Relationship Between Thyroid Hormones and Left Ventricular Mass in Peritoneal Dialysis Patients

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Original Paper

Introduction. Non-thyroidal illness is prevalent in patients with advanced stages of chronic kidney disease and could be considered as a risk factor for cardiovascular mortality; this relation is partially explained by malnutrition and the concomitant condition of high inflammation. This study is designed to investigate the relationship between left ventricular mass and thyroid hormone abnormalities and evaluate this relationship after adjustment of inflammatory factors and nutritional status in peritoneal dialysis patients.
Methods. A total of 71 patients undergoing maintenance peritoneal dialysis were included. Serum concentration of total and free triiodothyronine (fT3), total and free thyroxine (fT4), and TSH were measured. LV mass index and the structural properties of heart including LVEDD, LVESD, PWD, and EF were assessed by transthoracic echocardiography. The Surrogates of Inflammation, including IL-6, albumin, and hs-CRP were measured. The nutritional status of patients was assessed by one point SGA scoring and biochemical data. The relation between thyroid hormones and echocardiographic variables, inflammatory and nutritional markers was determined.
Results. LVMI was significantly higher in the group with lower fT3 levels. Linear regression analyses showed statistically significant univariate association between fT3, tT3, and tT4; and LVMI. In multiple linear regression analysis adjusted for albumin, hs-CRP, IL-6, and ferritin; only fT3 level had a meaningful negative correlation ($P < .05$) with LVMI, free T3 level was positively correlated with rGFR ($P < .05$, c = 0.39) and KT/V ($P < .05$, c = 0.27).
Conclusion. Low fT3 level was negatively and significantly associated with LVML even after adjustments for known risk factors in peritoneal dialysis patients.

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INTRODUCTION

Alterations in the level of thyroid hormones has been observed in many patients with chronic kidney disease (CKD) without a known underlying dysfunction in the hypothalamus–pituitary–thyroid axis and this condition increases the mortality risk in this population. It had been shown that Low thyroid hormone can directly result in a deterioration of both renal and cardiac functions.[1,2,3]

Previous studies have reported that low serum concentrations of free and/or total triiodothyronine, low serum levels of free and/or total thyroxine and low TSH level are present in 75%, 10% to 50%, and 10% of individuals with end-stage renal disease

(ESRD); respectively. However, serum levels of reverse triiodothyronine do not increase.[4,5]

The suggested factors providing explanation about the high prevalence of nonthyroidal illness in patients with ESRD include: a persistent and chronic state of inflammation in the majority of these patients,[6] nutritional disorders and hypo albuminaemia which are common in patients with ESRD,[7] anemia,[8] and metabolic acidosis;[9] co-morbidities such as diabetes mellitus, infections and gout; and the use of certain drugs such as corticosteroids, amiodarone, propranolol, and lithium and phosphate binders such as calcium carbonate which may reduce the intestinal absorption of thyroxine.[10]

Cardiovascular mortality is the leading cause of death in patients with ESRD. Observational studies suggest that this finding has been partly attributed to the presence of nonthyroidal illnesses.[3]

Overt hypothyroidism contributes largely to structural alterations of heart and reduction in its inotropic and lusitropic properties.[12] previous studies suggested that Low levels of free triiodothyronine are associated with an increase in the left ventricular mass and reduction in left ventricular function in patients on dialysis.[13]

In addition, vascular calcification, which is prevalent in patients with ESRD; could be partially explained by the presence of nonthyroidal illness.[14,15] Low level of thyroid hormones is associated with endothelial dysfunction and in turn, atherosclerosis.[16]

The aim of this study was to evaluate the relationship between serum level of thyroid hormones and structural or functional echocardiographic properties of heart in peritoneal dialysis patients and investigating the association between these factors and some inflammatory markers (IL-6 and hs-CRP) and the nutritional status of this population. In addition,the association between residual renal function and peritoneal dialysis adequacy with above factors was evaluated.

MATERIALS AND METHODS

Study Population

A total of 71 ESRD patients undergoing peritoneal dialysis were included based on following criteria.

Inclusion criteria was considered: ESRD patients (15 to 68 years old) undergoing peritoneal dialysis for at least 3 months and free of comorbidities including infectious diseases, peritonitis, liver cirrhosis, advanced heart failure (NYHA class3, 4), and malignancies.

