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Evaluation of electrocardiogram findings and inflammatory parameters in patients with benign paroxysmal positional vertigo

H. TIBILLI[1], S. ABUŞ[1], M. KOPARAL[2], H. KAYA[1], O. BAYAR KAPICI[3]

[1]Department of Cardiology, Faculty of Medicine, Adıyaman University, Adıyaman, Turkey
[2]Department of Otorhinolaryngology, Faculty of Medicine, Adıyaman University, Adıyaman, Turkey
[3]Department of Radiology, Adıyaman Training and Research Hospital, Adıyaman, Turkey

Abstract. – OBJECTIVE: Benign Paroxysmal Positional Vertigo (BPPV) is a common vestibular disorder characterized by brief episodes of intense vertigo, often accompanied by nausea and nystagmus. The frontal QRS-T (fQRS-T) angle, a novel indicator of ventricular depolarization and repolarization heterogeneity, has garnered attention due to its potential to reveal insights into cardiac function. This study aimed to investigate the potential relationship between the fQRS-T angle and inflammation markers in individuals with BPPV.

PATIENTS AND METHODS: The study encompassed 49 BPPV patients and 51 healthy individuals as a control group. Laboratory assessments were conducted to measure inflammation parameters. Electrocardiogram (ECG) data was analyzed, focusing on conduction parameters including fQRS-T angle, QRS duration, QT interval, and corrected QT interval.

RESULTS: The study revealed that the fQRS-T angle was significantly higher in BPPV patients compared to the control group ($p<.001$). Moreover, inflammation markers such as the neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR), monocyte-to-lymphocyte ratio (MLR), and C-reactive protein-to-albumin ratio (CAR) were notably elevated in BPPV patients ($p<.001$, for all). The findings of the correlation analysis demonstrated a strong association between NLR and the fQRS-T angle (r=.718, $p<.001$). Additionally, the results of the linear regression analysis indicated that NLR positively predicted the fQRS-T angle ($p<.001$).

CONCLUSIONS: The study's outcomes have underscored a significant increase in the fQRS-T angle among BPPV patients, suggesting altered ventricular repolarization dynamics. The strong correlation between NLR and the fQRS-T angle raises intriguing possibilities of inflammation's potential role in influencing cardiac electrophysiology. The study contributes to the growing body of evidence suggesting that BPPV might have implications beyond its immediate vestibular manifestations.

Key Words:
Benign paroxysmal positional vertigo, Neutrophil to lymphocyte ratio, Frontal QRS-T angle.

Introduction

Benign paroxysmal positional vertigo (BPPV) is a common vestibular disorder characterized by recurrent episodes of vertigo triggered by specific head movements[1]. Cardiovascular diseases, on the other hand, encompass a spectrum of conditions affecting the heart and blood vessels[2]. Despite their seemingly disparate nature, recent research has prompted investigations[3] into the potential links and underlying mechanisms connecting BPPV and cardiovascular diseases.

A 2015 study conducted by Messina et al[4] observed a higher prevalence of cardiovascular diseases among individuals diagnosed with BPPV. The study suggested a potential correlation between BPPV and cardiovascular conditions, sparking further interest in exploring this relationship.

Research[5] has revealed a potential connection between autonomic dysfunction and both BPPV and cardiovascular diseases. The autonomic nervous system regulates various involuntary bodily functions, including heart rate and blood pressure. It has been observed[5] that individuals with BPPV may experience disruptions in autonomic function. This is intriguing, given that autonomic dysfunction is also associated with cardiovascular risk factors.

The frontal QRS-T (fQRS-T) angle reflects the absolute angle difference between ventricular depolarization (QRS axis) and repolarization (T axis). This novel indicator highlights depolarization and repolarization heterogeneity. Derived from common electrocardiogram (ECG) records, it is computed by subtracting the QRS axis from the T axis. Past research[6] has shown the fQRS-T

Corresponding Author: S. Abuş, MD; e-mail: sabus@adiyaman.edu.tr

angle's predictive role in diverse cardiovascular disorders, including its correlation with arrhythmia and sudden cardiac death risks.

Some studies[7-9] have suggested an association between BPPV and cardiac arrhythmias. Since the fQRS-T angle reflects ventricular repolarization, abnormalities in this angle might signify a shared autonomic dysfunction that contributes to both BPPV and cardiac arrhythmias. It is hypothesized[10] that autonomic imbalances might affect the inner ear's function, leading to BPPV, while simultaneously influencing cardiac repolarization dynamics, as reflected by the fQRS-T angle.

