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ORIGINAL ARTICLE

Iodine Level and Thyroid Hormonal Profile in Patients on Regular Hemodialysis

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Abstract

Background: ***Low serum T3 and T4 levels can be caused by hormone loss during dialysis, decreased T3-binding capacity, serum thyroid autoantibodies, altered hormonal catabolism, increased thyroid iodine storage, and impaired peripheral conversion.***

Aim and objectives: ***To investigate thyroid function tests and their correlation with serum iodine levels in patients on regular hemodialysis.***

Subjects and methods: ***This prospective cross-sectional trial involved 100 cases of hemodialysis selected from attendees at Al-Azhar University Hospitals and Fayoum General Hospital. Participants were divided into two groups.***

Results: ***There was no substantial variance amongst the two investigated groups concerning gender, age, and BMI. There was a statistically significant distinction in TSH, free T3, and free T4 across the two groups; however, there was no distinction in iodine. Free T4 and free T3 levels were positively and significantly correlated with iodine. Meanwhile, there is a significant negative association between iodine and TSH.***

Conclusion: ***Our study revealed that, despite the significant variation among the two groups concerning TSH, free T3, and free T4, there was no significant difference regarding iodine level, and there was a significant positive correlation among iodine values with free T4 and free T3 but not TSH.***

Keywords: Thyroxine (T4); Triiodothyronine (T3); Thyroid stimulating hormone(TSH); Iodine; Hemodialysis patients

1. Introduction

Reduction in glomerular filtration rate and impairment of kidney function are hallmarks of the pathophysiological disorders known together as chronic kidney disease. Globally, CKD claimed over 1.2 million lives. Between 1990 and 2017, there was a 41.5 percent rise in the global death rate due to CKD. Researchers have described oxidative stress as playing a crucial role in the progression of disease and complications in cases of chronic kidney disease (CKD). [1]

When applying hemodialysis, the aim is to artificially execute the duties usually handled by the kidneys to bring about balance inside and around the cells. The semipermeable membrane reverses the concentration gradient, allowing the solute to diffuse from the blood plasma into the dialysate at a rate proportional to its particle size. Consequently, the rate of diffusion of ions and small molecules is considerably higher than that of larger molecules and protein-bound solutes. [2]
The value of iodine as a trace element is well established. Many physiological and metabolic processes must happen to make T4 and T3, which are thyroid hormones. These include controlling body growth, reproduction, and neurological activity. [3]
In chronic renal failure, decreased urinary iodine

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clearance contributes to elevated inorganic iodine (iodide) and thyroid iodine concentrations. These elevated levels can potentially obstruct thyroid hormone production (via the Wolff-Chaikoff effect) and cause glandular enlargement. Nevertheless, a complex web of interrelated mechanisms can impact thyroid function and induce hyperthyroidism, whether overt or subclinical, in people who are more susceptible to it (the elderly, those with preexisting thyroid illness, patients with other risk factors, etc.).[4] Removing hormones during dialysis, altered hormonal catabolism, decreased T3-binding capacity, elevated thyroid iodine storage, serum thyroid autoantibodies, and poor peripheral conversion can all lower serum levels of peripheral thyroid hormone (T3 and T4). Individuals with ESRD have a low T3. This condition is caused by persistent metabolic acidosis and decreased peripheral T3-to-T4 conversion. [5]

It is necessary to quantify the prevalence of thyroid disease because thyroid abnormalities, such as hypo- and hyperthyroidism, may increase mortality and morbidity among ESRD patients. [6]

This study aimed to investigate thyroid function tests and their relationship with serum iodine levels in patients on regular hemodialysis.

2. Patients and methods

T This prospective cross-sectional study involved 100 cases selected from attendees at Al-Azhar University Hospitals and Fayoum General Hospital for hemodialysis. Participants were divided into two groups: The study group consisted of 75 patients with ESRD on hemodialysis. Control group: 25 healthy individuals who were a statistical match for the research group regarding age and sex.

