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RESEARCH

IMPACT OF SUBCLINICAL HYPOTHYROIDISM ON FREQUENCY OF PREMATURE VENTRICULAR CONTRACTIONS IN ELDERLY PATIENTS WITHOUT STRUCTURAL HEART DISEASE

Abstract

Introduction: Premature ventricular contractions (PVCs) are common arrhythmias, most of them requiring no follow-up or treatment. Subclinical hypothyroidism (SH) is defined as normal levels of free triiodothyronine (FT3) and free thyroxine (FT4), with elevation of serum thyroid stimulating hormone (TSH) levels (>4.2 mIU/l). We aimed to demonstrate the association of SH with the frequency of PVCs in elderly patients without structural heart disease.

Materials and Method: We included 327 consecutive elderly patients who underwent 24-hour Holter monitoring for the frequency of PVCs. The patients were initially divided into two groups with respect to the presence of SH. Later, the patients were divided into three groups with respect to the frequency of PVCs, with groups 1, 2 and 3 representing PVCs (<1,000/day), (1,000–5,000/day) and (>10,000/day), respectively.

Results: A total of 28 patients had SH. Number of PVCs was significantly higher in patients with SH (13104.7 ± 7007 vs 9286.7 ± 7724, p = 0.012) than in those without SH. On comparing the groups based on the frequency of PVCs, TSH levels were significantly higher in group 3 patients (>10,000 PVCs/day) than those in the others (p < 0.001). The percentage of patients with SH was significantly higher in group 3 (p = 0.005). According to univariate and multivariate analysis, SH was found to be an independent predictor of PVCs.

Conclusion: We found that SH is independently associated with frequent PVCs. We concluded that serum TSH concentration has a role in the frequency of PVC, therefore, SH may lead to a predisposition to ventricular arrhythmias.

Keywords: Hypothyroidism; Ventricular premature complexes; Electrocardiography ambulatory

ARAŞTIRMA

YAPISAL KALP HASTALIĞI OLMAYAN YAŞLI HASTALARDA SUBKLİNİK HİPOTİROİDİZMİN ERKEN VENTRİKÜLER KONTRAKSİYON SIKLIĞI ÜZERİNE ETKİSİ

Öz

Giriş: Prematür ventriküler kontraksiyonlar (PVK) sık görülür ve yapısal kalp hastalığı yokluğunda çoğu takip ve tedavi gerektirmez. Subklinik hipotroidi (SH), tiroid stimülan hormon yüksekliği (TSH>4.2 mIU/l) mevcutken serbest triiyodotironin(fT3) ve serbest tiroksin(fT4) düzeylerinin normal olduğu ve hipotirodizmin aşikar klinik bulgularının olmadığı durum olarak tanımlanır. Biz bu çalışmada yapısal kalp hastalığı olmayanlarda SH varlığının sık PVK ile ilişkili olup olmadığını araştırmayı amaçladık

Gereç ve Yöntem: Bu çalışmaya 24 saatlik holter monitorizasyonu (HM) ile PVK saptanan 327 geriatrik hasta dahil edildi. Öncelikle hastalar SH varlığına göre 2 gruba, sonrasında da PVK sıklığına göre 3 gruba ayrıldı. PVK sıkılığına göre sırasıyla grup 1 (<1000 PVK/gün), grup 2 (1000-5000 PVK/gün) ve grup 3 (>10000 PVK/gün) olarak tanımlandı.

Bulgular: Toplam 327 geriatrik hastanın 28'inde SH mevcuttu. SH olan hastalarda olmayanlara kıyasla PVK sayısı anlamlı olarak yüksek bulundu (13104.7±7007 vs. 9286.7±7724, p=0.012). PVK sıklığı ile yapılan gruplandırmaya göre, grup 3'te (>10000 PVK/gün) TSH düzeyleri anlamlı olarak yüksek bulundu (p<0.001) ve grup 3'te SH olan hasta yüzdesi diğer gruplara göre anlamlı olarak yüksekti (p=0.005). Tek ve çoklu değişkenli analiz sonuçlarına göre de SH sık PVK açısından öngördürücü olarak saptandı.