The aim of this study is to examine how different inflammation markers and fQRS-T angles in patients with BPPV are compared to healthy people and whether there is a relationship between these values.

Patients and Methods

Patients and Study Setting

This study adopts a descriptive cross-sectional design and received approval from the Adiyaman University's Ethics Committee (Approval No.: 2022/3-2). In accordance with the Helsinki Declaration, informed consent was obtained from all participants. The research encompassed patients aged 18-45 diagnosed with BPPV, evaluated in the cardiology outpatient clinic following otolaryngology assessment. Doppler ultrasonography was performed in patients with additional symptoms such as tinnitus and hearing loss. Patients with BPPV and concomitant conditions like diabetes mellitus (n=4), hypertension (n=2), epilepsy (n=1), subclavian steal syndrome (n=1) and colon cancer (n=1) were excluded. The study excluded those with a family history of coronary artery disease (n=2), arrhythmia, and heart valve disease (n=1). Subjects with iron deficiency anemia (n=2) or acute infection (n=2) based on hemogram and biochemistry results were not included. Consequently, the study comprised 23 males and 26 females, all aged 18-45. A flowchart illustration of the study's sample is shown in Figure 1.

The healthy control group consisted of individuals attending the cardiology outpatient clinic for employment checkups, military service examinations, and driver's license assessments, devoid of any diseases. The healthy control group encompassed 34 females and 17 males.

Electrocardiogram Examination

Two independent cardiologists blindly analyzed the 12-lead ECG of each patient. A CardioFax S device (Nihon Kohden, Tokyo, Japan) was employed to record a 12-lead ECG (50 mm/s, 10 mm/mV) in a supine position. Resting heart rate

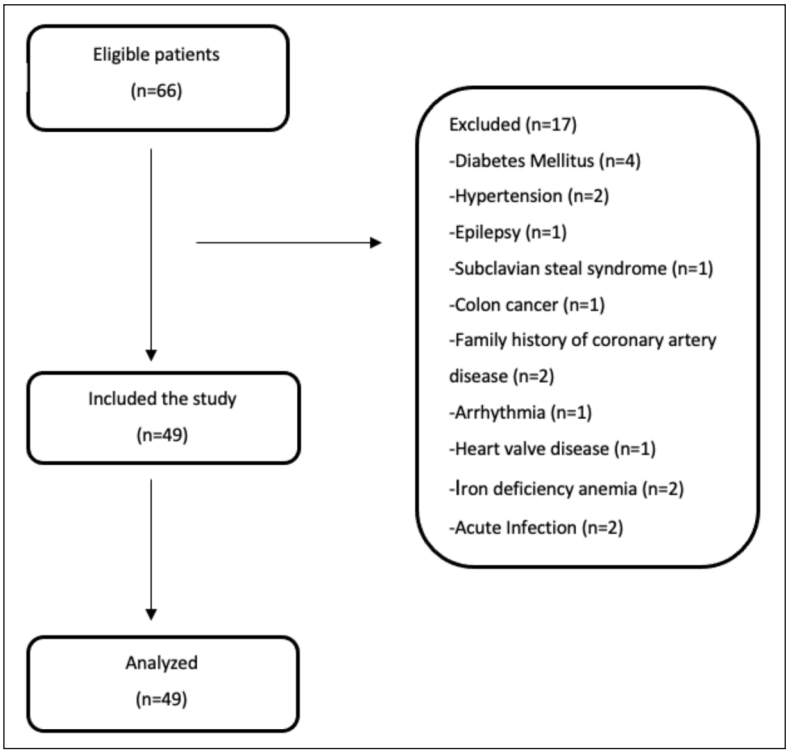

Figure 1. Flowchart illustration of the study's sample.

was calculated from ECG data, using calipers and magnifying glasses to reduce errors. QRS duration marked the beginning to the end of the QRS complex, while the QT interval spanned from the QRS start to the T-wave end. QT interval was heart rate corrected via Bazett's formula[11].

The QRS and T wave axes were available in ECG machine's automated records. From these axes, the fQRS-T angle was computed as the absolute difference between frontal plane QRS and T axes. The axes themselves were derived by subtracting 360° if the direction exceeded 180° on a 0-360° scale.

