Evaluation of Tp-e interval and Tp-e/QT ratio in patients with rheumatoid arthritis

Romatoit artritli hastalarda Tp-e süresi ve Tp-e/QT oranının değerlendirilmesi

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ABSTRACT

Objectives: Several studies have suggested that the interval from the peak to the end of the electrocardiographic T wave (Tp-e) may correspond to the transmural dispersion of repolarization and that increased Tp-e interval and Tp-e/QT ratio are associated with malignant ventricular arrhythmias. The aim of this study was to evaluate ventricular repolarization by using the Tp-e interval and Tp-e/QT ratio in patients with rheumatoid arthritis (RA), and to assess the relation with inflammation.

Study design: Ninety-six patients (72 females, 24 males; mean age 43.8±11.8 years) with RA and 50 controls (35 females, 15 males; mean age 44.2±11.1 years) were included. From the 12-lead electrocardiogram, Tp-e interval and Tp-e/QT ratio were measured. Blood samples were taken for erythrocyte sedimentation rate (ESR) and plasma levels of C-reactive protein (CRP). These parameters were compared between groups. The relationship between ventricular repolarization and inflammation was assessed by Pearson correlation coefficients.

Results: Tp-e interval and Tp-e/QT ratio were increased in RA patients compared to the controls (72.6±8.2 *vs* 66.4±8.5 ms, 0.20±0.02 *vs* 0.18±0.02; p<0.001 and p<0.001, respectively). The Tp-e interval was significantly correlated with CRP, ESR, and disease activity score (DAS-28) (r=0.56, p<0.001, r=0.57, p<0.001, and r=0.29, p=0.02, respectively). The Tp-e/QT ratio was also correlated with CRP, ESR, and DAS-28 score (r=0.43, p<0.001, r=0.53, p<0.001, and r=0.25, p=0.03, respectively).

Conclusion: In RA patients, the increased frequency of ventricular arrhythmias may be explained by increased indexes of ventricular repolarization and their relationship with inflammation.

ÖZET

Amaç: Elektrokardiyogramda T dalgasının tepesinden sonuna kadar olan aralığın (Tp-e) repolarizasyon dispersiyonuna karşılık gelebileceği ve Tp-e aralığı ve Tp-e/QT oranındaki artışın hayatı tehdit eden ventrikül aritmileri ile ilişkili olduğu çeşitli çalışmalarda gösterilmiştir. Bu çalışmanın amacı romatoit artrit (RA) saptanan hastalarda Tp-e aralığı ve Tp-e/QT oranını kullanarak ventrikül repolarizayonunu değerlendirmek ve enflamasyon ile ilişkisini araştırmaktır.

Çalışma planı: Romatoit artritli 96 hasta (72 kadın, 24 erkek; ort. yaş 43.8±11.8 yıl) ve kontrol grubu için 50 kişi (35 kadın, 15 erkek; ort. yaş. 44.2±11.1 yıl) çalışmaya alındı. On iki derivasyonlu elektrokardiyogramdan Tp-e süresi ve Tp-e/QT oranı ölçüldü. Eritrosit sedimentasyon hızı (ESH) ve C-reaktif protein (CRP) düzeyleri için kan örneği alındı. Bu parametreler gruplar arasında karşılaştırıldı. Ventrikül repolarizasyonu ve enflamasyon arasındaki ilişki Pearson korelasyon analizi ile değerlendirildi.

Bulgular: Kontrol grubu ile karşılaştırıldığında, Tp-e aralığı ve Tp-e/QT oranı RA'lı hastalarda daha yüksek idi (sırasıyla, 72.6±8.2 ve 66.4±8.5 ms, p<0.001 ve 0.20±0.02 ve 0.18±0.02, p<0.001). Tp-e aralığı, CRP, ESH ve hastalık aktivite skoru (DAS-28) ile anlamlı olarak bağıntılı idi (sırasıyla, r=0.56, p<0.001, r=0.57, p<0.001, ve r=0.29, p=0.02). Tp-e/QT oranı da CRP, ESH ve DAS-28 skoru ile anlamlı olarak bağıntılıydı (sırasıyla, r=0.43, p<0.001, r=0.53, p<0.001 ve r=0.25, p=0.03).

