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ARAŞTIRMA / RESEARCH

Is there any relationship between frontal QRS-T angle and subclinical hypothyroidism?

Frontal QRS-T açısı ile subklinik hipotiroidizim arasında herhangi bir ilişki var mı?

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Abstract

Purpose: The aim of this study was to evaluate whether there is a relationship between frontal QRS-T angle on ECG and subclinical hypothyroidism (SCH).

Materials and Methods: A total of 115 individuals included in this study, 41 patients have SCH and 74 individuals as healthy control group. Serum thyroid-stimulating hormone (TSH) level > 4.5 mU/mL was determined for the diagnosis of SCH. All patients demographic, electrocardiographic and echocardiographic data were collected and compared between the two groups.

Results: The TSH level (5.7 [4.5-20] vs 1.2 [0.4-4.3] mlU/L), body mass index (BMI) (30.7 ± 6.4 vs 28.1 ± 5.3 kg/m2) and frontal QRS-T angle values (40 [2-188] vs 15 [1-86] degree) were higher in patients with SCH. However, left ventricular ejection fraction (LVEF) values were lower in subclinical hypothyroidism patients (58.9 ± 3.1 vs 60.8 ± 2.9). Correlations analysis showed the BMI (r=0.267, p=0.007) and frontal QRS-T angle were positively correlated (r=0.294, p=0.001), but LVEF were negatively correlated (r=-0.218, p=0.019) with TSH level. The multivariable linear regression analysis revealed that only age (OR:0.304 95% CI [0.242 – 1.147]) and), and subclinical hypothyroidism (OR:0.407 95% CI [15.175 – 42.494]) were a potential risk factors for increased of frontal QRS-T angle.

Conclusion: The SCH was found to be an independent risk factor for increased of frontal QRS-T angle.Frontal QRS-T angle can be considered as one of the ECG parameters that should be noted in cardiovascular risk estimation in patients with SCH.

Keywords: Subclinical hypotyroidism, electrocardiography, frontal QRS-T angle

Öz

Amaç: Bu çalışmada amacımız frontal QRS-T açısı ile subklinik hipotiroidizim (SKH) arasında bir ilişki olup olmadığını değerlendirmektir.

Gereç ve Yöntemler: Çalışmaya, 41 hasta SKH ve 74 sağlıklı kontrol grubu olmak üzere toplam 115 kişi dahil edildi. SKH tanısı için, serum tiroid uyarıcı hormon (TSH) düzeyi> 4,5 mU / mL olarak belirlendi. Tüm hastaların demografik, elektrokardiyografik ve ekokardiyografik verileri iki grup arasında karşılaştırıldı.

Bulgular: TSH seviyesi (5.7 [4.5-20]'e karşın 1.2 [0.4-4.3] mlU / L), vücut kitle indeksi (BMI) (30.7 ± 6.4'e karşın 28.1 ± 5.3 kg/m²) ve frontal QRS-T açı değerleri (40 [2-188]'e karşın 15 [1-86] derece)) SKH'li hastalarda daha yüksekti. Bununla birlikte, sol ventrikül ejeksiyon fraksiyonu (SVEF) değerleri, SKH hastalarında daha düşüktü (58.9 ± 3.1'e karşın 60.8 ± 2.9) Korelasyon analizlerinde, VKİ (r = 0.267, p = 0.007) ve frontal QRS-T açısı arasında pozitif korelasyon (r = 0,294, p = 0,001), ancak SVEF ile TSH düzeyi arasında negatif korelasyon (r = -0,218, p = 0,019) saptandı. Çok değişkenli doğrusal regresyon analizlerinde, sadece yaş (OO:0.304 95% GA [0.242 – 1.147) and) ve subklinik hipotroidizim (OO:0.407 95% GA [15.175 – 42.494) frontal QRS-T açısı artışı için olası bağımsız birer risk faktörü olarak bulundular.

Sonuç: Frontal QRS-T açısının artması, SKH hastalarda bağımsız bir risk faktörü olduğu bulundu.Frontal QRS-T açısı, SKH'li hastalarda, kardiyovasküler risk tahmininde, önemsenmesi gereken EKG parametrelerinden biri olarak düşünülebilir.