Laboratory Analyses

Upon hospital admission, venous blood samples were analyzed. The CELL-DYN Ruby device (Abbott Diagnostics, Abbott Park, IL, USA) quantified total white blood cell count, along with neutrophil, lymphocyte, monocyte, eosinophil, and basophil counts in x 10^3 cells/mm^3. Hemoglobin, hematocrit, and thrombocyte counts were also measured. C-reactive protein (CRP), creatinine, and urea levels were assessed using Abbott Diagnostics' biochemistry kits and the Architect c8000 Chemistry System machine. Plasma concentrations of total cholesterol, fasting triglycerides, high density cholesterol (HDL), low density cholesterol (LDL), and fasting blood glucose were determined through Cobas 6000 (Roche Diagnostics GmbH, Mannheim, Germany) enzymatic chemical clearing process. Calculations included neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR), monocyte-to-lymphocyte ratio (MLR), and CRP to albumin ratio (CAR).

Statistical Analysis

Analysis was conducted using SPSS 26.0 for Mac (IBM Corp., Armonk, NY, USA). Categorical data were presented as percentages and numbers. Data normality was assessed with the Kolmogorov-Smirnov test. For normally distributed parameters, mean and standard deviation were reported, while parameters deviating from normality were indicated as median [minimum-maximum]. To compare ECG and laboratory data between patients and controls, independent samples *t*-test, Mann-Whitney U test, and Chi-square test were employed. Pearson's correlation analysis and linear regression explored inflammation-ECG connections. Significance was set at $p<0.05$.

Results

Table I presents the comparison of age, gender, and ECG parameters between BPPV patients and healthy controls (HCs). The results revealed no significant age or gender differences between the groups ($p=.477$ and $p=.165$, respectively). ECG parameters including QRS duration, QT interval, and corrected QT interval exhibited no significant disparities ($p>.05$, all). Notably, the fQRS-T angle was notably wider in BPPV patients than in HCs ($p<.001$).

Table II highlights the contrast in laboratory parameters between BPPV patients and HCs. BPPV patients exhibited significantly higher neutrophil counts ($p=.005$), while lymphocyte counts were significantly lower in comparison ($p<.001$). Elevated CRP values were observed in BPPV patients, accompanied by significantly lower albumin levels ($p=.001$ and $p<.001$, respectively). Additionally, BPPV patients displayed significantly elevated CAR, NLR, MLR, and PLR values ($p<.001$, all).

Pearson's correlation analysis (Table III) unveiled a significant positive correlation between NLR, MLR, PLR, and the fQRS-T angle in BPPV patients.

Table I. Comparison of sociodemographic and ECG parameters of patients with benign paroxysmal positional vertigo and healthy controls.

	BPPV Patients (n=49) M±SD or n (%)	HCs (n=51) M±SD or n (%)	p
Age	34.65±6.70	33.73±6.31	.477[1]
Gender			
Female	26 (53.1)	34 (66.6)	.165[2]
Male	23 (46.9)	17 (33.4)	
Heart Rate (bpm)	82.69±13.95	78.14±12.85	.092[1]
QRS (msec)	90.00±9.05	88.16±8.36	.292[1]
QT (msec)	365.80±29.61	367.41±29.45	.785[1]
QTc (msec)	411.35±24.45	407.04±31.24	.446[1]
fQRS-T angle (^{0})	51.10±17.44	21.78±17.37	**<.001[1]**

[1]Independent *t*-test was used. [2]Pearson's Chi-square test was used. $p<.05$ was accepted as statically significant. BPPV: benign paroxysmal positional vertigo; HCs: Healthy Controls; QTc: corrected QT interval; bpm: beat per minute; fQRS-T: Frontal QRS-T.

Table II. Comparison of laboratory parameters of patients with benign paroxysmal positional vertigo and healthy controls.