Sonuç: Biz bu çalışmada SH'nin PVK sıklığı ile bağımsız bir şekilde ilişkili olduğunu belirledik. Aynı zamanda TSH düzeyinin PVK sıklığında rolü olduğu, dolayısıyla SH'nin ventriküler aritmiler için öngördürücü olabileceği sonucuna vardık.

Anahtar sözcükler: Subklinik hipotroidi; Prematür ventriküler kontraksiyon; Holter monitorizasyonu

INTRODUCTION

Premature ventricular contractions (PVCs) are common in the general population, and most of them are considered clinically insignificant in the absence of an underlying structural heart disease (1). Conversely, recent studies have shown that an increased risk of sudden cardiac death, myocardial infarction and all-cause mortality in patients with frequent PVCs, but with no structural heart disease (2). According to recent studies, it is now known that PVCs can cause impaired ventricular contractility, a larger left ventricle end diastolic diameter (LVEDD) and a larger left ventricle end systolic diameter (LVESD), known as the ventricular premature complex-induced cardiomyopathy. Additionally, PVCs without an underlying heart disease may be associated with ventricular tachycardia (VT), and an elimination of these PVCs with catheter ablation prevents further occurrence of VT (3).

Subclinical hypothyroidism (SH) is defined as normal free triiodothyronine (FT3) and free thyroxine (FT4) levels with an isolated elevation of serum thyroid stimulating hormone (TSH) levels, while the clinical findings of overt hypothyroidism are absent. The incidence of SH varies between 1% and 10% and increases with age (4). Hypothyroidism is associated with various cardiac pathologies, including impaired cardiac contractility, decreased cardiac output, increased systemic vascular resistance and cardiac electrical abnormalities (5). Electrocardiographic changes, including bradycardia, low voltage and varying degrees of heart block are commonly observed in hypothyroid patients. Hypothyroidism affects autonomic regulation of cardiovascular system and ventricular repolarisation. In some studies, QT prolongation and increased QT dispersion (QTd) have been shown to be directly related to TSH levels in overt hypothyroidism (6). These changes have been linked to the occurrence of malignant ventricular arrhythmias and sudden cardiac death (7). However, sustained or life-threatening ventricular ectopy is rarely seen in hypothyroid patients. Despite these findings in patients with hypothyroidism, there are very few studies that have evaluated the effects of SH on the cardiac electrophysiology (8,9). Although SH commonly occurs due to a thyroid hormone disorder, disagreement still exists about the necessity of treatment of this condition. Although some publications claim that patients with TSH > 10 mIU /l should be treated, there is no consensus on whether a treatment is necessary.

To the best of our knowledge, no previous study has analysed the impact of SH on PVCs. Therefore, in this study, we aimed to demonstrate whether the presence of SH is associated with the frequency of PVCs in geriatric patients without overt structural heart disease.

MATERIALS AND METHOD

Study Population

In the present study, we enrolled 327 consecutive elderly patients referred to our clinic for frequent PVCs, in excess of 1000/24 h on Holter monitoring (HM), and with no evidence of structural heart disease. Patients with a known cardiomyopathy, severe valvular heart disease, atrial fibrillation, serum electrolyte abnormalities, pacemaker or cardioverter defibrillator implantation were excluded from the study. The patients included in the study were divided into two groups based on the presence or absence of SH, and then, the same patients were divided into three groups with respect to the frequency of PVCs, as groups 1, 2 and 3 representing rare PVCs (<1,000 PVCs/day), moderate-frequency PVCs (1,000–5,000 PVCs/day) and frequent PVCs (>10,000 PVCs/day), respectively.

The baseline demographic and clinical characteristics were reviewed. The baseline laboratory findings including fasting plasma glucose, creatinine, potassium, haemoglobin (Hb), leukocytes, TSH, FT4, triglycerides (TG), low-density lipoprotein-cholesterol (LDL-C), high-density lipoprotein-cholesterol (HDL-C) and total cholesterol levels were noted from the laboratory

recordings obtained prior to HM. SH was diagnosed by an elevated TSH value (>4.2mU/L) and FT4 values within the normal range. The patients with SH were not under any thyroid hormone replacement therapy. A standard 12-lead surface electrocardiogram (ECG) (25 mm/s and 10 mm = 1 mV) in the supine position was performed on each patient.

