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Prevalence of Echocardiographic Abnormalities and its Relation to Thyroid Abnormalities among Hemodialysis Patients

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Abstract

Background and aim: The kidney is involved in the regulation of thyroid hormones metabolism. Numerous abnormalities of thyroid hormones in end-stage kidney disease (ESKD) have been described. The aim of our study was to estimate the prevalence of thyroid dysfunction and its relation to Echocardiologic abnormalities in patients with end stage kidney disease.

Material and method: 60 patients with ESKD on regular hemodialysis were recruited for this study. All subjects were investigated with Transthoracic Echocardiography and laboratory tests to determine thyroid function, including: serum triiodothyronine (T3), free T3, serum thyroxine (T4), freeT4, thyroid-stimulating hormone (TSH). Results were compared with the same measurements in 20 normal control subjects.

Results: The prevalence of thyroid abnormalities (78.3%) and low T3 (70%). T3 and T4 were significantly low (68.6 ± 21.4 ng/dL, 5.1 ± 2.1 µg/dL) compared to control group (104.1 ± 20.2 ng/dL, 7.8 ± 2.0 µg/dL). The prevalence of Echocardiologic abnormalities (71.7%) and left ventricular hypertrophy (70%). There was significant low T3 in patients with Echocardiologic abnormalities. 90.7% in patients with Echocardiologic abnormalities and 90.5% of patients with left ventricular hypertrophy had thyroid abnormalities. T3 levels show negative correlations with LVMI, LVEDD, SWT and PWT and a positive correlation with LVEF. No significant correlations between FT3, FT4, TSH and Echocardiographic data can be observed detected.

Conclusion: Echocardiographic abnormalities were linked by thyroid abnormalities especially low T3. Screening for early detection of thyroid disorders is useful and important.

Keywords

Echocardiologic abnormalities, Thyroid abnormalities, ESKD

Introduction

The kidney plays an important role in the metabolism, degradation and excretion of several thyroid hormones. It is not surprising, therefore, that impairment in kidney function leads to disturbed thyroid physiology [1]. Chronic renal failure affects thyroid function in multiple way, including low circulating thyroid hormone concentration, altered peripheral hormone metabolism, disturbed binding to carrier proteins, possible reduction in tissue thyroid hormone content, and increase iodine store in thyroid glands. Both plasma triiodothyronine (T3) and thyroxin (T4) are reduced [2].

Thyroid function has been extensively evaluated in patients with chronic kidney disease, however the results are variable, primary hyperthyroidism is extremely rare, while the prevalence of hypothyroidism is increased in patients with chronic renal failure [3]. The prevalence of hypothyroidism was increased in persons with reduced glomerular filtration rate (GFR), ranging from 5.4% for persons with estimated GFR ≥ 90 mL/min/1.73 m^2 to more than 20% in persons with estimated GFR < 60 mL/min/1.73 m^2 [4]. In spite of a large number of previous investigations of thyroid function abnormalities in end stage kidney disease (ESKD) patients, the echocardiographic findings were not included in the analysis in most of these studies. Therefore, the aim of this study was to evaluate thyroid function abnormalities and its relation to echocardiographic abnormalities in ESKD patients.

Patients and Methods

Sixty clinically stable end stage kidney diseasepatients were enrolled in this cross sectional study. All patients were under regular hemodialysis at Al-Azhar University Hospital (Hussein Hospital) between October 2014 and Jun 2015 (4 hours session, thrice weekly), using polysulfone high flux dialyzer 1.6 m^2 surface area, with dialysate flow 500 ml/min and dialysate calcium concentration 1.25 mmol/l, using heparin as anticoagulant with tailored doses according to each case and bicarbonate based dialysate. The adequacy of dialysis was assessed using Kt/V formula. 20 normal individuals were used as control group.

Patients with the following criteria were excluded: patients younger than 18 year; patients with congestive heart failure, respiratory failure, and liver cirrhosis; patients who had a history of

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Table 1: Demographic and clinical characteristics for patients.