Exclusion criteria was considered: patients receiving lithium, amiodarone, propranolol, corticosteroids or other drugs which would interfere with thyroid function and those with clinical evidence of hypothyroidism.

Estimated sample size for a one-sample correlation test (Fisher's z test) was determined based on the previous studies[36] by STATA13 software.

Study protocol was reviewed and approved by ethics committee of KUMS (reference number: IR.KMU.REC.1395.723).

Laboratory Measurements

All blood samples were drown into vacationer tubes containing EDTA, and centrifuged within 30 min at 4 °C and the serum was stored at 80 °C. Serum ferritin, cholesterol, albumin, calcium, phosphate, hemoglobin, and intact parathyroid hormone were measured by standard methods in the same laboratory. Measurement of FT3, total T3 (TT3), free thyroxine (FT4), total T4 (TT4), and thyroid-stimulating hormone (TSH) was performed using the same method (electro chemiluminescence assay) and the same kit (commercially available Elecsys kit: Roche Diagnostic GmbH, Sandhofer strasse116) and by the same analyzer (Cobas 6000 Analyzer according to the manufacturer's instruction). Normal reference ranges for fT3, fT4, TSH, tT3, and tT4 were 2 to 4.4 pg/mL, 0.93 to 1.7 ng/dL, 0.27 to 4.2 μIU/mL, 0.8 to 2 ng/mL, and 5.1 to 14.1 μg/dL; respectively. Serum levels of IL-6 were measured using an enzyme linked immunosorbent assay with the use of a BT-Human IL-6 kit. Serum level of hs-CRP was measured by ELISA assay using Monobind hs-CRP kit.

Nutritional Assessment

Biochemical parameters of nutrition including serum albumin, creatinine, Hb, phosphate, cholesterol, and nPCR were measured. Anthropometric measurements including height, weight, and body mass index (BMI) were determined.

The SGA score was calculated once based on the history and physical examination as described by Destky *et al*.[17] The history focused on 7 variables: weight change in the last 6 months and 2 weeks

before the study, change in dietary intake, presence of GI symptoms, change in functional capacity, subcutaneous loss of fat (triceps, biceps, and the fat pads below the eyes), muscle wasting (through the examination of temples, clavicle, and shoulders), and edema. A seven point scoring system was applied to the above 7 variables. The patients were classified into 3 groups according to their scores: well nourished (score 1 to 14), mild to moderately malnourished (score 15 to 35) and severely malnourished (score 36 to 49).

Echocardiography

Transthoracic echocardiography was performed at the same participating local hospital. To decreased inter observer variability all patients were examined two times by two participant cardiologist and the mean amount of following variables were determined: left ventricular end-diastolic diameter (LVEDD), end-systolic diameter (LVESD), posterior (PWD), septal wall thickness, and LVM.

Normalized LVM was calculated using Devereux and Reickek formula.[18] Indexed for body surface area (BSA), LVH was determined as left ventricular mass index (LVMI) of > 115 g/m² for men and > 95 g/m² for women.

Statistical Analysis

Data were reported as mean ± standard deviation or percentage frequency. Correlations between continuous variables were analyzed using standard Spearman analysis. Data was expressed in terms of correlation coefficients (r) and *P* values.

The independent associations between LVMI and thyroid hormones, nutritional variables, IL-6, and CRP were analyzed using multiple linear regression analysis. Data was expressed in term of standard regression coefficients (c) and *P* values.

The characteristics of participants are presented across two fT3 strata based on its median value and compared with independent simple t test. For all the tests *P* value < .05 was considered to indicate a statistical significance.

RESULTS

The study population consisted of 71 PD patients with a mean age of 52.4 ± 15.7 years while 47.8% of the whole population was male. 50.7% of the patients were diabetic and 70.4% of them were hypertensive. None of the patients was a smoker at the time of the study. A total of 55 patients were being treated with erythropoietin analogues. The mean PD vintage was 31.07 ± 22.4 months. Demographical, clinical, and laboratory data of all patients are listed in Table 1.