We include participants who are over eighteen years old, have ESRD, and have been on regular hemodialysis for at least six months, with three sessions per week, each lasting four hours—a clinically stable condition.

The exclusion criteria for cases include Acute infection, hepatic disease, thyroid hormones or thyroid-influencing drugs such as phenytoin, amiodarone, glucocorticoids, or lithium, thyroidectomy, malignancy, autoimmune diseases, and patient's refusal to participate.

2.1.Operational design: We carried out the following actions for cases and management, with informed consent and a detailed explanation of the methods. We took a thorough history, including personal information, during the initial assessment. Past medical and past surgical history Family history: The patient may have a history of diabetes, autoimmune diseases, or other comorbid conditions such as cardiac disease. Clinical examination: The thyroid gland involves a comprehensive neck examination to detect any abnormalities in the thyroid gland's size, shape, or consistency and the surrounding lymph nodes. Investigations: The laboratory study included a complete blood picture (CBC), creatinine, BUN, GFR estimation for all participants, and the calculation of sodium, potassium, calcium, phosphorus, PTH, HCO3, AST, ALT, serum albumin, serum glucose level, and Kt/v for all patients in the study group. Following an overnight fast, we took blood samples from all patients in the study group's thyroid hormonal profile just before their midweek dialysis session. We took blood from all participants in the control group while they fasted. We isolated the plasma from the rest of the sample and stored it at -20 °CC until the hormonal test. Using a chemiluminescence immunometric assay, we measured serum-free T3, T4, and TSH. We conducted a serum iodine test on both the patient and control groups.

2.2.Administrative and Ethical Design: The Faculty of Medicine at Al-Azhar University provided official authorization. Al-Azhar University Hospitals, as well as Fayoum General Hospital, granted their official approval. The Faculty of Medicine's ethical committee (Institutional Research Board, IRB) approved. We collected the participants' informed consent prior to their inclusion in the investigation. Withdrawal from the trial did not influence participants' care during the investigation.

We used SPSS version 22 (SPSS Inc., Chicago, IL, U.S.A.) for data collection, tabulation, and statistical analysis. We were revising and programming. On a computer, key in data. We used the mean and standard deviation (SD) to represent quantitative data for parametric data and the median and range for non-parametric data. Regarding qualitative data, frequencies, and relative percentages were utilized. We assessed the normality of the data by employing Shapiro-Wilk's test. We processed the data using relevant statistical analyses of significance. We used the Mann-Whitney test and independent t-test to determine the difference between the quantitative variables of the two groups. We applied a paired t-test to compare two dependent groups of normally distributed variables. Fisher exact and the chi-square test (x2) were utilized to compute the variance among qualitative variables. We identified significant predictors of iodine levels in cases using the sequential regression analysis method. We conducted all statistical analyses with two-tailed significance levels. We considered p-values below 0.05 as significant, p-values equivalent to or under 0.001 as highly significant variance, and p-values above 0.05 as non-significant variance.

3. Results

Table 1. Demographic data of the two examined groups.

Variable		HD (n=75)	Controls (n=25)	t/χ^2	p
Age (years) Mean ± SD		48.57 ± 7.29	46.82 ± 6.53	1.55	.124
Sex	Male	41 (54.7%)	12 (48%)	.335	.563
	Female	34 (45.3%)	13 (52%)		
BMI (kg/m^2) Mean ± SD		26.12 ± 3.57	25.43 ± 2.96	1.29	.199
Duration of disease (years) Mean ± SD		7.86 ± 3.17	---	--	--

There was no significant variance among the two examined groups concerning age, sex, and BMI Table 1.