	Parameters	N (%)
Etiology of renal diseases	Hypertension	19 (31.7%)
	Diabetes mellitus	16 (26.7%)
	Chronic glomerulonephritis	4 (6.7%)
	Analgesic nephropathy	5 (8.3%)
	Others*	10 (16.6%)
	Unknown	6 (10%)
Comorbid diseases	Diabetes mellitus	18 (30%)
	Liver disease	12 (20%)
	Chronic lung disease	5 (8.3%)
	Coronary arterial disease	8 (13.3%)
	Prepheral arterial disease	5 (8.3%)
Medications	RAS blockers	27 (45%)
	CCB	21 (35%)
	BB	17 (28.3%)
	Aspirin	9 (15%)
	Vitamin D	33 (55%)
	ESA	39 (65%)
Echocardiographic abnormalities	**43 (71.7%)**	
	Left ventricular hypertrophy	42 (70%)
	Left ventricular dilatation	13 (21.7%)
	Systolic dysfunction	25 (41.7%)
	Left atrial dilatation	33 (55%)
	Aortic annulus dilatation	19 (31.7%)
Thyroid functions abnormalities	**47 (78.3%)**	
	Low total T3	42 (70%)
	Low total T4	36 (60%)
	Elevated TSH	12 (20%)

SBP: Systolic Blood Pressure, DBP: Diastolic Blood Pressure, BMI: Body Mass Index, RAS: Renin-Angiotensin System, BB: Beta Blockers, CCB: Calcium Channel Blockers, ESA: Erythropiotin-Stimulating Agent.

*Other causes of renal failure include aquired obstructive uropathy, polysystic kidney disease, gouty nephropathy, chronic polynephritis, lupus nephritis.

peritoneal dialysis or kidney transplantation prior to hemodialysis; patients who have been receiving thyroid hormone replacement or suppressive therapy due to overt thyroid disease; patients who have been receiving medications affecting thyroid hormone levels, such as amiodarone, glucocorticoids, and lithium; patients who have active malignancy.

Each patient underwent complete history and physical examination with special emphasis on age, gender, body mass index, primary renal disease, comorbidities, and medications. Measurement of thyroid hormones was performed. As heparin may interfere with competitive assays, blood was drawn just before the start of hemodialysis procedure from the inserted dialysis needle or from the arterial port of dialysis circuit, before contact of blood with dialyzer and before heparin administration. This ensured that there was an interval of at least 48 hours since the last heparin application. The battery of tests thus included: TSH determined by immune radiometric assay, reference range (RR) 0.17-4.05 μIU/mL; total T4, RIA RR 5.4-12.4 μg/dL; total T3, RIA, RR 78-182 ng/dL; freeT4 measured by RIA, RR 0.89-1.79 ng/dL; free T3, RIA, RR 1.6-3.77 pg/dL (IMMUNOTECH s.r.o. -Radiovo1-102 27, Prague-Czech Republic). Transthoracic Echocardiography was performed on a non-dialysis day, close to the time of discharge based on the imaging protocol recommended by American Society of Echocardiography [5]. All patients were informed about the content of the study and gave their written approvals before enrollment. All procedures were performed in accordance with the ethical standards of Al-Azhar University's committee on human experiments.

Statistical Analysis

Statistical analysis was performed using SPSS for Windows, version 20.0 (SPSS, Inc.). Values were expressed as mean ± Standard deviation (SD) and as percentage for categorical parameters. Independent t-test was applied for comparison between two independent groups with parametrical data. Chi-square test was applied for estimating the occurrence of categorical variables. Pearson's correlation coefficient

Table 2: Distribution of thyroid funtion abnormalities among patients with echocardiologic abnormailties.