Anahtar kelimeler: Subklinik hipotroidizim, elektrokardiyografi, frontal QRS-T açısı

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INTRODUCTION

Subclinical hypothyroidism (SCH) is an endocrine disease that is thought to be associated with cardiovascular diseases, accelerates the atherosclerosis process with endothelial dysfunction, increases thyroid-stimulating hormone (TSH) hormone, but normal free T3 and T4 levels[1]. Its prevalence has been reported to be around 4 - 15% in the population. Iodine deficiency, age, gender and race play a role in determining the incidence of hypothyroidism. Patients with SCH usually do not have any symptoms or clinical signs and it is a likelihood to be detected incidentally. SCH is usually diagnosed with the presence of TSH (≥ 4.5 mU / mL) above the upper limit and normal FT 4 values. Generally, there is a consensus that patients with TSH ≥ 10 mU / mL should be treated[2,3].

Although there are conflicting results in meta-analysis related to SCH, researches demonstration that it is predominantly associated with cardiovascular disease and cardiovascular mortality[4]. In subclinical hypothyroidism, bradycardia, QT prolongation and heart failure may be developed[5]. Hypothyroidism contributes to increased serum cholesterol levels, development of atherosclerosis and increased susceptibility to cardiovascular diseases by impairing endothelial functions[5]. It has been shown that cardiovascular mortality increases significantly especially in those with TSH levels > 10 mU /L[6]. Hypothyroidism is also, reduce nitric oxide synthesis causes decreased relaxation of vascular smooth muscle cells, impaired relaxation functions of the heart, and reduced cardiac output[7].

The frontal QRS-T angle is one of the ECG parameters indicating increased cardiovascular mortality[8]. The frontal QRS angle is the value of the absolute difference between the ventricle's depolarization and repolarization vectors[8]. Increased frontal QRS-T angle has been associated with ventricular arrhythmia, sudden cardiac death, cardiovascular mortality and total mortality[8].

For the frist time, with this study, we aimed to define the relationship between the frontal QRS angle and cardiovascular diseases electrocardiographically in patients with subclinical hypothyroidism.

MATERIALS AND METHODS

Ethics committee approval (decision no: 2020/59, date: 16.12.2020) was obtained from the Non-Interventional Clinical Research Ethics Committee of Istanbul Sancaktepe Sehit Professor İlhan Varank Education and Research Hospital, before the initiation of the study. Written and verbal consents were obtained from all participants. Declaration of Helsinki's was followed in the application of the ethical rules of the study.

Sample

The patients included in the study were those who applied to Sancaktepe Şehit Prof. Dr. İlhan Varank Education and Research Hospital with complaints of fatigue, weakness and myalgia. Patient files included in anamnesis, blood tests, physical examination, echocardiographic data of all patients are recorded in the hospital computer system.

A total of 144 patients were included in the study. Among these patients, 25 patients with serum TSH levels of 20 mlU/mL and above and whose history of taking thyroid medication were excluded from the study. Four patients, who apparently receiving radiation to the thyroid region, were excluded from the study. Consequently, a total of 115 individuals included in the study, 74 were healthy volunteers and 41 patients who attend to our hospital and were found to have SCH. Patients with SCH are those who had a history of hypothyroidism documented in patient's medical record or those with a serum TSH levels (≥ 4.5 mU/mL) that is documented 2 times one month apart. The control euthyroid group, defined as serum TSH levels between (0.3 - 4.5 mU/mL) and/or no documented history of hypothyroidism in the medical records. The diagnosis of SCH was considered when TSH level ≥ 4.5 mU/ml according to the National Health and Nutrition Examination Survey (NHANES) study results[9].

All patients demographic data such as cardiovascular risk factors including age, sex, hypertension, diabetes mellitus, hyperlipidemia, smoking, and body mass index (BMI) were recorded. Blood samples collected from patients in the fasting state and routine clinical laboratory tests (complete blood count, thyroid function tests, glucose, creatinine, and lipid profile) were studied.

Patients with a TSH level < 0.3 mU/mL or a documented history of hyperthyroidism, who received treatment, those who have chronic renal or liver diseases, and those with cardiovascular diseases including arrhythmias, valvular heart disease,

coronary artery disease or heart failure were excluded from the study. SCH patients and control group were compared in terms of demographic data, electrocardiographic (ECG) parameters and echocardiography values.

