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Evaluation of left atrial mechanical functions and atrial conduction abnormalities in patients with clinical hypothyroid

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Abstract

Background: *The aim of this study was to investigate left atrial (LA) mechanical functions, atrial electromechanical delay and P wave dispersion in hypothyroid patients.*

Methods: *Thirty-four patients with overt hypothyroid and thirty controls were included. A diagnosis of overt hypothyroid was reached with increased serum TSH and decreased free T4 (fT4) levels. LA volumes were measured using the biplane area length method and LA active and passive emptying volumes and fraction were calculated. Intra- and interatrial electromechanical delay (EMD) were measured by tissue Doppler imaging (TDI). P wave dispersion was calculated by 12 lead electrocardiograms.*

Results: *LA diameter were significantly higher in patients with overt hypothyroid ($p = 0.021$). LA passive emptying volume and LA passive emptying fraction were significantly decreased with hypothyroid patients ($p = 0.002$ and $p < 0.001$). LA active emptying volume and LA active emptying fraction were significantly increased with hypothyroid patients ($p < 0.001$ and $p < 0.001$). Intra- and interatrial EMD, were measured significantly higher in hypothyroid patients (30.6 ± 6.1 vs 18.0 ± 2.7, $p < 0.001$; and 10.6 ± 3.4 vs 6.9 ± 1.4, $p < 0.001$, respectively). P wave dispersion were significantly higher in hypothyroid patients (48.8 ± 6.2 vs 44.3 ± 7.2, $p = 0.022$). In stepwise regression analysis demonstrated that, interatrial EMD and LA active emptying fraction related with TSH and fT4.*

Conclusions: *This study showed that impaired LA mechanical and electromechanical function in hypothyroid patients. TSH and T4 were independent determinant of interatrial EMD and LA active emptying fraction.* (Cardiol J 2012; 19, 3: 287–294)

Key words: atrial functions, interatrial delay, thyroid hormones

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Introduction

Thyroid hormone receptors are rich in the myocardium, so the heart is very sensitive to the thyroid hormones [1]. There are many regulatory effects of thyroid hormones, such as cardiac protein transcription and gene expression; these are effective, especially cardiovascular endothelial and smooth muscle cells [2]. So that, important cardiovascular changes may occur in hypothyroid patients, such as impaired myocardial contractility, decreased cardiac output, and heart rate, increased systemic vascular resistance [3], cardiomyocyte atrophy, endothelial dysfunctions [4, 5], higher prevalence of atherosclerosis and development of heart failure [6, 7]. Therefore, thyroid hormone deficiency could result in significant changes in the cardiovascular system.

Currently, measurement of the left atrial (LA) size is the most commonly used method to estimate the amount of atrial remodeling. LA volumes and LA mechanical functions have recently been identified as a potential indicator of cardiac disease and arrhythmias [8–10]. In all other respects, the additional parameters are needed in the evaluation of atrial remodeling. Intra- and interatrial conduction delay and non-homogeneous propagation of sinus impulses are well known electrophysiological distinctiveness of the atria prone to fibrillation [11]. Contrary of LA size, atrial conduction times reflect the amount both electrical and structural remodeling of the atria. Recently, it has been shown that rheumatoid arthritis, paroxysmal atrial fibrillation and systemic lupus erythematosus may impair LA functions and atrial conduction times [11–13]. However, LA mechanical functions and atrial conduction abnormalities have not been investigated in hypothyroid.

The present study was investigated to LA mechanical functions and atrial conduction times in patients with overt hypothyroidism.