Echocardiographic findings		Thyroid function abnormalities					
		Normal		Abnormal			
	N	N	%	N	%	P	Sig
Echocardiographic abnormalities	43	4	9.3%	39	90.7%	0.002	HS
Left ventricular hypertrophy	42	4	9.5%	38	90.5%	0.004	HS
left ventricular dilation	13	1	7.7%	12	92.3%	0.199	NS
Systolic function	25	3	12%	22	88%	0.163	NS
left atrial dilatation	33	3	9.1%	30	90.9%	0.022	HS
Aortic annulus dilatation	19	2	10.5%	17	89.5%	0.186	NS

HS: Highly Significant, NS: Non Significant, Sig: Significant.

Table 3: Comparison between patients and controls as regards tested variables.

Parameters	Patients	Controls	P
Number of participants	60	20	
Age/year	46.7 ± 13.2	43.5 ± 15.7	0.372
Gender (male/female)	35/25	10/10	
SBP (mmHg)	137.6 ± 25.4	127.5 ± 14.7	0.094
DBP (mmHg)	82.8 ± 13.2	81.2 ± 6.6	0.611
BMI (kg/m²)	27.2 ± 7.7	28.1 ± 5.6	0.627
TSH (μIU/mL)	3.1 ± 4.2	1.6 ± 1.3	0.122
T3 (ng/dL)	68.6 ± 21.4	104.1 ± 20.2	0.001
FT3 (ng/dL)	3.6 ± 0.75	3.5 ± 1.3	0.376
T4 (μg/dL)	5.1 ± 2.1	7.8 ± 2.0	< 0.001
FT4 (ng/dL)	1.0 ± 0.5	1.1 ± 0.4	0.641
LAD (cm)	3.9 ± 0.7	3.4 ± 0.6	0.003
LVMI (g/m²)	125.4 ± 46.4	79.7 ± 21.4	0.001
LVEDD (cm)	5.2 ± 0.8	4.8 ± 0.5	0.060
LVESD (cm)	4.7 ± 3.8	4.2 ± 1.7	0.577
SWT (cm)	1.1 ± 0.2	0.9 ± 0.15	0.001
PWT (cm)	1.0 ± 0.2	0.9 ± 0.17	0.001
AOD (cm)	2.8 ± 0.4	2.9 ± 0.4	0.683
LVEF (%)	56.7 ± 13.1	65.8 ± 6.7	0.004

SBP: Systolic Blood Pressure, DBP: Diastolic Blood Pressure, BMI: Body Mass Index, LAD: Left Atrium Diameter, LVMI: Left Ventricular Mass Index, LVEDD: Left Ventricular End Diastolic Diameter, LVESD: Left Ventricular End Systolic Diameter, SWT: Septal Wall Thickness, PWT: Posterior Wall Thickness, AOD: Aortic Orifice Diameter, LVEF: Left Ventricular Ejection Fraction.

was used to test the correlation between thyroid function tests and echocardiographic parameters. P-values < 0.05 were considered statistically significant.

Results

Characteristics of the 80 participants are summarized in the table 1, table 2 and table 3. The major etiology of ESKD is hypertension (31.7%) and the major comorbid disease is diabetes mellitus (30%). T3 and T4 levels are significantly low (68.6 ± 21.4 ng/dL, 5.1 ± 2.1 μg/dL) as compared to control (104.1 ± 20.2 ng/dL, 7.8 ± 2.0 μg/dL). No significant differences are detected as regards TSH, FT3, and FT4 levels. The left ventricular mass index (LVMI), septal wall thickness (SWT), posterior wall thickness (PWT) and left atrium diameter (LAD) are significantly high as compared to control. On the other hand, the left ventricular ejection fraction (LVEF) is significantly low (Table 2). Out of 60 patients, 43 patients (71.7%) haveechocardiographic abnormalities, 42 of them (70%) haveleft ventricular hypertrophy, 13 patients (21.7%) haveleft ventricular dilatation, 25 patients (41.7%) havesystolic dysfunction, 33 patients (55%) haveleft atrial dilatation and 19 patients (31.7%) haveaortic annulus dilatation. As regards thyroid functions, 78.3% of the patients have thyroid function abnormalities, 70% have low total T3, 60% have low T4 and 20% have elevated TSH (Table 1). Significant numbers of patients with echocardiographic abnormalities (39 out of 43) have thyroid function abnormalities (90.7%) with a P-value < 0.005. thirty eight out of forty two of patients with left ventricular hypertrophy (90.5%) have thyroid function abnormalities (P < 0.005), 30 out of 33 patients with left atrial dilatation (90.9%) have thyroid function abnormalities (P < 0.05) (Table 3). The patients with echocardiographic abnormalities have a significantly low T3