VARIABLES	HD (N=75)	CONTROLS (N=25)	T	P
HR (BEAT/MIN) MEAN± SD	81.64 ± 8.83	75.81 ± 6.44	3.84	<0.001
SBP (MMHG) MEAN± SD	134.5 ± 8.45	128.42 ± 7.78	4.58	.001
DBP (MMHG) MEAN± SD	80.33 ± 7.92	77.26 ± 6.94	2.52	.013

Table 2. Vital signs among the 2 groups.

There was a significant variance among 2 examined groups concerning heart rate, SBP, and DBP Table 2.

Table 3. Laboratory parameters between the two studied group.

	HD (n=75)	Controls (n=25)	T	p
Hemoglobin (g/dl) Mean ±SD	10.32 ± 1.24	11.88 ± 1.69	6.45	<0.001
TLC (x103/L) Mean ±SD	7.24 ± 1.83	7.86 ± 2.99	1.53	.128
PLT (x103/L) Mean ±SD	258.24 ± 86.95	273.44 ± 35.92	1.4	.164
RBS (mg/dl) Mean ± SD	136.14 ± 19.61	125.57 ± 16.84	3.54	.001
Creatinine (mg/dl) Mean ±SD	5.68 ± 2.34	0.825 ± 0.173	18	<0.001
Urea (mg/dl) Mean ±SD	138.24 ± 39.18	19.32 ± 5.27	26	<0.001
GFR (mL/min/1.73m2) Mean ±SD	11.87 ± 3.72	102.19 ± 14.42	53	<0.001
ALT (U/L) Mean ±SD	28.42 ± 13.56	30.38 ± 11.60	.951	.343
AST (U/L) Mean ±SD	32.49 ± 13.01	29.38 ± 10.9	1.59	.115
Albumin (g/dl) Mean± SD	3.45 ± 0.667	4.34 ± 0.382	8.34	<0.001

There was significant variance among 2 examined groups concerning hemoglobin, creatinine, urea, GFR, and albumin Table 3.

Table 4. Electrolytes parameters among 2 examined groups.

	HD (n=75)	Controls (n=25)	t	p
Sodium (mEq/L) Mean ±SD	136.61 ± 5.27	138.74 ± 2.56	3.15	.002
Potassium (mEq/L) Mean ±SD	4.72 ± 0.643	4.78 ± 0.684	.554	.581
Calcium (mg/dl) Mean ±SD	8.68 ± 1.34	9.38 ± 0.724	2.96	.004
Phosphate (mg/dl) Mean ±SD	5.24 ± 1.08	4.93 ± 0.754	2.04	.043
PTH (ng/L) Mean ±SD	423.7 ± 323.4	87.38 ± 35.91	8.9	<0.001
Bicarbonate (nmol/L) Mean ±SD	23.4 ± 2.58	24.1 ± 2.33	1.74	.083

That there was a significant variance among 2 examined groups concerning sodium, calcium, phosphate and PTH. Table 4.

Table 5. Thyroid function distribution between the two studied groups

	HD (n=75)	Controls (n=25)	t	p
TSH (mU/L) Mean ± SD	4.16 ± 1.84	1.85 ± 0.46	MU 634	<0.001
Free T4(pmol/L) Mean ± SD	14.23 ± 3.18	16.42 ± 1.91	1.2	.233
Free T3(pmol/L) Mean ± SD	4.73 ± 1.59	5.49 ± 1.14	2.2	.030
Iodine (µg/L) Mean ± SD	67.52 ± 18.3	72.46 ± 14.75	1.22	.224

There was a significant disparity in TSH, free T3, and free T4, but not in iodine, among the two groups that were investigated. Table 5.

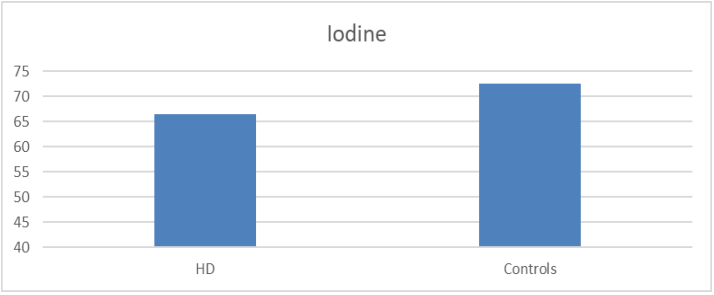

Figure 1. Iodine distribution between two studied groups.