Sonuç: Romatoit artritli hastalarda ventrikül aritmisi sıklığındaki artış, ventrikül repolarizasyon indekslerindeki artış ile ve bunların enflamasyon ile olan ilişkisi ile açıklanabilir.

Received: July 19, 2013 *Accepted:* August 13, 2013

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Rheumatoid arthritis (RA) is a chronic multisystemic and inflammatory disease. The frequency of cardiovascular disease is increased in RA patients, including ischemic heart disease, systolic and/or diastolic heart failure, pericarditis, myocarditis, vasculitis, conduction system abnormalities, and arrhythmias. In addition, this patient population suffers significantly increased cardiovascular mortality and sudden cardiac death, when compared with the general population.[1-3]

Increased sympathetic activity and inflammatory activity may cause cardiac arrhythmias and cardiovascular death by causing electrical disturbances during ventricular repolarization in these patients.[4-6] Myocardial repolarization can be evaluated with QT interval (QT), corrected QT interval (QTc), QT dispersion (QTd), and transmural dispersion of repolarization.[7,8] Previous studies have indicated that the Tp-e interval, which is the interval between the peak and the end of T wave on electrocardiogram (ECG), is accepted as an index of total dispersion of repolarization (transmural, apicobasal, and global).[8,9] Furthermore, an increased Tp-e interval might be a useful index to predict ventricular tachyarrhythmias and cardiovascular mortality.[10-13] However, the Tp-e interval might be affected by variations in body weight and heart rate.[14] Recently, it was suggested that the Tp-e/QT ratio may serve as an accurate index for the dispersion of ventricular repolarization, independent of alterations in heart rate.[14,15] It has also been suggested that the Tp-e/QT ratio is a more accurate predictor of ventricular arrhythmogenesis than the QT, QTc and Tp-e intervals.[14]

Ventricular repolarization has been evaluated by using T wave and QT interval measurements in patients with RA.[16-18] However, the Tp-e interval and Tp-e/QT ratio, novel repolarization indexes, have not been studied in these patients. The aim of this study was to evaluate ventricular repolarization by using the Tp-e interval and Tp-e/QT ratio in RA patients, and to assess the relation with inflammation.

PATIENTS AND METHODS

Study populations

The study included 96 consecutive patients (72 females, 24 males; mean age, 43.8±11.8 years) who were diagnosed as having RA according to the revised classification of the American College of Rheumatology.[19] The control group comprised 50 age- and gender-matched healthy volunteers (35 females, 15 males; mean age, 44.2±11.1 years) selected among office staff in our hospital.

Abbreviations:

CRP	*C-reactive protein*
DAS-28	*Disease Activity Score-28*
ECG	*Electrocardiogram*
ESR	*Erythrocyte sedimentation rate*
LV	*Left ventricular*
QT	*QT interval*
QTc	*Corrected QT interval*
QTd	*QT dispersion*
RA	*Rheumatoid arthritis*

Exclusion criteria were the presence of the following: coronary artery disease, arterial hypertension, left ventricular (LV) wall motion abnormality, LV ejection fraction less than 50%, primary cardiomyopathy, valvular heart disease, bundle branch block and atrioventricular conduction abnormalities on the ECG, thyroid dysfunction, anemia, hypercholesterolemia, electrolyte imbalance, renal failure, pulmonary disease, and poor electrocardiographic imaging. All the patients were in sinus rhythm, and none was taking medications such as anti-tumor necrosis factor drugs, antiarrhythmics, tricyclic antidepressants, antihistaminics, or antipsychotics. All the patients were receiving one or more disease-modifying anti-rheumatic drugs (hydroxychloroquine, methotrexate, and sulfasalazine) and steroids. The findings of transthoracic echocardiographic examination and Disease Activity Score-28 (DAS-28)[20] were obtained from the medical records. Blood samples were taken for erythrocyte sedimentation rate (ESR), plasma levels of C-reactive protein (CRP, mg/L), and rheumatoid factor (IU/ml). Written informed consent was obtained from each subject, and the institutional ethics committee approved the study protocol.