Electrocardiography

After resting for at least 15 minutes all patients had a 12-lead ECG test in the supine position (GE Marquette Mac 1200). Each ECG was taken at a paper rate of 25 mm/s, a gain of 10 mV, and a paper format of 3x4. ECG's was interpreted by two different cardiologists independently. QRS duration was referred to as the time interval from the onset to the end of the QRS complex. QT interval was measured from the onset of the QRS complex to the end of the T-wave. The cQT interval was measured using Bazett's formula[10]. The frontal QRS-T angle between the QRS wave vector and the T wave vector is called the QRS-T angle and can be measured in two different methods, frontal and spatial[8]. While the spatial method requires complex computer measurements[8], frontal measurements can be calculated by simple methods on ECG. The frontal QRS-T angle measurement was taken as the absolute value of the difference between the QRS and T wave axes [Frontal QRS-T angle = (QRS axis-T axis)]. (Figure 1). If such a difference exceeds 180 degrees, then the frontal QRS-T angle is calculated as 360° minus the absolute value of the difference between the frontal plane QRS axis and T axis[8,10].

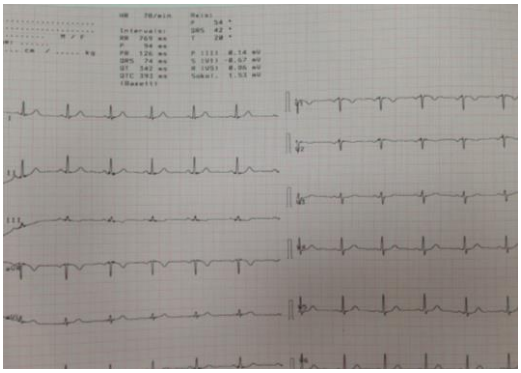

Figure 1. An example of the measurement of frontal QRS-T angle from the automatic report of 12-lead surface electrocardiography.

Echocardiography

Echocardiography was performed to all patients following the recommendations of the American Society of Echocardiography. All patients underwent a transthoracic echocardiographic examination with a commercially available device using 4 MHz probes (Vivid 9 Pro, GE Vingmed, Milwaukee, Wisconsin, USA) in the left lateral decubitus position[11]. All conventional measurements (LV end-diastolic and end-systolic diameters, LA end-systolic diameter etc.) were performed from the parasternal long-axis and apical four-chamber views. Left ventricular ejection fraction (LVEF) was calculated by Simpson's method [11].

Statistical analysis

The sample size was calculated in this study by taking into account the healthy population, and specific time period using the "simple random sampling method". Power analysis was calculated by using the post hoc analysis method and G power 3.1 package program. The study sample size was 115 patients, the margin of error was 0.05, the power of the study was 0.72 %, and the standard effect power was 0.51 %.

Statistical analysis was performed using SPSS 20.0 (IBM Corporation, Armonk, NY, USA) from the collected data, continuous variables are expressed as mean ± standard deviation (SD) or median interval interquartile range (IQR), and categorical variables are expressed as proportions of the group total %. Kolmogorov Smirnov test was used to determine whether the variables showed normal distribution. Continuous variables with normal distribution were evaluated using Student's t-test and continuous variables with abnormal distribution were analysed using Mann Whitney u test. Fisher's exact test or Chi-square test were used for categorical variables. Multivariable linear regression analyses were performed for the association between frontal QRS-T angle and age, BMI, heart rate, LVEF and subclinical hypothyroidism. Pearson's correlation coefficient (r) was used to evaluate the strength of the relation between frontal QRS-TA and other variables. For all statistics, a p-value below 0.05 was considered significant.

RESULTS

There was no difference between subclinical SCH and control group in terms of demographic and clinical features (Table 1). The TSH level (5.7 [4.5 - 20] vs 1.2 [0.4 - 4.3] mlU/L, p = 0.001), body mass index (BMI) (30.7 ± 6.4 vs 28.1 ± 5.3 kg/m2, p =

0.027) and frontal QRS-T angle values (40 [2 - 188] vs 15 [1 - 86] degree, p = 0.006) were higher in patients with SCH group (Table 1). The average frontal QRS-T angle was 49 ± 45.5 degree in patients with SCH and 22.0 ± 19.1 degree in the control group. However, LVEF values were lower in SCH patients (58.9 ± 3.1 vs 60.8 ± 2.9, p = 0.001) (Table 2), (Figure 2). Spearman's correlations analysis showed that BMI (r = 0.267, p = 0.007) and frontal QRS-T angle were positively correlated (r = 0.294, p = 0.001), but LVEF were negatively correlated (r = -0.218, p = 0.019) with TSH level (Table 3), (Figure 3).