Methods

Study population

We studied 34 patients (25 female and 9 male with mean age of) who were newly or untreated, previously diagnosed patients with overt hypothyroidism. The study patients had normal sinus rhythm on electrocardiography (ECG). In addition, 30 healthy control subjects (19 female and 10 male with mean age of) were included. The study was designed as prospectively. A diagnosis of overt hypothyroid was reached with increased serum TSH and decreased free T4 (fT4) levels in fasting blood samples (normal values in our laboratory were; 0.4––4.0 mU/L for TSH and 0.9–1.9 ng/ml for fT4). The entire study population's demographic characteristics, biochemical parameters, lipid values and ECGs were obtained. Exclusion criteria were as follows: subclinical hypothyroidism or hyperthyroidism, acute coronary syndrome, prior myocardial infarction and coronary artery disease, congestive heart failure, left ventricular (LV) hypertrophy, prolonged QRS duration (≥ 120 ms), reduced LV ejection fraction (< 55%), chronic obstructive pulmonary disease, significant valvular heart disease, pacemaker implantation, atrial flutter or fibrillation, frequent ventricular pre-excitation and atrio-ventricular conduction abnormalities, hypertension (resting blood pressure ≥ 140/90 mm Hg), diabetes mellitus, medications known to alter cardiac conduction, peripheral vascular diseases, pulmonary or neurological disease, pericarditis, peripheral neuropathy, congenital heart disease, alcohol abuse, renal or hepatic disease and poor echocardiographic imaging. Approval for the study, was obtained by the local ethics committee and all subjects gave inform consent.

Electrocardiography

At study entry, all subjects underwent standard 12-lead ECG, acquired using the MAC 5500 electrocardiograph (GE Healthcare, Milan, Italy) at a paper speed of 50 mm/s and 20 mm/mV. All recordings were performed in the same quiet room through spontaneous breathing, the subsequent 20 min of adjustment in the supine position. P wave duration measurements were performed manually by two of the observers using calipers and magnifying lens for exact definition of the ECG deflection as define in a previous study [14]. The beginning of the P wave was defined as the point where the initial deflection of the P wave crossed the iso-electric line, and the end of the P wave was defined as the point where the final deflection of the P wave crossed the iso-electric line. The ECG recordings with measurable P waves in less than 10 leads were excluded from the analysis. The difference between P wave maximum and P wave minimum durations was defined as P wave dispersion [14, 15]. Intra-observer and inter-observer mean percent mistake (absolute difference between two observations divided by the mean and expressed in percent) for maximum and minimum P wave duration measurements were determined in 50 randomly selected study applicant (30 patients/20 controls) and were < 5% for P maximum and < 6% for P minimum.

Standard echocardiography

All patients were evaluated by transthoracic M mode, two dimensional (2D), pulsed-wave (PW), continuous wave (CW), color flow and tissue Doppler imaging (TDI). All examinations were performed with the GE-Vivid-7 system (GE Vingmed, Horten, Norway) with a 2–4 MHz transducer at a dept of 16 cm. During echocardiography, continuous single-lead ECG recording was obtained. All patients were imaged in the left lateral decubitus position. 2D and conventional Doppler examinations were obtained in the parasternal and apical views according to the guidelines of the American Society of Echocardiography [15]. LV diameters and wall thickness were measured by M-mode echocardiography. LV ejection fraction was calculated using the apical two-and four-chamber views by Simpson's method, according to American Society of Echocardiography guidelines [15]. The mitral valve inflow pattern (E-wave, A-wave, E-wave deceleration time (Dt), E/A ratio and isovolumic relaxation time [IVRT]) were measured using pulsed wave Doppler. LV mass index was calculated using the formula with the Deveraux equation [16]. LA volumes were obtained echocardiographically from the apical four-chamber views by the biplane area length method [17, 18]. LA maximum volume (Vmax) at the end-systolic phase (onset of the mitral opening), LA minimum volume (Vmin) at the end-diastolic phase (onset of the mitral closure) and LA volume before atrial systole (Vp) were measured at the beginning of atrial systole (onset of p wave on ECG) and calculated indexed to body surface area. The LA function parameters were calculated as follows:

- LA passive emptying volume = Vmax–Vp;
- LA passive emptying fraction = [(Vmax–Vp)/ /Vmax] × 100%;
- LA active emptying volume = Vp–Vmin;
- LA active emptying fraction = [(Vp–Vmin)/Vp] × 100% [14].

Tissue Doppler echocardiography

TDI was performed by transducer frequencies of 3.5 to 4.0 MHz, adjusting the spectral pulsed Doppler signal filters to acquire the Nyquist limit of 15 to 20 cm/s was reached and using the minimal optimal gain. Myocardial TDI velocities (peak systolic [Sm], early diastolic [Em] and late diastolic velocities [Am]) were measured via spectral pulsed Doppler as of the LV-free wall from the apical four chamber view [15]. The ultrasound beam was positioned as parallel as possible with the myocardial segment to acquire the optimal angle of imaging.