Electrocardiography

The 12-lead ECG was recorded at a paper speed of 50 mm/s (Hewlett Packard, Page-writer, USA) in the supine position. ECG measurements of QT and Tp-e intervals were performed by two cardiologists who were blinded to the patient data. To decrease the error measurements, QT and Tp-e intervals were measured manually with calibers and magnifying glass. Subjects with U waves on their ECGs were excluded from the study. An average value of three readings was calculated for each lead. The QT interval was measured from the beginning of the QRS complex to the end of the T wave, and was corrected for heart rate using the Bazett formula: $cQT = QTd\sqrt{}$ (R-R interval). The QTd

was calculated by subtracting the shortest QT or QTc interval in any lead from the longest one. The Tp-e interval was defined as the interval from the peak of T wave to the end of T wave, and was corrected for heart rate. Measurements of the Tp-e interval were performed from precordial leads. The Tp-e/QT ratio was calculated from these measurements. Interobserver and intraobserver coefficients of variation were 2.5% and 2.8%, respectively.

Statistical analysis

The Statistical Package for the Social Sciences 15.0 program (SPSS Inc., Chicago, IL, USA) was used for the statistical study. All values are given as mean±standard deviation. Mean values of continuous variables were compared between groups using the Student t test or Mann-Whitney U-test, according to whether they were normally distributed or not, as tested by the Kolmogorov-Smirnov test. The chi-square test was used to assess differences between categorical variables. Pearson's correlation coefficients were used to assess the strength of the relationship between continuous variables. A *p* value of less than 0.05 was considered significant.

RESULTS

Clinical characteristics and echocardiographic findings of the two groups are shown in Table 1. Age, sex, body mass index, body surface area, smoking status, systolic and diastolic blood pressure, heart rate, LV end-diastolic dimension, LV end-systolic dimension, LV mass, left atrium dimension, and LV ejection fraction were similar between the two groups (p>0.05). ESR, plasma level of CRP and rheumatoid factor were significantly higher in the patients with RA as compared with controls (all p values <0.001). The mean DAS-28 score was 4.4±1.2 in RA patients, and the mean disease duration was 70.6±65.4 months (Table 1).

Table 1. Clinical characteristics, laboratory and echocardiographic findings of the patients

	Patients with RA (n=96)			Control group (n=50)			*p*
	n	%	Mean±SD	n	%	Mean±SD	
Age (years)			43.8±11.8			44.2±11.1	NS
Sex							
Female	72	75		35	70		NS
Male	24	25		15	30		NS
Body mass index (kg/m^2)			27.9±4.6			27.8±5.1	NS
Body surface area (m^2)			1.8±0.2			1.8±0.1	NS
Smoking	20	21		11	22		NS
Systolic blood pressure (mmHg)			116.2±11.4			117.0±10.1	NS
Diastolic blood pressure (mmHg)			74.7±7.5			74.3±7.2	NS
Heart rate (beats/min)			81.1±10.6			79.6±11.3	NS
LV EDD (mm)			46.9±3.7			47.3±4.0	NS
LV ESD (mm)			28.9±3.4			29.4±3.4	NS
LV mass index (g/m^2)			101.6±10.7			100.5±9.8	NS
Left atrial dimension (mm)			33.9±4.2			34.1±4.7	NS
LV ejection fraction (%)			67.5±8.6			66.7±5.4	NS
Erythrocyte sedimentation rate (mm/h)			30.3±14.3			14.6±11.7	**<0.001**
C-reactive protein (mg/l)			7.5±4.5			4.0±2.3	**<0.001**
Rheumatoid factor (IU/ml)			175.6±130.5			10.1±5.2	**<0.001**
Disease Activity Score 28			4.4±1.2			–	
Disease duration (months)			70.6±65.4			–	