Table 1. Demographic, clinical and electrocardiographic features of patients with subclinical hypothyroidism and control group.

Variables	Subclinical hypothyroidism group (n=44)	Control group (n=74)	p-value
Age, years	46.3 ± 13.0	44.4 ± 12.2	0.439
Female gender, n, (%)	32 (78.0)	49 (66.2)	0.183
Body mass index, kg/m²	30.7 ± 6.4	28.1 ± 5.3	0.027
Systolic blood pressure, mmHg	127.6 ± 24.2	125.8 ± 23.7	0.457
Diastolic blood pressure, mmHg	78.9 ± 13.6	77.8 ± 14.1	0.543
Hypertension, n (%)	13 (34.2)	16 (22.5)	0.189
Diabetes mellitus, n (%)	5 (12.2)	11 (15.5)	0.631
Current smoking, n (%)	10 (25.0)	19 (27.1)	0.806
Glucose, mg/dL	97.6 ± 11.3	100.5 ± 30.6	0.219
Creatinine, mg/dL	0.7 ± 0.2	0.7 ± 0.1	0.235
Total cholesterol, mg/dL	191.3 ± 36.9	193.6 ± 38.1	0.766
LDL-C, mg/dL	111.7 ± 29.3	115.5 ± 31.8	0.551
HDL-C, mg/dL	48.6 ± 14.2	49.7 ± 17.5	0.734
Triglyceride, mg/dL	159.4 ± 76.5	147.9 ± 94.7	0.534
Vitamin B12, pg/mL	267.7 ± 91.0	310.7 ± 98.3	0.675
Vitamin D, ng/mL	23.8 ± 11.2	19.2 ± 10.9	0.328
TSH, mlU/L	5.7 (4.5-10.0)	1.2 (0.4-4.3)	<0.001*
Free T 4, ng/dL	0.8 (0.6-1.4)	0.9 (0.7-1.5)	0.089
C-reactive protein, mg/dL	0.3 ± 0.6	0.2 ± 0.3	0.217
Albumin, mg/dL	4.2 ± 0.6	4.3 ± 0.4	0.725
Uric acid, mg/dL	5.8 ± 1.2	6.8 ± 3.8	0.550

ECG: Electrocardiography, HDL: High-density lipoprotein, LDL: Low-density lipoprotein cholesterol, n: number of patients, TSH, Thyroid-stimulating hormone; *: Mann-Whitney U test

Table 2. Electrocardiographic and echocardiographic features of patients with subclinical hypothyroidism and control group.

Variables	Subclinical hypothyroidism group (n=44)	Control group (n=74)	p-value
Echocardiographic parameters			
LVEF, %	58.9 ± 3.1	60.8 ± 2.9	0.001
SPAP, mmHg	24.0 ± 2.8	23.3 ± 2.3	0.162
ECG parameters			
Heart rate, bpm	77.4 ± 11.1	79.2 ± 12.6	0.215
PR interval, ms	144 (68-200)	142 (80-200)	0.555*
QRS duration, ms	85 (74-120)	84 (72-119)	0.779*
QT interval, ms	392 (380-484)	385 (370-472)	0.394*
cQT interval, ms	429 (358-487)	419 (363-491)	0.130*
Frontal QRS-T angle (⁰)	40 (2-188)	15 (1-86)	0.006*

Abbreviations: bpm: beats per minute, ECG: Electrocardiography, LVEF: Left ventricular ejection fraction, n: number of patients, SPAP: Systolic pulmonary artery pressure; *: Mann-Whitney U test