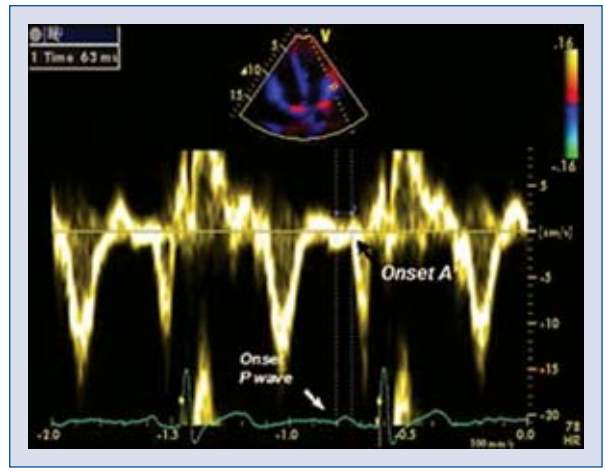

Figure 1. The time interval from the P wave onset on the surface ECG to the beginning of the late diastolic wave (Am), which is defined atrial electromechanical delay.

The time interval from the P wave onset on the surface ECG to the beginning of the late diastolic wave (Am), which is defined atrial electromechanical coupling (PA). It was obtained from lateral mitral annulus, septal mitral annulus, and right ventricular tricuspid annulus and named as PA lateral, PA septum, and PA tricuspid, respectively. The difference between PA lateral and PA tricuspid was defined as inter-atrial electromechanical delay (EMD), and the difference between PA lateral and septum was defined as intra-atrial EMD (Fig. 1) [14, 15]. All measurements were repeated three times, and average values were received for each of the atrial conduction delay times. All measurements were performed by two experienced investigators, who were unaware of the subject's clinical status. If a difference of > 5% in any of the variables measured by both investigators was found, the patient was not included, whereas if the difference was < 5%, the measurements were averaged.

Statistical analysis

All analyses were performed using the SPSS (SPSS for Windows 15.0) software package. Continuous variables were presented as mean ± standard deviation. Categorical variables were presented as the percentage. Fisher exact test and continuity correction were used for categorical variables and unpaired t-test was used for continuous variables if appropriate. Pearson's and Spearmen correlation exponents were used to force of relationship between continuous variables. A stepwise multiple regression analysis was used to recognize the

Table 1. Patients demographics, laboratory characteristics, echocardiographic findings and medication.

	Hypothyroid group (n = 34)	Control group (n = 30)	P
Age	44.2 ± 9.3	43.4 ± 6.0	0.377
Gender (female, %)	24 (70.4%)	20 (66.6%)	0.740
Smoking	5 (14.7%)	7 (23.3%)	0.386
BMI [kg/m^2]	28.3 ± 3.1	28.3 ± 2.1	0.956
BSA	1.85 ± 0.2	1.86 ± 0.2	0.699
Heart rate [beats/min]	74.2 ± 8.7	74.8 ± 6.8	0.840
Systolic blood pressure [mm Hg]	118.8 ± 8.3	119.3 ± 9.5	0.396
Diastolic blood pressure [mm Hg]	77.5 ± 7.0	78.7 ± 8.0	0.304
Total cholesterol [mg/dL]	194.8 ± 16.7	185.6 ± 17.6	0.037
LDL [mg/dL]	122.7 ± 15.9	111.5 ± 17.7	0.011
HDL [mg/dL]	46.3 ± 7.1	43.9 ± 7.7	0.016
Triglyceride [mg/dL]	151 ± 28.2	154 ± 29	0.364
Glucose	94.6 ± 7.1	97.9 ± 12.2	0.217
Hemoglobin [g/dL]	13.3 ± 1.2	13.7 ± 1.2	0.732
Creatinine [mg/dL]	0.79 ± 0.18	0.81 ± 0.15	0.312
TSH	17.4 ± 14.9	2.0 ± 0.5	< 0.001
fT3	2.7 ± 0.6	3.0 ± 0.8	0.097
fT4	0.67 ± 0.12	1.39 ± 0.17	< 0.001
LVEDD [mm]	49.1 ± 3.4	48.2 ± 2.6	0.535
LVESD [mm]	29.9 ± 3.6	30.2 ± 2.8	0.815
Ejection fraction [%]	64.0 ± 6.9	64.1 ± 2.3	0.879
Mass index [g/m^2]	101.5 ± 21.8	100.9 ± 19.9	0.542
Septum thickness [mm]	10.1 ± 1.1	9.8 ± 0.8	0.207
Posterior wall thickness [mm]	9.8 ± 0.8	9.5 ± 1.5	0.189
LV E/A	0.89 ± 0.29	1.03 ± 0.26	0.059
LA diameter [mm]	33.4 ± 2.1	32.2 ± 2.5	0.037