Table 6. Correlation between creatinine with other parameters among HD patients.

Variable	Creatinine	
	r	P
Age	0.184	0.204
BMI	0.193	0.178
Disease duration	0.318	0.021
Hb	-0.365	0.023*
PLT	0.297	0.074
RBS	0.245	0.108
Albumin	-0.474	0.006*
ALT	0.187	0.183
AST	0.313	0.062
TSH	0.409	0.037*
Free T4	-0.512	<0.001*
Free T3	-0.491	<0.001*
Iodine	0.363	0.035*

*p<0.001

There was a positive significant association among creatinine with disease period, TSH & iodine. Meanwhile, there is a negative significant connection among creatinine with hemoglobin, albumin, free T4, and free T3. table 6.

Table 7. Correlation between iodine with thyroid function among HD patients.

Variable	Iodine	
	r	P
TSH	-0.439	0.001*
Free T4	0.411	0.007*
Free T3	0.474	0.004*

*p<0.001

There was a positive significant correlation among iodine with free T4 and free T3. Meanwhile, there is a negative significant association among iodine with TSH. Table 7.

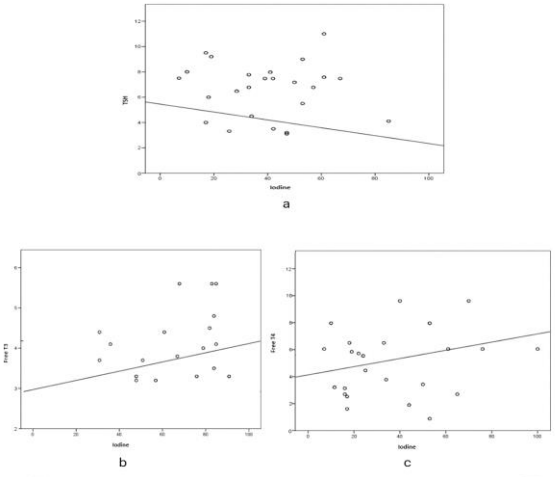

Figure 2. This figure showed that a): Negative significant correlation between iodine with TSH, b): Positive significant correlation between iodine with free T3 and c): Positive significant correlation between iodine with free T4.

4. Discussion

The decline in glomerular filtration rate and impairment of kidney function are hallmarks of the pathophysiological disorders known collectively as chronic kidney disease. There were 1.2 million deaths caused by CKD worldwide. Deaths from chronic kidney disease rose by 41.5 percent worldwide between 1990 and 20Oxidative stress, a significant factor in the development and progression of illness and its consequences, is present in individuals with chronic kidney disease (CKD). es. [1]

It has been known for quite some time that the thyroid gland and the kidney play an essential role in each other's functioning. Thyroid dysfunction affects renal physiology and development; kidney illness may cause thyroid dysfunction. Several thyroid hormones are primarily metabolized, degraded, and excreted by the kidney. [7]

Our research found that in terms of age, sex, and BMI, there was no significant variance among the two groups. The mean duration of diseases was 7.8 years. In a study by Punekar et al., 75 people took part, with 39 (52%) men and 36 (48%) women with CKD cases and 38 (50.7% of the participants) men and 37 (49.3% of the participants) women being healthy controls. There was no statistically significant difference in age between the two groups (p = 0.2818), with a mean age of 45.93 + 12.03 years for controls and 48.21 + 13.76 years for cases. [8]

Our study revealed a statistically significant distinction among both groups concerning heart rate, SBP, and DBP. In line with our results, Adani et al. found that among the causes of kidney failure, hypertensive renal disease ranked second. Out of the total number of patients, they identified hypertension as the primary cause of kidney failure in one hundred individuals (33.22 percent). [9]

Our results agreed with the study of Perez-Gurbindo et al. (2021, as they reported a significant difference between the hemodialysis and control groups regarding SBP and DBP.