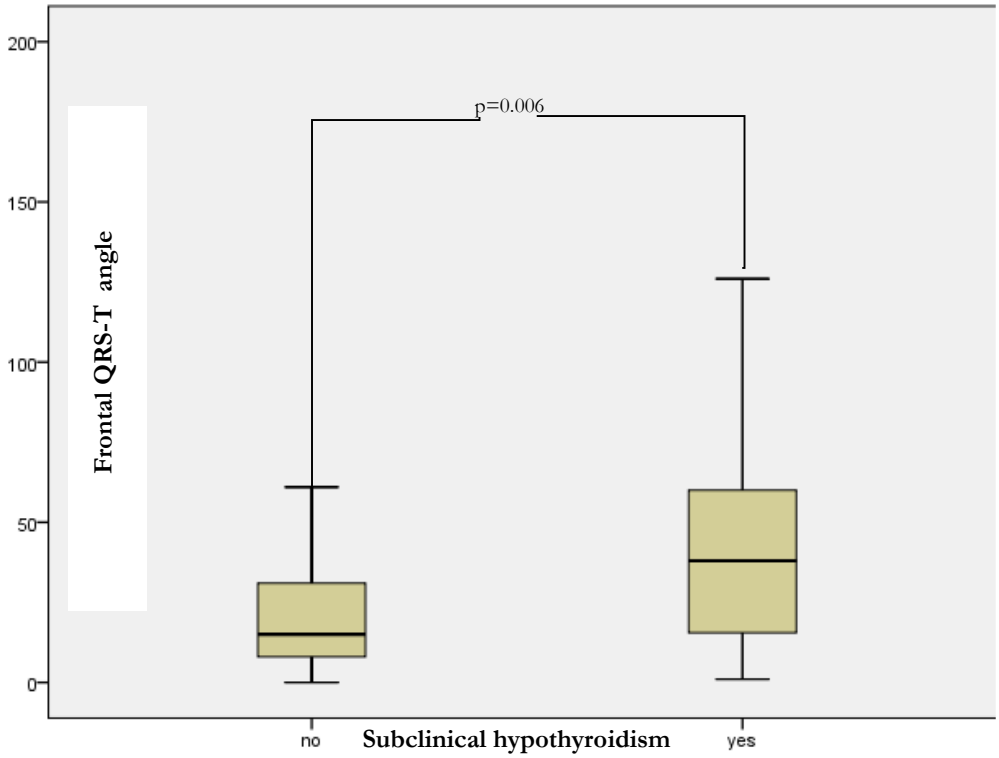

Figure 2. Demonstration of the frontal QRS-T angle between patients with subclinical hypothyroidism and patients without.

Table 3. Spearman's correlation analysis for the association between TSH and BMI, age, frontal QRS-T angle

Variables	Correlation Coefficient (r)	p-value
Age, years	0.050	0.599
BMI, kg/m²	0.267	0.007
Heart rate, bpm	-0.105	0.263
LVEF, %	-0.218	0.019
Frontal QRS-T angle (⁰)	0.294	0.001

Abbreviations: BMI: Body Mass Index, bpm: beats per minute, ECG: Electrocardiography, LVEF: Left ventricular ejection fraction, TSH: Thyroid-stimulating hormone

Table 4. The association between frontal QRS-T angle and subclinical hypothyroid patients, age, body mass index, heart rate with multivariable linear regression analysis

	Unstandardized Coefficients		Standardized Coefficients			
Variables	**B**	**SE**	**t**	**b**	**p**	**95 % CI**
Age (years)	0.844	0.304	2.781	0.304	**0.007**	0.242-1.147
BMI (kg/m²)	-0.658	0.590	-1.116	-0.122	0.267	-1.828-0.513
Heart rate (bpm)	0.340	0.274	0.119	0.119	0.217	-0.203-0.884
LVEF (%)	-0.308	1.174	-0.262	-0.262	0.794	-2.639-2.024
Subclinical hypothyrodism	**28.843**	**6.881**	**4.1**	**0.407**	**<0.001**	15.175-42.494

Abbreviations: BMI: Body mass index, bpm: beat per minutes, FQRST-A: Frontal QRS T-Angle, LVEF: Left ventricular ejection fraction