	BPPV Patients (n=49) M±SD or med. [min-max]	HCs (n=51) M±SD or med. [min-max]	p
Hemoglobin, mg/dL	14.81±1.96	14.34±2.14	.251[1]
Glucose, mg/dL	93.80±18.81	88.92±16.92	.176[1]
Platelet, 10^3/µL	243.20 [150.10-342.70]	232.70 [170.50-366.50]	.264[2]
WBC, 10^3/µL	8.18±2.08	8.08±2.04	.812[1]
Neutrophil, 10^3/µL	5.86±1.60	4.91±1.64	**.005[1]**
Lymphocyte, 10^3/µL	1.52±0.60	2.39±0.73	**<.001[1]**
Monocyte, 10^3/µL	0.54±0.23	0.53±0.21	.813[1]
Eosinophil, 10^3/µL	0.13±0.12	0.15±0.11	.306[1]
Basophil, 10^3/µL	.09 [.00-0.27]	.08 [.00-0.27]	.904[2]
Albumin (mg/dL)	4.00 [3.30-4.30]	4.30 [3.50-6.70]	**<.001[2]**
CRP (mg/dL)	.20 [.20-.60]	.20 [.20-2.00]	**.001[2]**
Total Cholesterol (mg/dL)	176.49±44.58	170.01±40.14	.444[1]
HDL (mg/dL)	62.95±13.75	67.00±17.11	.196[1]
LDL (mg/dL)	79.57±29.27	76.81±29.37	.644[1]
Fasting Triglyceride (mg/dL)	163.82±149.51	127.59±114.71	.176[1]
CAR	.05 [.05-.17]	.04 [.03-.57]	**<.001[2]**
NLR	4.18±1.19	2.25±1.09	**<.001[1]**
MLR	.40±.23	.24±.14	**<.001[1]**
PLR	170.69 [66.12-474.17]	101.04 [59.77-324.29]	**<.001[2]**

[1]Independent *t*-test was used. [2]Mann-Whitney U test was used. $p<.05$ was accepted as statically significant. BPPV: Benign Paroxysmal Positional Vertigo; HCs: Healthy Controls; CRP: C-reactive protein; HDL: High-density cholesterol; LDL: Low-density Cholesterol; CAR: C-reactive protein/albumin ratio; NLR: Neutrophil/Lymphocyte ratio; MLR: Monocyte/Lymphocyte ratio; PLR: Platelet/Lymphocyte ratio.

The strong relationship between NLR and fQRS-T angle was also affirmed through linear regression analysis (ANOVA F: 37.126, $p<.001$, Adjusted R2: .790), as demonstrated in Table IV.

Table III. Correlation analysis of inflammation markers and frontal QRS-T angle in the patients with benign paroxysmal positional vertigo.

		fQRS-T Angle
Age	r	.104
	p	.303
NLR	r	.718
	p	**<.001**
MLR	r	.527
	p	**<.001**
PLR	r	.608
	p	**<.001**
CAR	r	.058
	p	.567

Pearson's correlation analyses were used. $p<.05$ was accepted as statistically significant. CAR: C-reactive protein/albumin ratio; NLR: Neutrophil/Lymphocyte ratio; MLR: Monocyte/Lymphocyte ratio; PLR: Platelet/Lymphocyte ratio.

Discussion

The study revealed a significant increase in the fQRS-T angle among BPPV patients. Notably, this is the first literature study to associate the elevated fQRS-T angle with heightened cardiovascular risk in BPPV patients. Additionally, inflammatory parameters were notably elevated in these patients, with a discernible link between inflammatory markers and the fQRS-T angle observed.

The fQRS-T angle is an electrocardiographic measure that quantifies the spatial relationship between the QRS and T-wave vectors. It reflects the overall dispersion of ventricular repolarization and is associated with cardiac autonomic function. A wider fQRS-T angle indicates increased inhomogeneity of ventricular repolarization, which has been linked to autonomic dysregulation and an increased risk of arrhythmias[12]. Some studies[13] have suggested an association between BPPV and cardiac arrhythmias. Since the fQRS-T angle reflects ventricular repolarization, abnormalities in this angle might signify a shared autonomic dysfunction that contributes to both BPPV and cardiac arrhythmias. It is hypothesized[13] that autonomic imbalances might affect the inner ear's function, leading to BPPV, while simultaneously influencing cardiac repolarization dynamics, as reflected by the fQRS-T angle.

The NLR is a biomarker reflecting the inflammatory state of the body. Elevated NLR values can

Table IV. Linear Regression Analysis of Frontal QRS-T angle by inflammation markers in the patients with benign paroxysmal positional vertigo.