RA: Rheumatoid arthritis; LV: Left ventricular; LV EDD: LV end-diastolic dimension; LV ESD: LV end-systolic dimension; NS: Not significant.

Table 2. Electrocardiographic findings of the groups

	Patients with RA (n=96)	Control group (n=50)	*p*
	Mean±SD	Mean±SD	
QT maximum (ms)	372.8±27.3	369.3±22.4	0.44
Corrected QT maximum (ms)	428.5±24.9	414.1±21.2	**0.001**
QT minimum (ms)	334.8±27.4	340.4±20.6	0.21
Corrected QT minimum (ms)	384.6±22.7	381.8±21.0	0.46
QT dispersion (ms)	38.0±14.2	28.9±7.9	**<0.001**
Corrected QT dispersion (ms)	43.9±17.8	32.3±8.4	**<0.001**
Tp-e (ms)	72.6±8.2	66.4±8.5	**<0.001**
cTp-e (ms)	83.6±9.6	74.4±8.6	**<0.001**
Tp-e/QT	0.20±0.02	0.18±0.02	**<0.001**

RA: Rheumatoid arthritis; Tp-e: Transmural dispersion of repolarization; cTp-e: Corrected transmural dispersion of repolarization.

Electrocardiographic parameters of the groups are shown in Table 2. Maximum corrected QT (cQTmax), QTd, cQTd, and Tp-e intervals were significantly increased in RA patients compared to the control group (p=0.001, p<0.001, p<0.001, and p<0.001, respectively). The Tp-e/QT ratio was also significantly higher in RA patients compared to the control group (p<0.001).

The Tp-e interval and Tp-e/QT ratio were not correlated with age, systolic/diastolic blood pressure, LV ejection fraction, left atrium diameter, or disease duration. The Tp-e interval was significantly correlated with CRP (r=0.56, p<0.001) (Fig. 1), ESR (r=0.57, p<0.001), and DAS-28 score (r=0.29, p=0.02). The Tp-e/QT ratio was significantly correlated with CRP (r=0.43, p<0.001) (Fig. 2), ESR (r=0.53, p<0.001), DAS-28 score (r=0.25, p=0.03), and rheumatoid factor (r=0.22, p=0.04).

DISCUSSION

The present study showed that the Tp-e interval and Tp-e/QT ratio were prolonged in patients with RA when compared to control subjects. In addition, these electrocardiographic parameters were positively correlated with plasma level of CRP and ESR.

Rheumatoid arthritis is a systemic disease characterized by increased inflammatory activity. Several forms of cardiac involvement have been described in

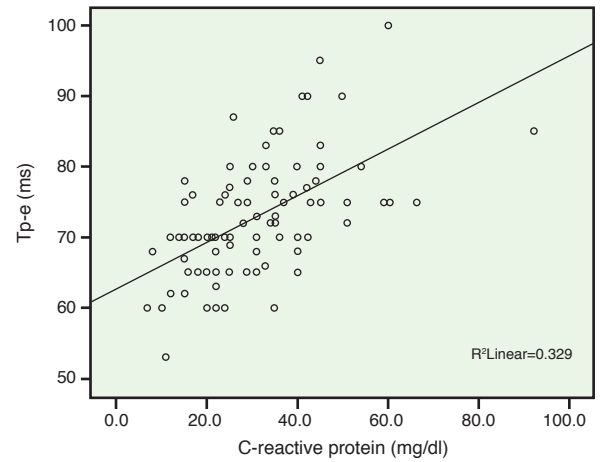

Figure 1. Correlation between the Tp-e interval and plasma level of C-reactive protein.