Mean fT3 level was 2.20 ± 0.58 pg/mL while fT4 and TSH were 1.17 ± 0.27 ng/dL and 3.41 ± 1.08 mIU/mL, respectively. The mean levels of interlukin-6 and hs-CRP were, respectively; 40.61 ± 17.19 ng/L and 3.85 ± 3.15 µg/mL. 28.1% of patients were anuric and mean residual GFR of others was 3 ± 1.81 cc/min. Mean stdKT/V of the participants was 2.41 ± 0.82. Evaluation of the relationship between thyroid hormones and nutritional state showed a statistically significant correlation between tT3 and albumin (*P* < .05,

Table 1. Demographical, Clinical, and Laboratory Data of the Studied Patients

Parameter	Mean ± SD
Age, y	52.4 ± 15.17
Female sex (%)	51.1
Diabetes (%)	50.7
Hypertensive (%)	69.01
Dialysis Vintage, mo	31.07 ± 22.14
Weekly Kt/V	2.41 ± 0.82
BMI, Kg/m^2	22.84 ± 4.21
SGA Score	15.12 ± 4.96
nPCR, g/Kg/d	0.77 ± 0.20
rGFR, cc/min	3 ± 1.81
Cholesterol, mg/dL	163 ± 45.06
LDL, mg/dL	95.45 ± 32.43
TG, mg/dL	172.16 ± 85.94
Hb, g/dL	10.39 ± 1.54
SI, µg/dL	73.81 ± 34.62
TIBC, µg/dL	309.7 ± 71.28
Ca, mg/dL	8.64 ± 0.81
P, mg/dL	5.19 ± 1.33
PTH, pg/mL	201.58 ± 182.07
Ferritin, ng/mL	350.09 ± 239.81
Hs-CRP, µg/mL	3.85 ± 3.15
IL-6, ng/L	40.61 ± 17.19
Alb, g/dL	3.69 ± 0.51
TSH, µIU/mL	3.41 ± 1.08
tT3, ng/mL	91.25 ± 24.71
tT4, µg/dL	8.07 ± 1.81
fT3, pg/mL	2.2 ± 0.58
fT4, ng/dL	1.17 ± 0.27
EF (%)	52.47 ± 8.88
LVMI	113.79 ± 32.09
LVEDD, mm	49.65 ± 6.69
LVESD, mm	34.45 ± 7.19
PWD, mm	11.4 ± 2.27
MPI	0.46 ± 0.11

c = 0.27), tT3 and Hb ($P < .05$, c = 0.3), as well as, fT3 and Hb ($P < .05$, c = 0.25; Table 2).

The free T3 levels were positively correlated with rGFR ($P < .05$, c = 0.39), and KT/V ($P < .05$, c = 0.27). The same results were found for tT3 and rGFR ($P < .05$, c = 0.37), as well as, tT3 and KT/V ($P > .05$, c = 0.24, Table 3). No significant correlation was found between peritoneal properties and thyroid hormone levels (Table 3).

In the evaluation of thyroid hormones and inflammatory variables, a significant correlation was found between tT3 and albumin ($P < .05$, c = 0.28) but no significant correlation was detected between thyroid hormones and IL-6 nor hs-CRP (Table 4). Mean LVMI was equal to 113.79 ± 32.09 g/m² and mean EF was equal to 52.47 ± 8.88 percent. The correlations concerning echocardiographic variables and inflammatory markers are shown in table 5, where LVMI was positively correlated with hs-CRP, albumin, and ferritin. The EF was negatively correlated with IL-6 and hs CRP.

Results from linear regression analyses (Table 6) showed statistically significant univariate associations between fT3, tT3, tT4, and LVMI. In multiple linear regression analysis adjusted for albumin, hs-CRP, IL-6 and ferritin; only fT3level

Table 2. Relationship Between Thyroid Hormones and Nutritional State

	fT3		fT4		tT3		tT4		TSH	
	C	*P*	C	*P*	C	*P*	c	*P*	c	*P*
BMI	-0.10	> .05	-0.01	> .05	-0.15	> .05	-0.22	> .05	-0.00	> .05
Creatinin	-0.01	> .05	-0.10	> .05	-0.06	> .05	-0.07	> .05	0.03	> .05
Albumin	0.20	> .05	-0.21	> .05	0.27	< .05	0.07	> .05	0.10	> .05
Hemoglobin	0.25	< .05	-0.04	> .05	0.30	< .05	0.10	> .05	0.08	> .05
Phosphate	-0.19	> .05	0.03	> .05	-0.15	> .05	-0.09	> .05	-0.15	> .05
nPCR	0.09	> .05	0.21	> .05	0.17	> .05	0.19	> .05	-0.50	> .05
cholestrol	0.00	> .05	-0.02	> .05	0.06	> .05	0.19	> .05	-0.01	> .05
SGA	-0.08	> .05	-0.19	> .05	-0.82	> .05	-0.09	> .05	0.00	> .05