	B	Beta	*t*	*p*	95% CI Lower	Upper
Age	-.158	-.061	-.866	.391	-.526	.210
Gender	-2.605	-.075	-1.087	.283	-7.438	2.228
NLR	11.951	.821	8.951	**<.001**	9.258	14.644
MLR	10.945	.146	1.656	.105	-2.380	24.269
CAR	61.560	.104	1.470	.149	-22.893	146.013
Constant	-1.010		-.104	.917	-20.530	18.511

Linear regression analyses were used. $p<.05$ was accepted as statically significant. NLR: Neutrophil/Lymphocyte ratio; MLR: Monocyte/Lymphocyte ratio; PLR: Platelet/Lymphocyte ratio; CAR: C-reactive protein/albumin ratio.

signify increased levels of inflammation[14]. Inflammation can impact the function of the inner ear's balance organs. Damage caused by inflammation might contribute to the development of BPPV[15]. A study[16] has demonstrated that BPPV patients exhibit higher NLR values. This suggests a potential association between inflammation or immune system activation and BPPV. Alterations in immune system regulation can affect the balance mechanisms within the inner ear, potentially paving the way for the development of BPPV. NLR, as a marker of inflammation, may serve as a factor that influences the balance mechanisms within the inner ear, potentially triggering the development of BPPV. However, further research and evidence are required for the utilization of NLR as a causative or therapeutic factor for BPPV. Neutrophils damage the vessel walls through the mediators they secrete, while lymphocytes exert an antiatherosclerotic effect. Therefore, elevated NLR is associated with impaired vascular health and an increased risk of cardiovascular disease[17]. This study found a positive correlation between fQRS-T, an indicator of cardiovascular disease risk, and NLR.

The MLR, an emerging biomarker, reflects the balance between pro-inflammatory monocytes and anti-inflammatory lymphocytes in the bloodstream[18]. Inflammation can impact the inner ear's delicate structures. The immune system plays a role in inner ear homeostasis. Dysregulation of immune responses could disturb vestibular function, potentially leading to BPPV. Immune system alterations can influence the inner ear's sensitivity to changes in pressure and fluid dynamics, which are associated with BPPV attacks. Limited research has explored the association between MLR and BPPV. However, studies[19] have suggested that an elevated MLR might be linked to the presence of BPPV. Imbalances in the immune response, as reflected by MLR, might contribute to the development or persistence of BPPV symptoms.

CRP levels reflect inflammation occurring in the body. Elevated CRP levels are associated with increased inflammation. Albumin levels reflect the body's protein balance[20]. Inflammation can influence albumin levels, potentially leading to imbalances that affect BPPV development. Inflammation can cause changes in inner ear structures, contributing to the onset of BPPV. Studies[21] have indicated an increased CAR in BPPV patients. This suggests a potential association between inflammation and BPPV. In situations of heightened inflammation, the sensitivity of balance organs and pressure changes in the inner ear can alter, potentially triggering BPPV symptoms.

The PLR, a biomarker reflecting the balance between platelets and lymphocytes in the bloodstream, has gained attention as a potential indicator of inflammation and systemic stress. Elevated PLR values are often associated with increased inflammation and oxidative stress. Immune system dysfunction and inflammation have been implicated in inner ear disorders, including BPPV. Changes in immune response could disrupt vestibular function, potentially leading to the onset of BPPV. Immune system alterations might also influence the sensitivity of the inner ear to changes in pressure, which are known triggers for BPPV attacks. Research exploring the link between PLR and BPPV is limited, but studies[22] have suggested that increased PLR values might be associated with BPPV occurrence.

Among the multitude of ECG parameters, the fQRS-T angle stands out as an intriguing measure that offers insights into the spatial orientation of ventricular depolarization and repolarization vectors. This parameter has gained recognition for its potential prognostic value in assessing cardiovascular health and arrhythmia risk[23]. In this article, we delve into the significance of the fQRS-T angle in cardiac electrophysiology and its implications for clinical practice.