Echocardiography

Echocardiographic assessment was performed using a VIVID 7 Dimension Cardiovascular Ultrasound System (Vingmed-General Electric, Horten, Norway), with a 3.5 MHz transducer. Echocardiographic examination was performed in the left lateral decubitus position. Parasternal long- and short-axis views and apical views were used as the standard imaging windows. Ejection fraction (EF) was calculated by using modified Simpson's rule. All echocardiographic examinations were performed by an experienced cardiologist.

Holter monitoring and interpretation

Holter devices (Universal resting 12-lead Holter DMS 300-4A, MTM multitechmed gmbh, Schwarzwaldstrasse, Germany) were applied to the patients by our clinic's nurse; the patient came back after 24 hours and the nurse removed the device and uploaded the recordings to the Holter archive. Two cardiologists independently evaluated the recordings for PVCs, and the number of PVCs was recorded.

The study was approved by the Ethics Committee of Health Sciences University Ankara City Hospital.

Statistical analysis

Statistical analysis was performed using the SPSS 20.0 Statistical Package Program for Windows (SPSS, Inc., IL, USA). Continuous variables were presented as mean ± standard deviation and median with interquartile ranges, as appropriate, and the categorical variables as frequency and percentage. To test the normality of distribution, Kolmogorov–Smirnov test was used. A comparison between the two groups, according to the presence or absence of SH, was performed using the student's t-test. A comparison between the three groups, according to the number of PVCs, was performed using one-way ANOVA and Tukey's test for post-hoc analysis. Categorical variables were compared by χ^2 test. Multivariate logistic regression analysis, which included variables with $p < 0.1$, was performed to identify independent predictors of PVC frequency. The Pearson's correlation analysis was used to evaluate the relationship between the TSH levels and number of PVCs. A p-value < 0.05 was considered as statistically significant.

RESULTS

A total of 327 consecutive elderly patients who underwent 24-Hour HM for the frequency of PVCs were enrolled in our study. The clinical and baseline characteristics of the study population are shown in Table 1. The mean age of the study population was 73.7±3.8 years and 55.4% of the patients were females. SH was present in 28 patients of them. There were no differences between the two groups (defined according to the presence or absence of SH) with respect to age, gender, hypertension (HTN), diabetes mellitus (DM), medical treatments(ASA, β-blocker vs) and smoking status. Moreover, the baseline laboratory findings, except TSH and FT4, were not different between the two groups. Patients with SH had higher TSH levels (5.24 ± 1.2 vs. 2.26 ± 1.2, $p < 0.001$) than the patients without SH. ECG findings were similar regarding the baseline rhythm and heart rate with SH (88.6±13.0 vs. 84.4±15.3, p=0.160) and without SH ,respectively. None of the patients presented any sustained or non-sustained supra-ventricular tachyarrhythmias on 24-h ambulatory ECG monitoring. There were no differences between the 2 groups with respect to presence of premature atrial contractions with or without SH.(135.5±312 vs. 95.8± 206.3, p=0.356). Heart rate variability (HRV-SDNN24) on 24-h ambulatory ECG monitoring were similar between two groups (121.07±34.2 vs. 128.6± 30.1,

p=0.209).Number of PVCs was significantly higher in patients with SH (13104.7 ± 7007 vs. 9286.7 ± 7724, p = 0.012) than the patients without SH. None of the patients presented any sustained or non-sustained ventricular tachyarrhythmias on 24-h ambulatory ECG monitoring. Echocardiographic measurements, including EF, LVEDD, LVESD, IVSD,left atrial and systolic pulmonary artery pressure were similar between the two groups.

