, the serum creatinine and urea levels were significantly higher. At the same time, eGFR was considerably lower in the CKD groups compared to the control group, and these findings are analogous to the findings of Rajeev et al. [29].

Both total and free T3 came down significantly in non-dialysis and hemodialysis

CKD patient groups as opposed to those in the control group. In contrast, serum TSH levels failed to show significant elevation compared to the control group; this may be attributed to its inhibited response to thyroid-releasing hormone (TRH) and intact thyroid-pituitary axis. These results agree with those of Ahmed [24] and Rajeev et al. [29].

Several pathophysiological mechanisms can explain the reduction in T3 (and free T3). First, CKD is associated with decreased peripheral conversion of thyroxine (T4) to triiodothyronine (T3), mainly due to impaired activity of the type 1 deiodinase enzyme in peripheral tissues. Uremic toxins, chronic metabolic acidosis, and inflammatory cytokines prevalent in CKD may inhibit this enzymatic process. Second, protein-energy wasting and malnutrition, common in advanced CKD and dialysis, further suppress peripheral deiodination. Third, the accumulation of inhibitors or altered binding proteins in CKD patients' serum may reduce free thyroid hormone availability. The chronic inflammation and oxidative stress that characterize CKD also contribute to non-thyroidal illness syndrome (NTIS), where T3 levels drop as a metabolic adaptation to reduce catabolism during illness or physiological stress. Lastly, some medications frequently used in CKD (e.g., steroids, beta-blockers) may also impair thyroid hormone metabolism [8,33,34]. These combined factors result in the observed 'low T3 syndrome,' which is recognized as a marker of adverse prognosis in the CKD population.

Consistent with what Song et al. [30] found, the proportion of patients with low T3 levels rose as the severity of CKD increased: 1 in Stage III, 3 in Stage IV, and 6 in Stage V. Additionally, they found a positive association between eGFR and serum T3, and an increasing trend of low T3 prevalence with lowering eGFR. The chances of low T3 were 2.40 times greater for eGFR < 60 ml/min/1.73 m^2 compared to eGFR ≥ 60 ml/min/1.73 m^2, even after accounting for age and sex.

Altay et al. [31] investigated the effects of thyroid hormones on elderly dialysis patients and whether or not the patients' advancing age and the severity of their disease pose any extra risks for abnormal thyroid function tests. They discovered thyroid function problems were more common in older dialysis patients than in controls. Chronic dialysis patients of all ages, including the young and middle-aged, had lower fT3 levels.

According to the current study, thyroid dysfunction is more common in individuals in stages 4 and 5 than in patients in stage 3. Lo et al. [32] discovered that the risk of hypothyroidism increased with the decrease in GFR, and this data so corroborates those findings.

Non-thyroidal illness is the biochemical alteration of thyroid hormones without preexisting hypothalamic-pituitary and thyroid gland dysfunction. This is characterized by low T3 and fT3, increased rT3, and normal TSH, whereas T4 can be normal or low [33].

In ESRD patients, Zoccali et al. [34] observed reduced free T3 (fT3) levels with unchanged free T4 (fT4), indicating a true selective T3 deficiency likely due to impaired T4-to-T3 conversion rather than an artifact from binding inhibitors. The absence of a compensatory TSH increase may be explained by stress-induced pituitary inhibition, a blunted TSH response to TRH, and chronic metabolic acidosis contributing to low fT3 levels.

In our study, subclinical hypothyroidism was observed in 15% of patients, with the majority of affected individuals in CKD Stage 5 on hemodialysis. These findings are consistent with those reported by Chonchol et al. [35], who demonstrated an increased prevalence of subclinical hypothyroidism in CKD patients, notably among older adults and those not yet requiring chronic dialysis.

Although patients may be considered euthyroid during a brief NTIS, they become biochemically hypothyroid with a longer NTI and appear to benefit from thyroid hormone replacement treatment. No effect or significant improvement was found in studies testing T3 replacement treatment in NTIS [36].

The correlation analysis in both the HD and CKD groups found no significant correlation between TSH and eGFR, urea, or creatinine. This finding corroborates the findings of Dudani et al. [37] and Swaminathan et al. [38], who reasoned that this was due to blunting of the TSH response to TRH in uremic patients. In contrast, Rajeev et al. [29] and Shamsuddin and Sajjan [39] found a significant negative correlation between TSH and eGFR. Total T3 and free T3 showed a positive correlation with eGFR and a negative correlation with urea and creatinine, which agrees with Ahmed [24] and Altay et al. [31].

The study has several limitations that affect the findings' generalizability and causal interpretation. The small sample size and cross-sectional design restrict broader applicability, while potential confounding factors such as comorbidities and concurrent medications were not comprehensively controlled. Additionally, the absence of long-term follow-up limits insights into the dynamic changes of thyroid function relative to the progression of CKD. It is important to note that using the MDRD equation for eGFR assessment has recognized limitations, especially in patients with near-normal renal function. It may underestimate GFR compared to the CKD-EPI equation. The CKD-EPI formula is now widely regarded as more precise across a broader range of GFR values and is recommended in updated clinical guidelines. This study relied on MDRD due to institutional standards during data collection. Future research in this population would benefit from utilizing the CKD-EPI equation for greater accuracy and comparability.

CONCLUSIONS

The study demonstrates a significant association between declining renal function and reduced T3/fT3 levels in elderly CKD patients, particularly those undergoing hemodialysis. The findings underscore a higher prevalence of subclinical hypothyroidism and non-thyroidal illness syndrome in advanced stages of CKD, emphasizing the clinical importance of regular thyroid function monitoring. These results suggest that routine thyroid assessments could enhance management strategies and potentially improve outcomes in this vulnerable patient population.

Conflicts of Interest

The authors report no conflicts of interest.

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