BMI — body mass index; BSA — body surface area; LDL — low density lipoprotein; HDL — high density lipoprotein; TSH — thyroid stimulant hormone; fT3 — free T3, fT4 — free T4; LVEDD — left ventricular end diastolic diameter; LVESD — left ventricular end systolic diameter; LA — left atrium

significant determinants intra- and interatrial EMD, which incorporated variables that correlated with a p value of less than 0.1 in the correlation analysis. A value of $p < 0.05$ was considered statistically significant.

Results

Patients characteristics. The clinical, laboratory characteristic and echocardiographic findings of the two groups shown in Table 1. Age, sex, body mass index (BMI), body surface area (BSA), smoking, heart rate, systolic and diastolic blood pressure, LV end diastolic diameter, LV end systolic diameter, LV mass index and LV ejection fraction were similar between two groups ($p > 0.05$). Total cholesterol, low density lipoprotein, high density lipoprotein and LA diameter were significantly higher in patients with overt hypothyroid than the normal group ($p = 0.037$, $p = 0.011$, $p = 0.016$, $p = 0.021$; respectively). LV E/A ratio was lower in overt hypothyroid patients but it did not reach levels of significance ($p = 0.059$). Additionally, LV E/A ratio < 1.0 was found 22 (64%) patients in overt hypothyroid group and LV E/A ratio < 1.0 was found 9 (30%) patients in control group. Both groups were similar in terms of the laboratory.

LA mechanical functions. LA volume measurements and mechanical functions were presented in Table 2. Both groups were similar in terms of Vmax, Vmin and Vp ($p = 0.828$, $p = 0.260$ and $p = = 0.118$, respectively). However, LA passive emptying volume and LA passive emptying fraction were significantly decreased with hypothyroid patients ($p = 0.002$ and $p < 0.001$). Morever, LA active emptying volume and LA active emptying fraction were significantly increased with hypothyroid patients ($p < 0.001$ and $p < 0.001$, respectively). There were the positive correlation between LA active emptying volume and LA active emptying

Table 2. Measurements of left atrial mechanical functions.

	Hypothyroid group (n = 34)	Control group (n = 30)	P
Vmax [mL/m^2]	28.9 ± 7.8	29.1 ± 8.0	0.828
Vmin [mL/m^2]	9.9 ± 4.2	8.8 ± 3.2	0.260
Vp [mL/m^2]	17.4 ± 6.1	15.1 ± 5.5	0.118
LA passive emptying volume [mL/m^2]	10.5 ± 4.6	14.0 ± 3.7	0.002
LA passive emptying fraction [%]	36.2 ± 9.2	49.8 ± 8.9	< 0.001
LA active emptying volume [mL/m^2]	9.1 ± 2.7	6.2 ± 2.5	< 0.001
LA active emptying fraction [%]	50.3 ± 8.4	40.7 ± 5.7	< 0.001

Vmax — left atrium maximum volume; Vmin — left atrium minimum volume; Vp — left atrium volume before P wave; LA — left atrium

Table 3. P wave analyses and atrial conduction times and they are relationship with overt hypothyroidism.