Table 4: Comparison between patients with echocadiographic abnormalities and those without as regards to tested variables.

	Echocardiographic results				
	Normal		Abnormal		
	Mean	± SD	Mean	± SD	P
Age (Year)	43.4	14.8	48	12.4	> 0.05
BMI	26.2	5.3	28	8.2	> 0.05
Dialysis period (Year)	6	4.2	5.8	3.8	> 0.05
SBP (mmHg)	140	26.3	136	25	> 0.05
DBP (mmHg)	83.5	13.6	82.5	13	> 0.05
Heart rate (b/m)	72.3	4.7	73.3	5	> 0.05
Creatinine (mg/dl)	9.8	3	9.3	3	> 0.05
Urea (mg/dl)	112	51.9	137	47.7	> 0.05
Kt/V	1.5	0.2	1.4	0.19	> 0.05
Haemoglobin (g/dl)	9.8	1.8	10	1.7	> 0.05
Calcium (mg/dl)	9.2	1	9.8	1.7	> 0.05
Phosphorus (mg/dl)	5.4	1.6	5.5	1.6	> 0.05
alkaline phosphatase (mg/dl)	184	145	164	140	> 0.05
Parathyroid hormone (Pg/ml)	251	143	237	236	> 0.05
Cholesterol (mg/dl)	171	29	173	41	> 0.05
Triglycride (mg/dl)	156	49	154	56	> 0.05
T3 (ng/dL)	81.1	20	64.2	20	< 0.005
T4 (µg/dL)	5.26	1.7	5.09	2.1	> 0.05
TSH (µIU/mL)	2.77	1.6	3.58	4.7	> 0.05
FT3 (ng/dL)	3.88	0.5	3.80	0.6	> 0.05
FT4 (ng/dL)	1.10	0.18	1.14	0.25	> 0.05

BMI: Body Mass Index, SBP: Systolic Blood Pressure, DBP: Diastolic Blood Pressure, TSH: Thyroid Stimulating Hormone, T3: Triiodothyronine, T4: thyroxine, FT3: Free Triiodothyronine, FT4: Free Thyroxine.

Table 5: Correlation between thyroid function tests and echocardiographic data.

		T3	T4	TSH	FT3	FT4
Left ventricular mass index	r	- 0.357	0.038	- 0.008	0.019	- 0.073
	Sig.	0.005	0.771	0.954	0.887	0.579
Left ventricular end systolic diameter	r	- 0.230	- 0.071	- 0.036	- 0.167	0.042
	Sig.	0.077	0.588	0.783	0.202	0.748
Left ventricular end diastolic diameter	r	- 0.356	- 0.050	- 0.077	- 0.136	0.053
	Sig.	0.005	0.702	0.559	0.300	0.690
Ejection fraction	r	0.300	0.260	- 0.044	0.178	0.042
	Sig.	0.020	0.045	0.736	0.173	0.752
Septal wall thickness	r	- 0.293	0.172	0.041	0.152	- 0.011
	Sig.	0.023	0.189	0.756	0.245	0.936
Posterior wall thickness	r	- 0.328	0.080	0.092	0.042	- 0.141
	Sig	0.011	0.546	0.483	0.750	0.281
Left atrial diameter	r	- 0.201	- 0.016	0.056	- 0.029	0.099
	Sig.	0.125	0.906	0.671	0.827	0.453
Aortic orifice diameter	r	- 0.210	0.201	- 0.046	0.058	0.256
	Sig.	0.107	0.123	0.729	0.659	0.048

as compared to patients with normal echocardiographic studies (Table 4). T3 levels show negative correlations with LVMI, LVEDD, SWT and PWT and a positive correlation with LVEF. Moreover, T4 levels have a positive correlation with LVEF. No significant correlations between FT3, FT4, TSH and Echocardiographic data can be observed detected (Table 5).