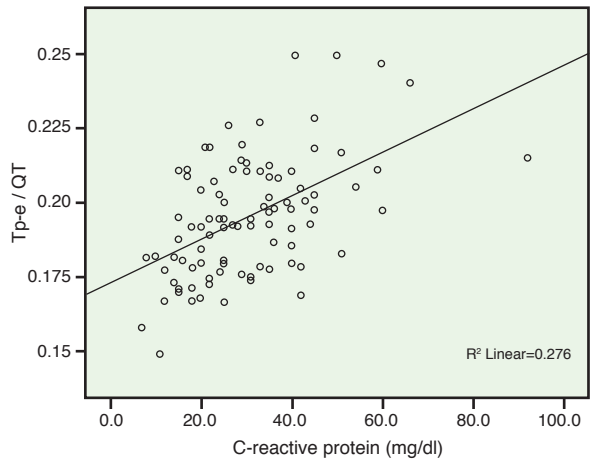

Figure 2. Correlation between the Tp-e/QT ratio and plasma level of C-reactive protein.

RA.[1-5,21] Inflammation is known to play an essential role in the pathogenesis of cardiovascular disease, such as coronary artery disease and arrhythmia.[6] Inflammatory biomarkers have been evaluated in the setting of coronary heart disease, in which chronic inflammation and thrombosis can transform a stable atherosclerotic plaque to an unstable lesion. This phenotype of acute plaque rupture is known to be related with ventricular arrhythmias and sudden cardiac death events. Elevated CRP may increase the risk of fatal complications from acute coronary syndromes. CRP may also have direct arrhythmogenic properties by locally activating complement and inducing oxidative stress and apoptosis.[22]

Cardiac rhythm disorder is one of the important causes of mortality and morbidity in patients with RA, and may be secondary to ischemia and conduction abnormalities.[1-3] Furthermore, abnormalities of ventricular repolarization due to cardiac structural changes, autonomic impairment and increased inflammatory activity could result in increased sudden cardiac deaths and ventricular arrhythmias in RA patients.[1-6] When compared with healthy controls, it has been shown that QTd and cQTd intervals were significantly longer in RA patients, and was then suggested that increased cardiovascular morbidity and mortality may be due to complex ventricular arrhythmias in these patients.[17,18] Additionally, Janse van Rensburg et al. and Evrengül et al.[4,5] reported that an inability of the autonomic nervous system could play a key role in the development of ventricular arrhythmias in RA.

The Tp-e interval and Tp-e/QT ratio are well-known electrocardiographic markers of increased dispersion of ventricular repolarization.[8,14] These markers are also used as an electrocardiographic index of ventricular arrhythmogenesis and sudden cardiac death.[8,10-16] Previous studies have shown that prolongation of the Tp-e interval is associated with increased mortality in Brugada syndrome, long QT syndromes, and hypertrophic cardiomyopathy, and in patients undergoing primary percutaneous coronary intervention for myocardial infarction.[10,12,15] Several investigators have studied repolarization patterns in patients with RA, and they have seen that QTd is increased compared to controls.[17,18] However, in these studies, only QTd had been used to assess the homogeneity of cardiac repolarization, and no information about the relation with inflammation was shown. In another study, we recently reported that the Tp-e interval and Tp-e/QT ratio were increased in ankylosing spondylitis patients, and these ventricular repolarization indexes were correlated with inflammation.[23] Myocardial fibrosis is a well-known complication of RA, which is thought to develop due to chronic inflammation.[24] Therefore, the involvement of myocardial tissue may cause the development of heterogeneity in repolarization, which may also contribute to the development of ventricular arrhythmias in patients with RA.