Table 2 demonstrates the characteristics of the study population with respect to the frequency of PVCs. There were no differences between the three groups with respect to gender, age, HTN, smoking status and family history of coronary artery disease. The EF was lower in group 3 than in group 1 and group 2 (p = 0.004). TSH levels were significantly higher in group 3 patients than those in group 2 and group 1 (3.10 ± 1.6 vs. 2.27 ± 1.2 and 2.07 ± 1.1, respectively, p < 0.001). The percentage of patients with SH was significantly different between all the three groups. While 14.8% of group 3 patients had SH, 7.1% and 2.8% (p = 0.005) of group 2 and group 1 patients, respectively, had SH.

According to univariate and multivariate analyses, SH (p = 0.003 / p = 0.005) was found to be predictive for frequent PVCs (Table 3).

In the correlation analysis, there was a moderate positive correlation between the TSH levels and number of PVCs in the patients (r = 0.482; p < 0.001) (Figure 1).

Table 1. Baseline characteristics of study patients according to the presence of subclinic hypothroidism (SH).

	SH – (n=299)	SH + (n=28)	P <0.001
Age, years	73.7±3.8	74.2±4.1	0.554
Gender, female n (%)	166(55.5)	15 (53.6)	0.843
Hypertension, n (%)	121(40.5)	9 (32.1)	0.389
Diabetes mellitus, n (%)	49(16.4)	4(14.3)	0.773
CAD, n (%)	31(10.4)	8(28.6)	0.005
Family CAD history, n (%)	10(3.4)	1(3.6)	0.957
Smoking, n (%)	28(9.5)	0(0)	0.089
Ejection fraction, %	58,7±5.2	59,3±4.6	0.522
LVEDD, mm	46.8±6.1	46.3±3.0	0.702
IVSD, mm	10.2±4.5	9.1±1.4	0.222
SPAP, mmhg	29.7±5.6	27.9±4.0	0.102
HR (bpm)	84.4±15.3	88.6±13.0	0.160

	SH – (n=299)	SH + (n=28)	P <0.001
PVCs	9286.7±7724	13104.7±7007	0.012
ASA, n (%)	69(23.1)	4(14.3)	0.285
β blocker, n (%)	209(69.9)	19(67.9)	0.822
ACEi, n (%)	62(20.7)	6(21.4)	0.931
ND-CCB, n (%)	71(23.7)	11(39.3)	0.70
Fpg, mg/dl	102.3±21.6	110.0±44.9	0.112
Creatinine, mg/dl	0.86±0.18	0.88±0.15	0.724
Hemoglobin, g/dl	14.1±5.3	13.7±1.1	0.741
Htc, %	42.7±3.6	42.4±3.6	0.705
Leukocyte, ×103 /ml	7.66±1.8	7.64±1.3	0.953
Platelet, $\times 10^3$ /ml	242±55	244±68	0.804
Ldl-c, mg/dl	112±32	110±20	0.674
Hdl-c, mg/dl	50±12.7	47±9.2	0.170
Triglyceride, mg/dl	158±69.4	177±90.2	0.171
Tsh, uı/ml	2.26±1.2	5.24±1.2	<0.001
fT4, UI/mL	1.16±0.4	1.00±0.1	0.037
Group 1, n (%)	103(34.7)	3(2.8)	0.010
Group 2, n (%)	92(30.8)	7(7.1)	0.525
Group 3, n (%)	104(34.8)	18(14.8)	0.002

Data are presented mean ± SD or n (%).

CAD- coronary artery disease; ASA - asetil salicylic acite; ND-CCB – nondihydropyridine calcium channel-blocking agent; FPG- fasting plasma glucose; HDL-C- high-density lipoprotein cholesterol; LAD- left atrium diameter; LDL-C- low-density lipoprotein cholesterol; LVEDD- left ventricle end-diastolic diameter; IVSD - interventricular septum end-diastolic

diameter;SPAP: systolic pulmonary artery pressure; PVC- premature ventricular contraction; fT4- free level of thyroxine; TSH- thyroid-stimulating hormone

Table 2. Patient characteristics according to PVC frequency.