Table 3. The Relationship Between Thyroid Hormones and Peritoneal Properties

	fT3		fT4		tT3		tT4		TSH	
	c	*P*	c	*P*	c	*P*	c	*P*	c	*P*
KT/V	0.26	< .05	-0.10	> .05	0.23	> .05	0.17	> .05	0.17	> .05
rGFR	0.39	< .05	0.09	> .05	0.37	< .05	0.17	> .05	-0.05	> .05
PET	0.17	> .05	-0.05	> .05	0.14	> .05	-0.09	> .05	0.05	> .05

Table 4. The Relationship Between Thyroid Hormones and Inflammatory Variables

	Ferritin		Albumin		hs-CRP		IL-6	
	c	*P*	c	*P*	c	*P*	c	*P*
fT3	-0.06	> .05	0.20	> .05	-0.01	> .05	-0.05	> .05
fT4	-0.01	> .05	-0.21	> .05	-0.02	> .05	-0.08	> .05
tT3	-0.08	> .05	0.27	< .05	-0.07	> .05	-0.04	> .05
tT4	-0.04	> .05	0.07	> .05	-0.16	> .05	-0.04	> .05
TSH	0.13	> .05	0.10	> .05	0.01	> .05	-0.10	> .05

Table 5. The Correlations of Echocardiographic Variables and Inflammatory Markers

	Ferritin		Albumin		hs-CRP		IL-6	
	c	*P*	c	*P*	c	*P*	c	*P*
EF	0.63	< .05	0.94	< .05	-0.44	< .05	-0.26	< .05
LVEDD	0.68	< .05	0.94	< .05	0.10	> .05	0.16	> .05
LVSD	0.66	< .05	0.92	< .05	0.19	> .05	0.16	> .05
PWD	0.69	< .05	0.93	< .05	-0.01	> .05	0.02	> .05
LVMI	0.66	< .05	0.88	< .05	0.25	< .05	0.10	> .05
MPI	0.65	< .05	0.92	< .05	0.12	> .05	0.19	> .05

Table 6. The Relationship Between Thyroid Hormones and Echocardiographic Parameters

	EF		LVEDD		LVSD		PWD		LVMI		MPI	
	c	*P*	c	*P*	c	*P*	c	*P*	c	*P*	c	*P*
fT3	0.02	> .05	-0.10	> .05	-0.06	> .05	-0.23	< .05	-0.29	< .05	0.02	> .05
fT4	-0.16	> .05	0.02	> .05	0.08	> .05	-0.22	> .05	-0.01	> .05	-0.33	> .05
tT3	-0.04	> .05	-0.05	> .05	0.01	> .05	-0.20	> .05	-0.30	< .05	0.03	> .05
tT4	-0.13	> .05	0.00	> .05	0.16	> .05	-0.14	> .05	-0.25	< .05	-0.05	> .05
TSH	-0.04	> .05	-0.04	> .05	0.02	> .05	0.27	< .05	0.14	> .05	0.32	> .05

($P < .05$) had a meaningful negative correlation with LVMI; however, the association of tT4 and tT3 with LVMI was confounded by these inflammatory markers.

There was a significant univariate association between TSH and PWD ($P < .05$) which remained significant after the necessary adjustments for inflammatory markers. We found a significant negative correlation between LVMI and rGFR (c = -0.05, $P < .05$). A meaningful positive correlation was found between KT/V and LVMI (c = 0.29, $P < .05$) but no correlation was found between LVMI and peritoneal properties (based on PET test). Patient characteristics are reported across two fT3 strata based on its median value and compared appropriately (Table 7).