	Hypothyroid group (n = 34)	Control group (n = 30)	P
PA lateral [ms]	68.9 ± 8.6	55.2 ± 2.9	< 0.001
PA septum [ms]	53.0 ± 6.8	44.0 ± 2.6	< 0.001
PA tricuspid [ms]	38.3 ± 5.1	37.1 ± 2.2	0.091
PA lateral–PA tricuspid [ms]*	30.6 ± 6.1	18.0 ± 2.7	< 0.001
PA septum–PA tricuspid [ms]**	10.6 ± 3.4	6.9 ± 1.4	< 0.001
Maximum P-wave duration [ms]	104.3 ± 10.8	101.1 ± 9.9	0.284
Minimum P-wave duration [ms]	55.6 ± 6.1	56.6 ± 6.7	0.586
P-wave dispersion [ms]	48.8 ± 6.2	44.3 ± 7.2	0.022

PA — the interval with tissue Doppler imaging from the onset of p wave on the surface electrocardiogram to beginning of the late diastolic wave (Am wave); *inter-atrial electromechanical delay; **intra-atrial electromechanical delay

fraction with TSH levels ($r = 0.41$, $p < 0.001$ and $r = 0.33$, $p = 0.001$, respectively). There were the negative correlation between the LA passive emptying volume and LA passive emptying fraction with TSH levels ($r = –0.365$, $p = 0.003$ and $r = –490$, $p < 0.001$, respectively).

Atrial conduction times. Atrial electromechanical time intervals and P wave analysis were presented in Table 3. The PA lateral and septal durations were significantly higher in patients with overt hypothyroidism than the control group but there was no difference in PA tricuspid duration between the two groups (68.9 ± 8.6 *vs* 55.2 ± 2.9, $p < 0.001$; 53.0 ± 6.8 *vs* 44.0 ± 2.6, $p < 0.001$ and 38.3 ± 5.1 *vs* 37.1 ± 2.2, $p = 0.091$, respectively). Intra- and interatrial EMD, were measured significantly higher in patients with overt hypothyroidism than control group (30.6 ± 6.1 *vs* 18.0 ± 2.7, $p < 0.001$; and 10.6 ± 3.4 *vs* 6.9 ± 1.4, $p < 0.001$, respectively). P wave dispersion (PWD) were significantly higher in patients with overt hypothyroidism than the control group (48.8 ± 6.2 *vs* 44.3 ± 7.2, $p = 0.022$). There was mildly and significantly correlation between PWD and TSH ($r = 0.319$, $p = 0.010$). Intra- and interatrial EMD, were positively correlated with TSH ($r = 0.745$, $p < 0.001$ and $r = 0.636$, $p < 0.001$, respectively; Fig. 2). There was positive correlation between TSH and LA active emptying fraction ($r = 573$, $p < 0.001$) and there was negative correlation between, TSH and LA passive emptying fraction ($r = –0.523$, $p < 0.001$; Fig. 2).

In stepwise linear regression analysis demonstrated that, interatrial EMD and LA active emptying fraction related with TSH and T4 levels. But there were no relation LA diameter, LV E/A ratio, LA active emptying volume, LA passive emptying volume, LA passive emptying fraction, intra-atrial EMD and P wave dispersion between thyroid hormones in stepwise linear regression analyses (Table 4).

Discussion

In the present study, we have been demonstrated that PWD, intra- and interatrial EMD significantly related with overt hypothyroidism. Furthermore, LA mechanical functions impaired in patients with clinical hypothyroid. Also, LA mechanical functions and atrial electromechanical functions were associated with TSH and T4 levels.