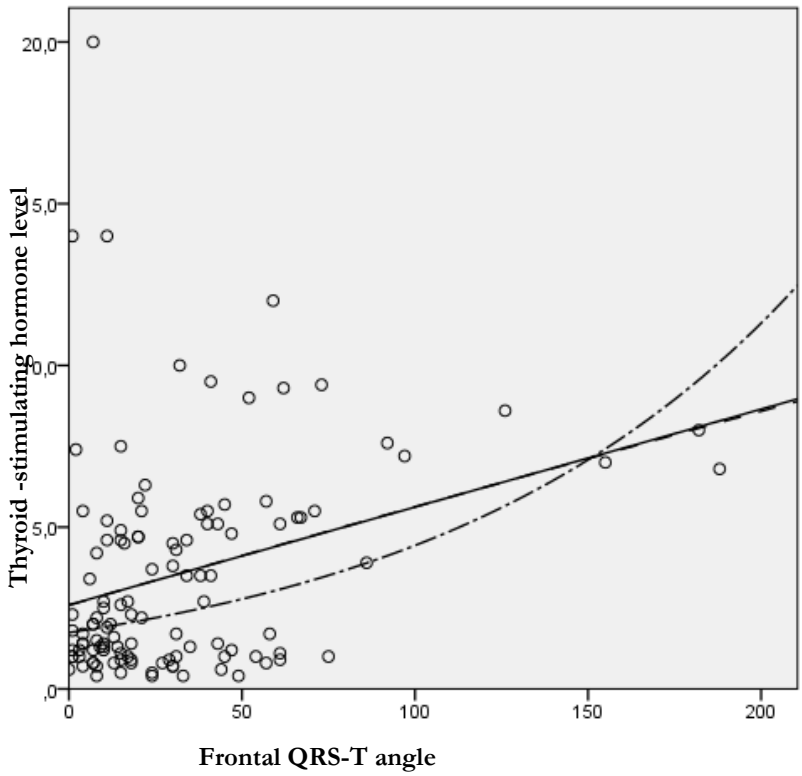

Figure 3. Diagram showing the positive correlation between the frontal QRS-T angle and the Thyroid-stimulating hormone level (r=0.294, p=0.001).

Multivariable linear regression analyses showed age (OR:0.304 95% CI [0.242 – 1.147], p = 0.007) BMI (OR:-122 95% CI [-1.828 - 0.513], p = 0.267), heart rate (OR: 0.119 95% CI [-0.203 – 0.884], p = 0.794), LVEF (OR:-0.262 95% CI [-2.639 – 2.024], p = 0.794), and subclinical hypothyroidism (OR:0.407 95% CI [15.175 – 42.494], p < 0.001) were a potential risk factors for increased of frontal QRS-T angle (Table 4).

DISCUSSION

The important findings of our study were that the frontal QRS-T angle was found to be significantly increased in patients with SCH. Subclinical hypothyroidism was shown to be associated with endothelial dysfunction and therefore it was thought to increase cardiovascular mortality [4,7]. Some mechanisms can explain the relationship between endothelial dysfunction in SCH and cardiovascular diseases. In hypothyroidism, endothelial functions are affected by the Thyroid hormone-mediated receptor (THR) -α1 and THR- β. When (THR) -α1 activation reduces, also coronary blood flow decreases with nitric oxide production and coronary resistance increases in endothelial and vascular smooth muscle cells [12,13,14]. As a result reduction of both contraction of the myocardium and cardiac output can lead to heart failure. In our study, patients with subclinical hypothyroidism were found to have reduced left ventricular ejection fraction. In addition to these changes, decreases in arterial compliance and increases in systemic vascular resistance accelerate the atherosclerosis process, leading to increases in cardiovascular disease. Previous researchers found that microvascular endothelial functions were impaired in patients with SCH in whom acetylcholine was administered into epicardial coronary arteries[15].

The interaction between thyroid hormones and ion channels in cardiac myocytes are responsible for depolarization and repolarization, which are activated by the stimulation of thyroid hormones, that may cause prolongation of action potential and an

increase in myocardial sensitivity in hypothyroidism [14]. The prolongation of myocardial relaxation may also be contributed to these electrophysiologic variances. The frontal QRS-T angle is determined the angle between the depolarization and repolarization vectors of the heart, in other words, it is related to the contraction and relaxation functions. Alterations in the duration and direction of depolarization and repolarization of the myocardium in hypothyroidism may be reflected on electrocardiography as an increase in the frontal QRS-T angle. In other words, the impaired thyroid hormones effects may increase the frontal QRS-T angle by causing decreasing cardiac contractility both electrophysiologically and at the molecular level.