Discussion

Most studies of thyroid hormones in clinically euthyroid patients with varying degrees of chronic renal failure showed significant decrease in total T3, total T4 and free T3 (FT3) levels compared with control [6,7]. A low T3 and T4 syndrome is evident when glomerular filtration rate (GFR) is reduced below 30 ± 16 ml/min [7]. Usually there is more distinct suppression of T3 than of T4 [8]. The concentrations of reverse T3 (rT3), the inactive metabolite of T4 in plasma are usually low but normal or even elevated values have been reported by some authors [8,9]. Similar to findings of this study, the previous studies have demonstrated that the most common thyroid imbalance in patients on hemodialysis was low T3 syndrome, while FT3 levels generally remain within the normal limits [6,10]. Ozen, et al. [11] demonstrated that up to 70% of stage 5 CKD patients have low T3. Soffer, et al. found that low serum levels of thyroxine in 69.5% of the patients studied and of triiodothyronine (T3) in 46.5% and 13.0% had high levels of TSH [12].

The reduction in thyroid hormone may be due to the effect of chronic renal failure on the thyroid hormones which include altered peripheral metabolism like impairment of peripheral deiodination of T4 which is the mainsource of T3. Due to reduced deiodinase activity, tissue and circulating levels of the active form of the thyroid hormone and T3are low in kidney failure [13]. Toxic uremic solute such as urea, creatinine, indoles and phenols inhibits protein binding of T4 [8]. Furthermore, systemic inflammation [14,15] and metabolic acidosis [14] may alter thyroid function in CKD patients. The concentration of serum iodine in patients with CKD is higher due to lower iodine clearance caused by reduced glomerular filtration. Elevated levels of serum inorganic iodine in patients with CKD may potentially block thyroid hormone synthesis (Wolff–Chaikoff effect) [16]. In contrast, the thyroid-pituitary feedback loop seems to remain intact, because steady-stateplasma TSH remains substantially normal [3]. The Reduced renal clearance may contribute to delayed recovery, since TSH and TRH are normally cleared by the kidney [17]. Low T3 associated with endothelial dysfunction, a harbinger of atherosclerosis, in stage 3 and 4 CKD patients [10], as well as cardiomyopathy [18] and with high risk of death in stage 5 CKD patients [19]. The cardiovascular mortality in these individuals is 10- to 20-fold more frequent than in the general population [20]. Although more than 50% of the individuals starting a dialysis program present some type of pre-existent cardiovascular disease, the traditional risk factors for cardiovascular disease do not completely explain this excess risk, which seems to be influenced by the so-called non-traditional risk factors associated with CKD. This set of factors accelerates the course of coronary artery disease (CAD) and is associated with a higher prevalence of ventricular hypertrophy, myocardial fibrosis, valvulopathies, arrhythmias and sudden death [21].

The cardiomiopathy of the patient undergoing dialysis is mainly due to the presence of ischemic cardiopathy and morphofunctional alterations of the left ventricle (LV) in response to pressure and volume overload. The physiopathology of the transformations induced by uremia in the left ventricular chamber is complex and multifactorial [22]. Arteriovenous fistula, which was used for vascular access in patients with ESRD, increases stroke volume load on the left ventricle and this may contributes to LVH and may results in LV systolic and diastolic dysfunction with time [23]. The left ventricular dysfunction and hypertrophy were most common echocardiographic findings and statistically correlated with anemia and presence of hypertension [24,25].