In the study of Adani et al., hypertensive renal disease was the second most prevalent cause of kidney failure among individuals. The study found that hypertension was the primary cause of kidney failure in 100 individuals or 33.22 percent of the total.

The present research demonstrated significant differences between the two groups' hemoglobin, creatinine, urea, glomerular filtration rate, and albumin levels. Sodium, calcium, phosphate, and parathyroid hormone (PTH) levels differed substantially across both groups. [9]

By our outcomes, Kashif et al. found that the average blood urea concentration in the cases was 153.94 ± 72.50 mg/dl, whereas in the control group, it was 22.91 ± 7.36 mg/dl. Similarly, the average serum creatinine concentration was 8.76 ± 3.48 mg/dl in the cases and 0.75 ± 0.31 mg/dl in the controls. The unpaired t-test revealed that blood urea and serum creatinine had two-tailed p-values less than 0.0001 when comparing the case and control groups. [10]

Regarding TSH, free T3, and free T4, there were substantial variations among the two groups in our investigation. However, we observed no significant variations in iodine across both groups.

It was backed up by Kashif et al., who found that the average levels of TT3, TT4, and TSH in the patients' blood were 40.77 ± 12.58 (ng/dl), 6.61 ± 3.06 (µg/dl), and 16.92 ± 26.97 (µIU/ml), respectively. In the control group, these concentrations were 109.27 ± 22.26 (ng/dl), 8.94 ± 1.91 (µg/dl), and 2.29 ± 1.24 (µIU/ml), respectively. The results of the unpaired t-tests between the case and control groups in TT3 and TT4 were very significant, with a two-tailed p-value of less than 0.0001, but the results of the test for TSH were substantial, with a two-tailed p-value of 0.0002.

In the Takeda et al. study, patients receiving regular dialysis treatment may develop iodine-induced hypothyroidism even in the absence of apparent underlying thyroid disease. [11]

Lower iodine levels in those on hemodialysis than controls may also be associated with lower dietary iodine intake, owing to dietary restrictions on milk and dairy products, which are critical sources of iodine in Western diets. [12]

Subclinical hypothyroidism is the most prevalent consequence of CKD related to thyroid function, and its incidence increases consistently with the fall in glomerular filtration rate. High levels of inorganic iodine (iodide) in the blood may cause the thyroid to get bigger and stop making hormones (the Wolff-Chaikoff effect). This can happen because people with chronic renal failure have trouble getting rid of iodine through their urine. In that case, excess iodine can cause hypothyroidism and goiter in individuals with chronic renal failure due to impaired renal iodide excretion. [13]

We got the same results as Novakova et al., who looked at serum iodine and bromine levels in chronic hemodialysis patients. They found that people on hemodialysis had normal serum iodine levels, a little lower than controls but not significantly different (67.6 ± 17.1 µg/L vs. 72.2 ± 14.8 µg/L; p = 0.1252). [14]

In our results, we found that there was a significant positive association between creatinine, disease period, TSH, and iodine. Meanwhile, creatinine has a significant negative association with hemoglobin, albumin, free T4, and free T3. A significant positive relationship existed between iodine, free T4, and free T3. Meanwhile, there was a significant negative association between iodine and TSH.

The study of Adani et al. supported our results by finding a correlation between hypothyroidism risk and a decreased creatinine level. [9] There was a significant difference in creatinine levels between individuals who had overt hypothyroidism and those with euthyroidism or subclinical hypothyroidism. This goes against what Bamashmoos et al. and Kaur et al. found in earlier studies, which said that a higher creatinine level was caused by overt hypothyroidism. This finding needs to be investigated further to ensure it is accurate. [15,16] Several factors, such as the fact that these mentioned studies analyzed a smaller sample size than their study, cases with a history of hypothyroidism, and cases not routinely receiving hemodialysis, explain this variance.