As shown in table 7, subjects whose serum fT3 levels were lower than the median value had lower hemoglobin and lower residual renal function (rGFR). Moreover, PWD and LVMI were significantly higher in the group with lower fT3 levels. Levels of IL-6, hs CRP, albumin, ferritin,

Table 7. Patients Characteristics According to Median Values of Free Triiodothyronine (fT3)

Characteristics	Low fT3 Group ≤ 2.1 pg/mL (n = 37)	High fT3 Group > 2.1 pg/mL (n = 34)	*P*
Sex			
Men	12	21	< .05
Women	25	13	
Age, y	56.3 ± 12.8	43.3 ± 16.7	< .05
Diabetes Mellitus	22	14	> .05
BMI, kg/m^2	23.4 ± 4.26	22.15 ± 4.09	> .05
Hypertension	29	21	> .05
PD Vintage, mo	30.81 ± 23.47	35.2 ± 18.9	> .05
SGA Score	15.25 ± 4.71		> .05
Alb, g/dL	3.63 ± 0.53	3.75 ± 0.49	> .05
Residual eGFR, mL/min	1.52 ± 1.74	3.57 ± 1.78	< .05
Kt/v	1.74 ± 0.36	3.13 ± 8.4	> .05
nPCR, g/Kg/d	0.77 ± 0.24	0.78 ± 0.81	> .05
IL-6, ng/L	40.69 ± 17.66	40.51 ± 16.93	> .05
Hs-CRP, µg/mL	3.83 ± 0.66	3.86 ± 3.64	> .05
Ferritin, ng/mL	369.5 ± 228.9	398.51 ± 252.9	> .05
PTH, pg/mL	225.4 ± 162.88	157.5 ± 200	> .05
Ca, mg/dL	8.76 ± 0.84	8.50 ± 0.76	> .05
P, mg/dL	5.34 ± 1.46	5.02 ± 1.16	> .05
SI, µg/ dL	71.80 ± 30.09	75.9 ± 30.8	> .05
TIBC, µg/dL	310.8 ± 71.6	308.33 ± 72	> .05
Hb, g/dL	10.08 ± 1.45	10.7 ± 1.5	> .05
EF (%)	51.61 ± 9.27	51.81 ± 12.48	> .05
LVEDD, mm	50.52 ± 6.99	48.69 ± 6.31	> .05
LVESD, mm	35.30 ± 7.06	33.5 ± 7.32	> .05
PWD, mm	12.05 ± 1.64	10.88 ± 2.65	< .05
MPI	0.46 ± 0.15	0.42 ± 0.10	> .05
LVMI	122.42 ± 30.66	104.38 ± 31.37	< .05

and SGA score did not differ significantly between the two fT3 groups.

DISCUSSION

The main findings of this study can be summarized as follows:

1) LVMI was significantly higher in the group with lower fT3 levels.
2) The free T3 and tT3 levels were positively correlated with rGFR and KT/V.
3) LVMI was positively correlated with hs-CRP, albumin, and ferritin.
4) fT3 level had a meaning full negative correlation with LVMI after adjustment for inflammatory markers.

Abnormalities of thyroid hormones are the result of a variety of non-thyroidal diseases such as severe infections, liver disease, malnutrition, and psychiatric disorders. In mild-to-moderate disease cases, levels of free and total triiodothyronine decrease; while, in sever forms of illnesses; there is a decrease in the levels of thyroid-stimulating hormone (TSH) and free and total thyroxine.[19]

The abnormalities of thyroid hormones are usually observed in patients with renal dysfunction and can be explained by the high prevalence of malnutrition and inflammation in this population. Reduced serum levels of tT3 and fT3 have previously been reported in several studies in patients with CKD both in pre-dialysis or on HD and PD.

Cardiovascular events are among the most common causes of mortality in patients with renal dysfunction and the abnormalities in thyroid hormones can be partially responsible for this outcome. Heart is especially vulnerable to adverse effects of low T3 level since cardiomyocytes are not able to generate T3 from T4 locally. Furthermore, T3 is an important regulator of cardiac-specific genes encoding various structural and functional proteins and considering the importance of T3 action on myocyte ion channels function and cardiac cytoskeleton, this hormone is vital for maintaining of appropriate cardiac electrophysiological properties. Consequently, low T3 levels may potentially lead to a wide range of adverse cardiovascular sequels including systolic and diastolic dysfunction, increased systemic vascular resistance and arterial stiffness and ventricular arrhythmias.