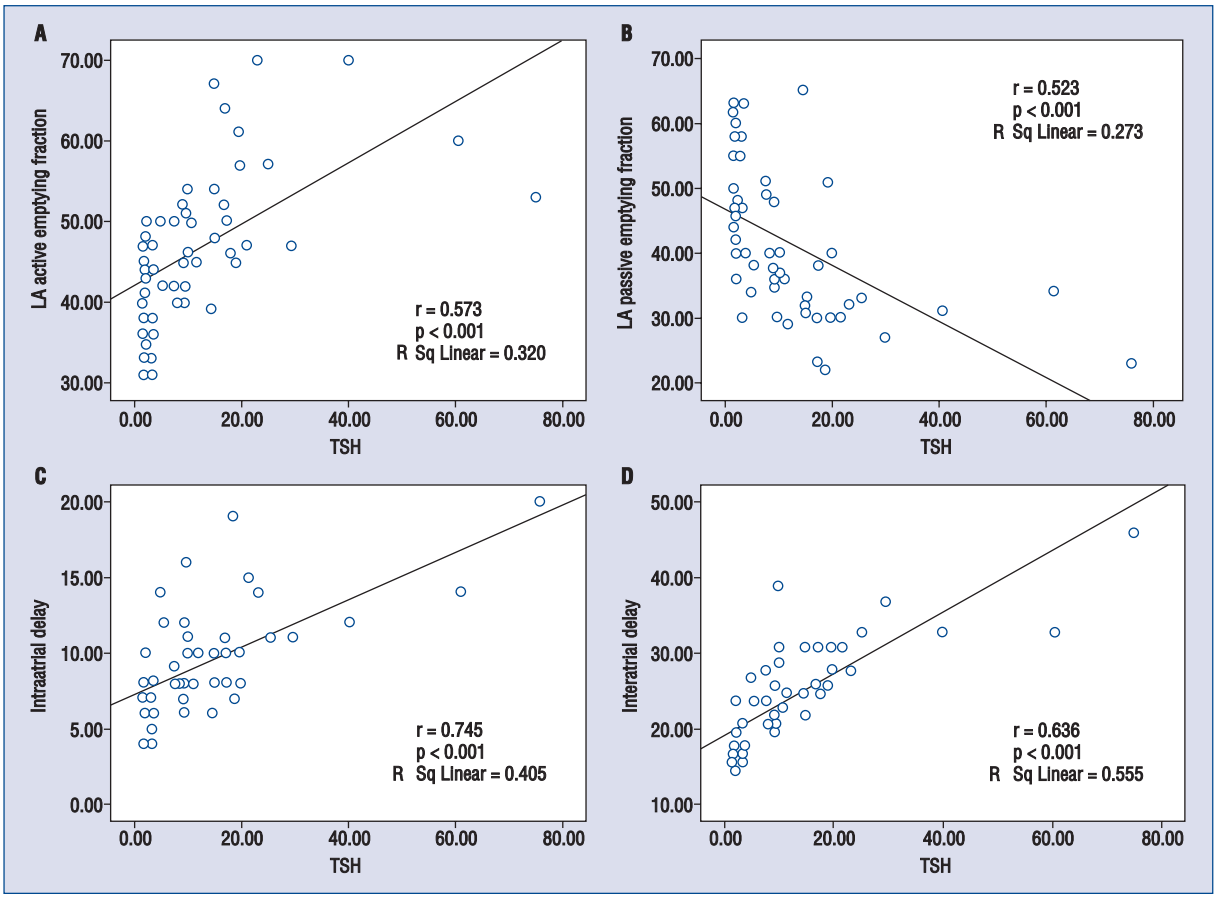

Figure 2. A. A positive correlation between left atrial (LA) active emptying fraction and thyroid stimulant hormone (TSH); **B.** A negative correlation between LA passive emptying fraction and TSH; **C.** A positive correlation between intraatrial delay and TSH; **D.** A positive correlation between interatrial delay and TSH.

The relationship between thyroid gland and the heart has been known for a long time. Previous studies demonstrated that effects of hypothyroidism on the ventricular function [19–22] The myocardium tissue is very sensitive to the thyroid hormones The myocardium is well-known among tissues that include thyroid hormone receptors so that myocardium is very sensitive to the thyroid hormones [21]. These effects can alter the activity of several enzymes occupied in the regulation of myocyte calcium fluxes [21] and the expression of several contractile proteins [22]. In addition, previous studies showed that thyroid hormone affect calcium uptake by the sarcoplasmic reticulum, to stimulate plasma membrane Ca-ATPase activity and to increase voltage-dependent channels in animal ventricular cells [21, 22]. On the other way, thyroid hormones are also effective transcriptions of structural and regulatory proteins on cardiovascular system [23]. Therefore, mildly changes in blood levels of thyroid hormones have many adverse effects on both function and structure of the heart. These effects are decreased cardiac contractility and cardiac output, cardiomyocyte atrophy, [1–4] myocardial fibrosis and development of heart failure [24–26].

Most of the previous studies focused on the relation between ventricular functions and thyroid hormones. The alterations in thyroid status may lead to changes, not only in ventricular function, but also atrial function. Some studies investigated the relation between different atrial parameters and hyperthyroidism. Guntekin et al. [27] found prolonged P wave duration and dispersion in patients with hyperthyroidism. Shenoy et al. [28] demonstrated the effect of sarcoplasmic reticulum calcium transporters by thyroid hormone in rat atria. Nevertheless, LA mechanical functions have not been evaluated in patients with overt hypothyroidism. Therefore, we investigated the atrial mechanical function and we found that LA mechanical functions were significantly impaired in patients with overt hypothyroidism. LA passive emptying volume

Table 4. In stepwise linear regression analyses relation between thyroid hormones and LA mechanical functions and atrial conduction times.