Our results may contribute to the pathophysiological mechanisms of the increased prevalence of ventricular arrhythmias and cardiovascular mortality risk by indicating increased ventricular repolarization heterogeneity and increased inflammation in these patients. The increased frequency of ventricular arrhythmia and sudden cardiac death might be explained by prolonged transmural dispersion and chronic ongoing systemic inflammation in RA patients. There are limited data evaluating the Tp-e interval and Tp-e/QT ratio in systemic inflammatory diseases. Therefore, our findings may be a reference for further studies.

Limitations

The major limitation of our study is its cross-sectional design and lack of follow-up of the patients. We did not assess the association between ventricular arrhythmias with the Tp-e interval and Tp-e/QT ratio. Further, the study population could not be followed up prospectively for ventricular arrhythmic episodes. Therefore, we could not evaluate the potential prognostic role of the electrocardiographic ventricular repolarization indexes with respect to future untoward events. Thus, long-term follow-up and large-scale prospective studies are needed to determine the predictive value of prolonged Tp-e interval and increased Tp-e/QT ratio in this population. Finally, manual measurement of QT and Tp-e intervals on paper-printed ECG might have underpowered the results because they would be more reliable if measured on the high-resolution screen of a digital system.

In conclusion, our study revealed that the Tp-e interval and Tp-e/QT ratio were increased in RA patients. Our results also indicated that these electrocardiographic ventricular repolarization indexes were significantly correlated with inflammation and disease activity. The Tp-e interval and Tp-e/QT ratio might be useful markers of cardiovascular morbidity and

mortality due to complex ventricular arrhythmias in patients with RA.