In current study, significant numbers of patients have echocardiologic abnormalities (71.7% of patients), majority of these patients have left ventricular hypertrophy (70%), 55% have left atrial dilatation and 41.7% have systolic dysfunction. Similar findings have been demonstrated by Foley, et al. [26] they reported that abnormalities of left ventricular structure and functions were very frequent on baseline echocardiography: 73.9% had left ventricular hypertrophy, 35.5% had left ventricular dilatation and 14.8% had systolic dysfunction in ESRD patients. Singh, et al. [27] reported that LVH in 76.92%, diastolic dysfunction in 72% but did not find systolic dysfunction in CKD patients. Zoccali, et al. [28] found 77% of patients had LVH and 22% had systolic dysfunction by in haemodialysis patients. Agarwal, et al. [29] had observed diastolic dysfunction in 60% and systolic dysfunction in 15% of patients. In addition, Laddha, et al. found that in hypertensive patients with ESRD LVH was present in 87.5%, diastolic dysfunction was present in 72.9% as measured by abnormal E/A ratio, systolic dysfunction as measured by reduced LVEF was present in 29.2% and pericardial effusion observed in 14.6%. In normotensive patient with ESRD LVH was present in 45.5%, diastolic dysfunction was present in 40.9%, and systolic dysfunction was present in 13.6% and pericardial effusion observed in 13.6% patients [25].

Left ventricular dysfunction and LVH are considered a maladaptive response to hypertension, volume overload, anaemia,

hyperphosphataemia, inflammation and other risk factors [30]. It is important to recognize that part of the alteration in the geometry of LV in CKD patients can be related to the moment at which the echocardiogram was performed. Shortly after the dialysis session, it is common to see a reduction in diastolic diameter of the LV and an increase in the thickness of the LV wall as a pure consequence of volume depletion by ultrafiltration. While, examination shortly before beginning the session can shows LV dilatation that will be 'converted' into concentric at the end of the session. Such fluctuation could be minimized by performing the echocardiogram during the interdialytic period [31].

Thyroid diseases can result in a wide range of cardiovascular manifestations and complications like atrial fibrillation, cardiomyopathy, and congestive heart failure. It is well known that T3 increases cardiac output by affecting tissue oxygen consumption, vascular resistance, blood volume, cardiac contractility, and heart rate [32]. Therefore, low T3, even in the normal range, has been suggested to be associated with various cardiovascular diseases in ESRD patients, which has been clarified by several previous studies [9,16,33,34]. Independently from the presence of primary thyroid hypofunction and differently from other organs, the heart is particularly vulnerable to reductions in biologically active T3 in plasma because cardiomyocytes have a negligible capability to generate T3 from locally converted precursor T4. Consequently, when circulating T3 is low, the myocardium may become relatively hypothyroid [35].

The large numbers of previous investigations were on the prognostic value of low T3 for all-cause and cardiovascular (CV) mortality in ESRD patients and echocardiographic findings were not included in the analysis in most of these studies. In this study, we have found majority of patients who have echocardiographic abnormalities (90.7%) have thyroid function abnormalities. The thyroid abnormalities are frequent among patients with left ventricular hypertrophy (90.5%). Moreover, in concordance with our observations previous studies [8,35-38] have been demonstrated that LV systolic function was depressed and LVMI was increased in the presence of low T3. Correction of malnutrition, anemia, acidosis, and thyroid hormone has associated with improvement of LVMI, LVEF [39,40]. Therapeutic use of thyroid hormone has not been adequately studied [41]. Finally, the small number of patients was the first limitation in this study. Diastolic dysfunctions are a frequent finding among ESKD patients who have thyroid abnormalities and must be included for better assessment.

Conclusions

The majority ofhemodialysis patients have echocardiographic abnormalities. The left ventricular hypertrophy and left atrial dilatation were the most common abnormalities. Echocardiographic abnormalities were linked by thyroid abnormalities especially low T3. Screening for early detection of thyroid disorders is useful and important. This is just a preliminary report regarding thyroid dysfunction and its cardiovascular complications and future studies with a larger sample size may be considered.

Conflict of Interest

There is no conflict of interest.

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