In addition, thyroid hormones are involved in the regulation of pacemaker-related genes in cardiomyocytes [14]. Therefore, bradycardia frequently appears in thyroid hormone deficiency. In our study, the heart rate of patients with SCH was slightly lower, but this difference was not found significant in statistical analysis.

The autonomic nervous system is a system that unconsciously carries out the functions of the glands, circulatory system and internal organs. Subclinical hypothyroidism may indicate impaired functions of the autonomic nervous system [15]. The interaction between the sympathetic and parasympathetic system leads to changes in heart rate, ventricular repolarization and depolarization time, and the heart axis [16]. Therefore, in subclinical hypothyroidism, with the disruption of the autonomic nervous system, the change in the electrical system of the heart may cause the frontal QRS-T angle to change [17].

Serum cholesterol levels especially serum LDL and apolipoprotein B levels also increase in hypothyroidism. Hepatic LDL receptors expression is reduced in hypothyroidism and cholesterol-α-monooxygenase activity decreases, which reducing LDL clearance [6]. So that, increased total cholesterol and LDL are expected findings of hypothyroidism. In our study, no significant difference was observed between the two groups in terms of cholesterol levels. Cohort studies related to SCH patients were not observed significant differences between the cholesterol levels and diastolic blood pressures [18]. Presumably, the fact that most of our patients have TSH levels below 10 Mu/ml which may explain the absence of severe metabolic effects of hypothyroidism.

Although the frontal QRS-T angle differs according to the results of studies conducted in normal healthy people, historically upper limits of normal values have been between 45^{o} and 60^{o}[19,20]. In our study, while the average frontal QRS-T angle was approximately 22^{o} in the normal healthy group it was nearly 49^{o} in patients with hypothyroidism. It was thought that most of our patients included in the study were young and did not have severe chronic diseases that might cause our results to be lower than that in the literature.

There are several limitations of this study. The number of patients is very limited, and a larger number of patients and long-term follow-up are required to reach a general interpretation. Unfortunately, the spatial QRS-T angle, which is more closely associated with cardiovascular mortality, could not be calculated as there was no computer program required for it. In the future, evaluation of the ECGs of hypothyroidic patients with Spatial QRS-T angle may give more correct results and increase the accuracy of our findings.

With this study, we tried to define the relationship between the frontal QRS angle and cardiovascular diseases electrocardiographically in patients with subclinical hypothyroidism. In our study the frontal QRS-T angle was found to be significantly increased in patients with SCH. Thus the frontal QRS-T angle may be a new ECG parameter that can explain the relationship between SCH and cardiovascular diseases. These findings may shed light on the literature, as there has been no such study before.

Yazar Katkıları: Çalışma konsepti/Tasarımı: SU; Veri toplama: AB; Veri analizi ve yorumlama: SU; Yazı taslağı: SU; İçeriğin eleştirel incelenmesi: BB; Son onay ve sorumluluk: SU, EA, BB, AB; Teknik ve malzeme desteği: EA-; Süpervizyon: BB; Fon sağlama (mevcut ise): yok.
Etik Onay: Bu çalışma için İstanbul Sağlık Bilmleri Üniversitesi Sancaktepe Şehit Prof.Dr. İLHAN VARAK Eğitim ve Araştırma Hastanesi Bilimsel Araştırmalar Etik Kurulundan 16.12.2020 tarih ve 2020/59 sayılı kararı ile etik onay alınmıştır.
Hakem Değerlendirmesi: Dış bağımsız.
Çıkar Çatışması: Yazarlar çıkar çatışması beyan etmemişlerdir.
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Author Contributions: Concept/Design : SU; Data acquisition: AB; Data analysis and interpretation: SU; Drafting manuscript: SU; Critical revision of manuscript: BB; Final approval and accountability: SU, EA, BB, AB; Technical or material support: EA; Supervision: BB; Securing funding (if available): n/a.
Ethical Approval: For this study, Istanbul University of Health Sciences Sancaktepe Martyr Prof.Dr. Ethical approval was obtained from the Scientific Research Ethics Committee of İLHAN VARAK Training and Research Hospital with the decision dated 16.12.2020 and numbered 2020/59..
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