	Group 1 (n=106) (PVC 1000 5000/day	Group 2 (n=99) (PVC 5000-10000 day)	Group 3 (n= 122) (PVC> 10000 day)	P
Age, years	74.2±4.1	73.2 ±3.4	73.8±3.9	0.151
Gender, female n (%)	73(40.3)	64(35.4)	44(24.3)	<0.001
Hypertension, n (%)	44(41.5)	36(36.4)	50(41.0)	0.709
Diabetes mellitus, n (%)	12(22.6)	4(7.5)	37(69.8)	<0.001
CAD, n (%)	5(12.8)	10(25.6)	24(61.5)	0.002
Family CAD history, n (%)	5(44.5)	0(0)	6(54.5)	0.081
Smoking, n (%)	6(21.4)	11(39.3)	11(39.3)	0.378
SH, n (%)	3(2.8)	7(7.1)	18(14.8)	0.005
Ejection fraction, %	59.6±5.4	59.2±5.5	57.5±4.3	0.004
LVEDD, mm	46.4±3.4	48.5±3.6	45.5±8.3	0.001
LAD, mm	36.8±4.7	38.5±3.3	36.6±3.0	< 0.001
SPAP, mmhg	28.8±5.8	29.9±6.4	29.9±4.4	0.298
FPG, mg/dL	97.9±19.5	98.3±13.8	111±31.9	<0.001
Creatinine, mg/dL	0.87±0.2	0.84±0.1	0.88±0.1	0.220
Hemoglobin, g/dL	14.3±8.8	13.9±1.2	13.9±1.5	0.772
Htc, %	41.8±3.2	42.8±3.3	43.3±3.9	0.009
Platelet, $\times 10^3$ /mL	244±52	237±51	237±63	0.580
Leukocyte, $\times 10^3$ /mL	7.6±2.0	7.6±1.5	7.6±1.7	0.951
LDL-C, mg/dL	113±31	104±31	117±29	0.008
HDL-C, mg/dL	52±15	44±9	52±10	<0.001
Triglyceride, mg/dL	136±66	162±61	177±78	<0.001
TSH, UI/mL	2.07±1.1	2.27±1.2	3.10±1.6	< 0.001
fT4 ,mg/dl	1.07±0.29	1.18±0.28	1.19±0.54	0.075

Data are presented mean ± SD or n (%).

CAD- coronary artery disease; FPG- fasting plasma glucose; Htc-Hematocrit; HDL-C- high-density lipoprotein cholesterol; LAD- left atrium diameter; LDL-C- low-density lipoprotein cholesterol; LVEDD- left ventricle end-diastolic diameter; SH – Subclinical hypothyroidism SPAP- systolic pulmonary artery pressure; PVC- premature ventricular contraction; fT4- free level of thyroxine; TSH- thyroid-stimulating hormone

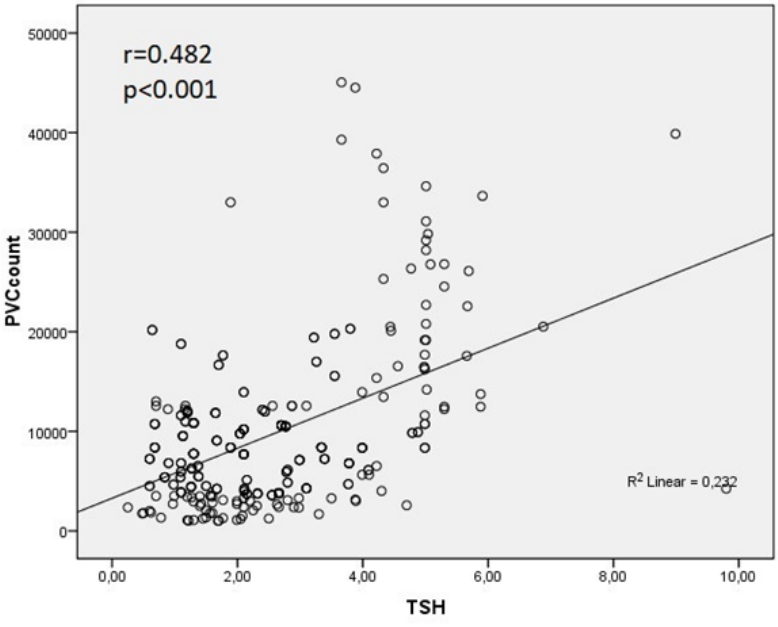
Table 3. Univariate and multivariate analyses for predictors of frequent premature ventricular contraction