Kang and colleagues have reported a lower ejection fraction in PD and HD patients with reduced T3 levels.[13,23] Sanchez and his colleagues found a relation between decreased T3 and diastolic dysfunction.[24]

Zoccali reported a direct association between low fT3 and all-cause mortality in stable hemodialysis (HD) patients[13] and similar findings were observed in peritoneal dialysis patients.[25]

Other studies suggest that low T3 is associated with decreased systolic function, endothelial dysfunction, atherosclerosis, vascular calcification, and altered ventricular conduction in patients on dialysis.[3,11,13,15] Moreover, some but not all studies have shown that low T3 levels are associated with higher all-cause and cardiovascular mortality in this population.[3,26]

Hence previous studies have pointed to the inverse relationship between low T3 levels and inflammatory factors[21,22] the causal relationship between thyroid hormones and all-cause or cardiovascular mortality is difficult to be established since this relationship makes it difficult to weigh up the individual effects of each factor on cardiovascular outcome, but some additive effects is illustrated.[26,29]

Christiaan and colleagues showed that both basal levels and trimestral variation of T3 and T4 are associated with increased mortality particularly due to CV causes and adjustment for inflammation (IL-6) and malnutrition did not affect on this association.[27]

The study of Takamura reported the consistent results on carotid intima–media thickness and low T4 and fT4.[29]

Chang found that a higher T3 level was associated with a lower risk of all-cause death and combined cardiovascular death independent of comorbidities and markers of nutrition and inflammation.[30] However, TSH and free T4 levels were not associated with all-cause or cardiovascular death. So there is no consensus between the special form of thyroid hormone abnormality and adverse cardiovascular outcomes.

Our study found a positive correlation between LVMI and measured inflammatory markers (albumin, ferritin, and hs-CRP); however, the association of LVMI and fT3 was not confounded after adjusting for these factors. This finding is parallel with Christiaan et al.[34] which showed the inverse association between serum fT3 levels and CAC scores.

Our study found a significant positive correlation between Hb and fT3 and tT3, as well as, between albumin and tT3. This finding is similar to Jingxian and colleagues which showed that serum T3 is positively related to albumin and Hb.[35]

Evaluation of the nutritional state of included patients was done by SGA score, nPCR, BMI, albumin, creatinine, and cholesterol. We found no association between these factors and thyroid hormones except for tT3 and albumin. This data is in agreement with Venice's study that reported an inverse correlation between serum T3 levels and MIS, and fT3 and albumin.[37] this finding can be explained by considering the bioactivity of T3 and the adaptive decline in T3 production in case of severe illnesses.

For the first time, we evaluated the relationship between the peritoneal properties (based on PET test) and thyroid hormone levels, where no significant correlation was found. This association was evaluated because a fraction of daily produced thyroid hormones and thyroxine binding globulin is lost in the dialysate of peritoneal dialysis patients and the properties of peritoneal membrane may have effects in the amount of loss; but we didn't find significant difference. The study of Robey and colleagues showed that there are no difference in thyroid hormones levels of HD and PD, nor with the urine volume, indicating that it had little effect.[20] So, the decline in thyroid hormone levels can not be explained by this phenomenon.

In addition, considering the importance of residual renal function as a potent predictor of patient's survival in peritoneal dialysis patients, we evaluated the correlation between rGFRs of study population and thyroid hormone levels and we found an independent positive correlation between rGFR and serum fT3 level. This finding is consistent with the results of Chang et.al which presented an association between higher T3 level and higher residual kidney function.[30] In another study, Wang et.al. observed an important relationship between the degree of RRF and the severity of LVH in PD patients.[31] Moreover, loss of RRF is also associated with more severe anemia, greater degree of hypoalbuminemia, and higher arterial pressure; all of which are important risk factors for cardiac hypertrophy in patients undergoing dialysis.[32,33]

This finding suggests that the role of thyroid hormones on survival can be partially explained through the maintenance of residual renal function or the higher residual kidney function which contributes to higher fT3 level and, in turn, better cardiovascular survival. Longitudinal studies are warranted to determine how the correlation between low T3 and other thyroid hormones derangements affect residual renal function in predialysis CKDs or newly incident dialysis patients.

The limitations of our study are the following: (1) The relatively low number of patients; future studies enrolling higher number of patients may provide better insights into the relation between thyroid hormones and cardiac structural and functional properties, (2) Given the cross-sectional nature of our study, thyroid hormone levels were measured at a single point in time and changes in thyroid hormone status over time were not taken into account.

CONCLUSION

This study found an independent relation between LVMI and an fT3 level that remains significant even after the application of necessary adjustments for nutritional and inflammatory markers. This finding can support the results of previous studies indicating the adverse cardiovascular outcome of non thyroidal illness in patients with renal failure and can open a new perspective for investigating the indications of thyroid hormones replacement therapy which is uncertain up to now.

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