The heart's electrical activity is orchestrated by a sequence of depolarization and repolarization events. The QRS complex corresponds to ventricular depolarization, while the T-wave signifies ventricular repolarization. The fQRS-T angle integrates information from both phases, capturing the spatial orientation of these electrical processes. Ventricular arrhythmias are often triggered by irregularities in the heart's electrical conduction. Increased dispersion of repolarization, as reflected by a wider fQRS-T angle, has been linked to an augmented risk of arrhythmias. Inhomogeneity in repolarization creates a substrate conducive to reentrant circuits, which can lead to tachyarrhythmias. Research[24] has indicated that a wider fQRS-T angle is associated with an elevated risk of cardiac mortality. This parameter has the potential to serve as an independent predictor of arrhythmias and cardiovascular events[25]. Changes in the fQRS-T angle have been observed in various cardiac conditions, including myocardial infarction, heart failure, and hypertrophic cardiomyopathy. Monitoring these changes can aid in disease identification and tracking. The fQRS-T angle's predictive capacity for arrhythmias opens doors for improved risk assessment and personalized management strategies. It could be integrated into existing risk-scoring systems to enhance their accuracy. Understanding the role of the fQRS-T angle in arrhythmogenesis might lead to novel therapeutic approaches. Targeting the factors that influence this parameter, such as inflammation, could mitigate arrhythmia risk[26].

Inflammation is a key player in the development and progression of various cardiovascular diseases, including atherosclerosis, heart failure, and arrhythmias. Inflammatory markers, such as CRP and interleukins, are often elevated in individuals with cardiovascular diseases. Inflammation can lead to structural and electrical remodeling of the heart, influencing its electrophysiological properties. Inflammation-induced alterations in myocardial tissue properties could influence the spread of electrical impulses and contribute to heterogeneity in ventricular repolarization, as reflected by the fQRS-T angle[27]. Immune responses and inflammatory cascades might directly or indirectly affect ion channels and cardiac ion channel expression, impacting the electrophysiological parameters captured by the fQRS-T angle. Emerging evidence[28] suggests a potential link between the fQRS-T angle and inflammation. Inflammation, a hallmark of many cardiovascular diseases, may influence cardiac electrophysiology and ventricular repolarization heterogeneity, as captured by the fQRS-T angle. As research advances, a clearer understanding of this relationship could have significant implications for risk assessment, treatment, and management of cardiac arrhythmias.

Günlü and Aktan[29] found that TpTe/QT and fQRST angles were significantly higher in BPPV patients compared to healthy controls. Apart from these ventricular arrhythmia risk indicators, they found autonomic dysfunction in favor of the sympathetic system in BPPV patients. Although our study is in favor of fQRS-T angle results, the close relationship between fQRS-T angle and inflammatory parameters makes our study different.

Limitations

Study limitations include small patient sample size, single-hospital data usage, and absence of follow-up for malignant cardiac arrhythmias or sudden cardiac death.

Conclusions

The study contributes to the growing body of evidence suggesting that BPPV might have implications beyond its immediate vestibular manifestations. The elevated fQRS-T angle observed in BPPV patients raises questions about its potential clinical significance. Additionally, the connection between inflammation markers and altered cardiac electrophysiology warrants further exploration. Future research could delve deeper into understanding the mechanisms behind these associations and their potential implications for cardiac arrhythmias and overall cardiovascular health.

This study sheds light on the correlation between the fQRS-T angle and inflammation markers in BPPV patients. The elevated fQRS-T angle among BPPV patients suggests a potential link between vestibular dysfunction and altered cardiac repolarization. The close relationship between NLR and the fQRS-T angle underscores the intricate interplay between inflammation and cardiac electrophysiology. As research progresses, these findings could open avenues for enhanced risk assessment and management strategies for individuals with BPPV.

Authors' Contributions

SA, HK, MK, HT, OBK collected data and designed the study. The manuscript was revised by SA, HK, HT. The

manuscript was written by SA, HK, MK, HT, OBK. All authors read and approved the final manuscript.

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Ethics Approval
The study was performed after the approval of the Adiyaman University Clinical Research Ethics Committee (IRB number: 2022/3-2). The study was carried out according to the Helsinki Declaration.

Informed Consent
A written informed consent form was taken from all participants to be included in the study, and they were informed that participation was voluntary, and they could be free to withdraw from the research.

Data Availability
The datasets used and analyzed during the current study are available from the corresponding author upon reasonable request.

Conflicts of Interest
The authors declare that they have no competing interests.

ORCID ID
H. Tibilli: 0000-0002-1058-1908
S. Abuş: 0000-0003-2464-4970
M. Koparal: 0000-0002-0749-0407
H. Kaya: 0000-0002-5925-5150
O. Bayar Kapici: 0000-0003-0863-9127.

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