Variable	Univariate analysis			Multivariate analysis		
	OR	95 % CI	p value	OR	95 % CI	p value
Age	1.001	0.945-1.061	0.962			
Hypertension	1.085	0.687-1.714	0.726			
Diabetes mellitus	5.142	2.712-9.750	<0.001	4.017	1.975-8.253	<0.001
Family CAD history	2.095	0.625-7.017	0.231			
Smoking	1.110	0.502-2.457	0.797			
CAD	3.086	1.548-6.150	0.001	2.613	1.227-5.510	0.013
Ejection fraction	0.930	0.888-0.973	0.002	0.960	0.912-1.010	0.117
LVEDD	0.943	0.899-0.988	0.014			
LAD	0.932	0.876-0.990	0.023			
FPG	1.026	1.014-1.037	<0.001			
Creatinine	1.790	0.515-6.217	0.359			
Hemoglobin	0.992	0.944-1.043	0.753			
Platelet	1.001	0.997-1.005	0.651			
Leukocyte	0.980	0.864-1.111	0.751			
LDL-C	1.009	1.001-1.016	0.022			
HDL-C	1.024	1.005-1.043	0.011			
Triglyceride	1.006	1.002-1.009	0.001	1.004	1.000-1.007	0.044
TSH	1.572	1.326-1.862	<0.001			
fT4	1.443	0.838-2.484	0.186			
SH	3.375	1.503-7.578	0.003	3.469	1.461-8.241	0.005

Bolded values indicate statistically significant odds ratio.

CI: confidence interval; OR: odds ratio; CAD- coronary artery disease; FPG- fasting plasma glucose; HDL-C- high-density lipoprotein cholesterol; LAD- left atrium diameter; LDL-C- low-density lipoprotein cholesterol; LVEDD- left ventricle end-diastolic diameter; SH – Subclinical hypothyroidism; fT4- free level of thyroxine; TSH- thyroid-stimulating hormone

Figure 1. Correlation between PVC count and TSH.



DISCUSSION

Two major findings of our study were as follows: (1) SH was independently associated with frequent PVCs, and (2) there was a moderate positive correlation between the TSH levels and number of PVCs in the patients. To our knowledge, this is the first study defining the relationship between frequent ventricular PVCs and SH in patients with structurally normal hearts.

PVCs are frequently seen in healthy population with increasing age. In patients without structural heart disease, this is regarded as benign by many physicians. However, studies in the last 15 years have showed their potential pathogenicity (10). PVCs can lead to impaired ventricular contractility and dilation of the ventricular size, known as PVC-induced cardiomyopathy. Previous studies have suggested that there is a correlation between the PVC load and left ventricular function, and a higher PVC load is associated with a lower left ventricular EF, a larger LVEDD and a larger LVESD (11). Other studies have shown improvement in left ventricular systolic function after suppression of PVCs in patients with dilated cardiomyopathy (11-13). PVCs are commonly associated with various cardiac conditions like myocardial ischaemia and heart failure, but can also be seen in non-cardiac conditions such as pulmonary disease, consumption of alcohol, caffeine, ?-agonists, cocaine or amphetamines.

The effects of thyroid hormone deficiency on the heart are well known. An elevated TSH level can adversely affect the cardiovascular system (14). Previous studies have shown that overt hypothyroidism is characterised with the prolongation of QTd and increased sympathetic influence. Inukai et al. showed that hypothyroidism was associated with increased sympathetic activity of the heart (15). Cacciatori et al. suggested that thyroid hormone deficiency was associated with an increased sympathetic influence on the autonomic cardiovascular system. Some studies demonstrated that SH increased the cardiovascular risk factors,