	β	t	p
TSH			
LA diameter	0.059	0.66	0.508
LV E/A	0.014	0.165	0.869
LA active emptying volume	0.192	1.77	0.094
LA passive emptying volume	0.022	0.22	0.821
LA active emptying fraction	0.212	2.09	0.041
LA passive emptying fraction	0.006	0.05	0.957
Intraatrial EMD	0.137	1.00	0.319
Interatrial EMD	0.622	6.14	< 0.001
P wave dispersion	0.027	0.29	0.729
fT4			
LA diameter	–0.040	–0.46	0.647
LV E/A	0.074	0.870	0.388
LA active emptying volume	–0.023	–0.20	0.840
LA passive emptying volume	–0.022	–0.23	0.813
LA active emptying fraction	–0.245	–2.54	0.014
LA passive emptying fraction	0.014	0.11	0.957
Intraatrial EMD	0.071	0.53	0.597
Interatrial EMD	–0.759	–9.14	< 0.001
P wave dispersion	0.027	0.29	0.729

LA — left atrium; LV — left ventricular; EMD — electromechanical delay; TSH — thyroid stimulant hormone; fT4 — free T4

and LA passive emptying fraction were significantly decreased also LA active emptying volume and LA active emptying fraction were significantly increased with hypothyroid patients in our study. Also we found that LV E/A ratio were mildly lower in overt hypothyroid patients than controls but this did not reach significance. Additionally, LV E/A ratio < 1.0 were more likely in clinical hypothyroid patients than controls. Therefore, impaired LA mechanical function could be the result of the increased end-diastolic LV pressure and/or associated strain on LA. In another study demonstrated that LV E wave to be a factor for atrial functions in patients with scleroderma [29]. LA mechanical functions contain reservoir, passive emptying and active emptying functions at different stages of cardiac cycle. The reservoir function takes effect during ventricular systole, passive emptying function in early diastole and active emptying function during ventricular diastole in the presence of sinus rhythm. When develops left ventricular dysfunction, the left atrium may possibly preserve adequate cardiac output by regulation of reservoir and booster pump functions. On the other hand, atrial functions intensely have an effect on heart function. This affects more especially in patients with reduced LV function [30, 31]. Hereby, impaired LA function may results development of heart failure in patients with overt hypothyroidism.

Thyroid hormone changes the speed of repolarization and the action potential duration of atrial and ventricular myocytes [32, 33]. Han et al. [34] demostrated that thyroid hormone administration shortens action potential duration and decreases the refractoriness of cardiomyocytes facilitating the maintenance of multiple reentrant circuits in rabbit hearts. Komiya et al. [35] showed the difference in the atrial effective refractory period and atrial conduction delay in patients with hyperthyroidism. Similarly, we found that prolonged intra- and interatrial EMD in patients with overt hypothyroidism in our study. As a consequence, prolonged intra- and interatrial EMD may be related with an increased risk for arrhythmias, in patients with overt hypothyroidism.

Limitations of the study

The major limitation of our study is the size of study population was relatively small. Patients could not be followed-up for arrhythmic episodes. Therefore, we do not know whether prolongation of intra- and interatrial EMD and impaired LA mechanical functions for prediction arrhythmias and heart failure in overt hypothyroid patients. For these reasons, long-term follow-up and large-scale prospective studies are needed to determine the predictive value of, prolonged intra- and interatrial EMD and LA mechanical functions in this population.

Conclusions

In conclusion, the current study is, firstly, reported that impaired LA mechanical and electromechanical function in patients with overt hypothyroidism. Secondly, impaired left atrial mechanical function and prolonged atrial electromechanical coupling times were related with TSH and T4 levels. Finally, interatrial EMD and LA active emptying fraction were increased in subjects with overt hypothyroidism, and TSH and T4 are independent determinant of interatrial EMD and LA active emptying fraction.

Conflict of interest: none declared

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