5. *Conclusion*

Our study revealed a significant difference between the two groups concerning TSH, free T3, and free T4. The two groups did not significantly differ in terms of iodine. A positive and significant correlation was also found between iodine levels and free T4 and T3. Meanwhile, there is a significant negative correlation between iodine values and TSH. Individuals with CKD were at risk of thyroid hypofunction, irrespective of their mode of treatment.

Disclosure

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Authorship

All authors have a substantial contribution to the article

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Conflicts of interest

There are no conflicts of interest.

References

1. Romagnani P, Remuzzi G, Glassock R, et al. Chronic kidney disease. Nat Rev Dis Primers. 2017;3:17088.
2. Himmelfarb J, Vanholder R, Mehrotra R, Tonelli M. The current and future landscape of dialysis. Nat Rev Nephrol. 2020;16(10):573-585.
3. Rohner F, Zimmermann M, Jooste P, et al. Biomarkers of nutrition for development--iodine review. J Nutr. 2014;144(8):1322S-1342S.
4. Leung AM, Braverman LE. Consequences of excess iodine. Nat Rev Endocrinol. 2014;10(3):136-142.

5. Campi I, Dell'Acqua M, Stellaria Grassi E, Cristina Vigone M, Persani L. Unusual causes of hyperthyrotropinemia and differential diagnosis of primary hypothyroidism: a revised diagnostic flowchart. Eur Thyroid J. 2023;12(4):e230012.
6. Jusufovic S, Hodzic E. Functional Thyroid Disorders Are More Common in Patients on Chronic Hemodialysis Compared with the General Population. Mater Sociomed. 2011;23(4):206-209.
7. Da Costa AB, Pellizzari C, Carvalho GA, et al. High prevalence of subclinical hypothyroidism and nodular thyroid disease in patients on hemodialysis. Hemodial Int. 2016;20(1):31-37.
8. Da Costa AB, Pellizzari C, Carvalho GA, et al. High prevalence of subclinical hypothyroidism and nodular thyroid disease in patients on hemodialysis. Hemodial Int. 2016;20(1):31-37.
9. Adani AA, Siyad MO, Adan AM, Jeele MOO. Prevalence and Determinants of Hypothyroidism in Patients on Routine Hemodialysis in Somalia: A Cross-Sectional Study. Int J Gen Med. 2023;16:905-913.
10. 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.
11. Takeda S, Michigishi T, Takazakura E. Iodine-induced hypothyroidism in patients on regular dialysis treatment. Nephron. 1993;65(1):51-55.
12. van der Reijden OL, Zimmermann MB, Galetti V. Iodine in dairy milk: Sources, concentrations and importance to human health. Best Pract Res Clin Endocrinol Metab. 2017;31(4):385-395.
13. Kutlay S, Atli T, Koseogullari O, Nergizoglu G, Duman N, Gullu S. Thyroid disorders in hemodialysis patients in an iodine-deficient community. Artif Organs. 2005;29(4):329-332.
14. Novakova G, Bonev P, Duro M, et al. Serum Iodine and Bromine in Chronic Hemodialysis Patients-An Observational Study in a Cohort of Portuguese Patients. Toxics. 2023;11(3):247.
15. Bamashmoos SA, Al-Nuzaily MA, Al-Meeri AM, Ali FH. Relationship between total homocysteine, total cholesterol and creatinine levels in overt hypothyroid patients. Springerplus. 2013;2:423.
16. 16. KAUR, Vaneet; SINGH, Kamaljit; VERMA, Minni. Changes in biochemical markers of renal function in subclinical and overt hypothyroidism. Int J Bioassays, 2015, 4.4: 3799-3802.