including altered lipid profile, impaired cardiac contractility, decreased cardiac output, increased systemic vascular resistance and cardiac electrical abnormalities (16-18). Galetta et al. showed that the patients with SH had a higher QTd and lower heart rate variability (HRV) measurements compared with the healthy control group, and that the QTd was positively associated with TSH value, with the measurements returning to normal after treatment (9). In general, increased QTd has been shown to be associated with increased risk of malignant ventricular tachyarrhythmia and sudden cardiac death (7). Çelik et al. proved that HRV and heart rate (HR) turbulence was reduced in the patient group, and the cardiac autonomic functions did not improve effectively with levothyroxine treatment (7,19). In a previous study by Mahajan et al., it was also demonstrated that autonomic dysfunction may be seen in both subclinical and frank hypothyroidism (5).

The mechanism of autonomic dysfunction in hypothyroidism is thought to be because of increased adrenalin levels associated with a receptor or post-receptor sensitivity, decreased chronotropic response to beta-adrenergic stimulation and an increase in thyrotropin-releasing hormone, which has a direct effect on the sympathetic activity (20). The thyroid hormones can also have direct effect on the heart, which includes increased protein deposition in the extracellular space, leading to accumulation of water in the myocardial wall. The resulting myocardial oedema may lead to fibrosis and regional homogeneity disorder in ventricular repolarisation (20,21). Bakiner et al. showed that QT and the corrected QT (QTc) intervals were prolonged in patients with SH and that the return of serum TSH levels to the values within the normal range resulted in normalisation of QTc (8). A recent study showed that SH may change the autonomic modulation of HR and cause heterogeneity of ventricular healing times (9). These differences may contribute to the prevalence of increased ventricular arrhythmias, as a result of more heterogeneous ventricular repolarisation in patients with SH. It is widely accepted that autonomic control plays an important role in the genesis, maintenance and interruption of ventricular arrhythmia (VA) (22). In most cases, VAs are evoked or aggravated by sympathetic activation and/or decreased vagal tone, despite the heart being structurally normal or abnormal. In addition, a considerable part of idiopathic PVCs is sensitive to beta-blockers in clinical practice. Suggested physiological mechanisms for PVCs include enhanced automaticity, re-entry and triggered activity. Sympathetic activation can enhance the automaticity in ectopic focus and induce the cAMP-mediated triggered activity, which might explain its facilitating effects on idiopathic PVCs (23).

In this study, we showed that the SH was associated with frequent PVCs. This result, as mentioned in older studies, may be related to the effects of SH on cardiac autonomic activity (increased sympathetic activity, decreased vagal tonus) and the effects on electrocardiographic repolarisation parameters. Thus, we suggest that SH, with increased sympathetic activity in the background, may cause frequent PVCs, but further studies are required to determine the relation between the frequency of PVC in patients with SH. Our study also showed that patients with frequent PVCs had lower EF values. Similar to previous studies, we have found that PVCs that are commonly seen in patients with apparently normal hearts are associated with reduced EF compared with the patients without frequent PVCs. However, as opposed to previous studies, the LVEDD values were not found to be higher in patients with frequent PVCs. Although the TSH level that dictates a need for treatment in patients with SH is controversial, it has been reported that the repolarisation anomaly in patients with SH can be corrected by treatment with L-thyroxine, and this condition is particularly evident in patients with TSH > 10mIU/l (8,9,24). Since there is no study evaluating the relationship between SH and the frequency of PVCs, further researches are required to recommend this treatment to the patients.

This study should be interpreted within several limitations. First, this was a retrospective

observational study. Without any intervention and a prospective design, it was not possible to illustrate the direct effect of SH on the frequency of PVCs. The direct relationship between the PVCs, SH and clinical outcome should be evaluated in prospective large-scale studies, with a longer follow-up period. Secondly, the PVC classification was dependent on a single 24-h HM, which is liable to higher variability and lesser clear classification, in comparison with a longer monitoring time.

In summary, our study showed that SH was significantly and independently associated with frequent PVCs. Furthermore, we found that there is a positive correlation between the TSH levels and number of PVCs. Further studies are needed to find out the relation between the frequency of PVCs and the SH.

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None.

CONFLICT OF INTEREST

The author declares that they have no conflicts of interest to declare.

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