Conflict-of-interest issues regarding the authorship or article: None declared

REFERENCES

1. Voskuyl AE. The heart and cardiovascular manifestations in rheumatoid arthritis. Rheumatology (Oxford) 2006;45 Suppl 4:iv4-7. CrossRef
2. Charles-Schoeman C. Cardiovascular disease and rheumatoid arthritis: an update. Curr Rheumatol Rep 2012;14:455-62.
3. Seferović PM, Ristić AD, Maksimović R, Simeunović DS, Ristić GG, Radovanović G, et al. Cardiac arrhythmias and conduction disturbances in autoimmune rheumatic diseases. Rheumatology (Oxford) 2006;45 Suppl 4:iv39-42. CrossRef
4. Evrengül H, Dursunoglu D, Cobankara V, Polat B, Seleci D, Kabukçu S, et al. Heart rate variability in patients with rheumatoid arthritis. Rheumatol Int 2004;24:198-202. CrossRef
5. Janse van Rensburg DC, Ker JA, Grant CC, Fletcher L. Autonomic impairment in rheumatoid arthritis. Int J Rheum Dis 2012;15:419-26. CrossRef
6. Mountantonakis S, Deo R. Biomarkers in atrial fibrillation, ventricular arrhythmias, and sudden cardiac death. Cardiovasc Ther 2012;30:74-80. CrossRef
7. Antzelevitch C, Shimizu W, Yan GX, Sicouri S. Cellular basis for QT dispersion. J Electrocardiol 1998;30 Suppl:168-75.
8. Kors JA, Ritsema van Eck HJ, van Herpen G. The meaning of the Tp-Te interval and its diagnostic value. J Electrocardiol 2008;41:575-80. CrossRef
9. Antzelevitch C, Sicouri S, Di Diego JM, Burashnikov A, Viskin S, Shimizu W, et al. Does Tpeak-Tend provide an index of transmural dispersion of repolarization? Heart Rhythm 2007;4:1114-9. CrossRef
10. Castro Hevia J, Antzelevitch C, Tornés Bárzaga F, Dorantes Sánchez M, Dorticós Balea F, Zayas Molina R, et al. Tpeak-Tend and Tpeak-Tend dispersion as risk factors for ventricular tachycardia/ventricular fibrillation in patients with the Brugada syndrome. J Am Coll Cardiol 2006;47:1828-34. CrossRef
11. Smetana P, Schmidt A, Zabel M, Hnatkova K, Franz M, Huber K, et al. Assessment of repolarization heterogeneity for prediction of mortality in cardiovascular disease: peak to the end of the T wave interval and nondipolar repolarization components. J Electrocardiol 2011;44:301-8. CrossRef
12. Erikssen G, Liestøl K, Gullestad L, Haugaa KH, Bendz B, Amlie JP. The terminal part of the QT interval (T peak to T end): a predictor of mortality after acute myocardial infarction. Ann Noninvasive Electrocardiol 2012;17:85-94. CrossRef
13. Watanabe N, Kobayashi Y, Tanno K, Miyoshi F, Asano T, Kawamura M, et al. Transmural dispersion of repolarization and ventricular tachyarrhythmias. J Electrocardiol 2004;37:191-200. CrossRef
14. Gupta P, Patel C, Patel H, Narayanaswamy S, Malhotra B, Green JT, et al. T(p-e)/QT ratio as an index of arrhythmogenesis. J Electrocardiol 2008;41:567-74. CrossRef
15. Zhao X, Xie Z, Chu Y, Yang L, Xu W, Yang X, et al. Association between Tp-e/QT ratio and prognosis in patients undergoing primary percutaneous coronary intervention for ST-segment elevation myocardial infarction. Clin Cardiol 2012;35:559-64. CrossRef
16. Letsas KP, Weber R, Astheimer K, Kalusche D, Arentz T. Tpeak-Tend interval and Tpeak-Tend/QT ratio as markers of ventricular tachycardia inducibility in subjects with Brugada ECG phenotype. Europace 2010;12:271-4. CrossRef
17. Göldeli O, Dursun E, Komsuoglu B. Dispersion of ventricular repolarization: a new marker of ventricular arrhythmias in patients with rheumatoid arthritis. J Rheumatol 1998;25:447-50.
18. Cindas A, Gökçe-Kutsal Y, Tokgözoglu L, Karanfil A. QT dispersion and cardiac involvement in patients with rheumatoid arthritis. Scand J Rheumatol 2002;31:22-6. CrossRef
19. Arnett FC, Edworthy SM, Bloch DA, McShane DJ, Fries JF, Cooper NS, et al. The American Rheumatism Association 1987 revised criteria for the classification of rheumatoid arthritis. Arthritis Rheum 1988;31:315-24. CrossRef
20. Prevoo ML, van 't Hof MA, Kuper HH, van Leeuwen MA, van de Putte LB, van Riel PL. Modified disease activity scores that include twenty-eight-joint counts. Development and validation in a prospective longitudinal study of patients with rheumatoid arthritis. Arthritis Rheum 1995;38:44-8. CrossRef
21. Acar G, Sayarlioğlu M, Akçay A, Sökmen A, Sökmen G, Yalçintaş S, et al. Evaluation of atrial electromechanical delay and left atrial mechanical functions in patients with rheumatoid arthritis. Turk Kardiyol Dern Ars 2009;37:447-53.
22. Lagrand WK, Visser CA, Hermens WT, Niessen HW, Verheugt FW, Wolbink GJ, et al. C-reactive protein as a cardiovascular risk factor: more than an epiphenomenon? Circulation 1999;100:96-102. CrossRef
23. Acar G, Yorgun H, Inci MF, Akkoyun M, Bakan B, Nacar AB, et al. Evaluation of Tp-e interval and Tp-e/QT ratio in patients with ankylosing spondylitis. Mod Rheumatol 2013 Apr 12.
24. Kobayashi Y, Giles JT, Hirano M, Yokoe I, Nakajima Y, Bathon JM, et al. Assessment of myocardial abnormalities in rheumatoid arthritis using a comprehensive cardiac magnetic resonance approach: a pilot study. Arthritis Res Ther 2010;12:R171. CrossRef

Key words: Arthritis, rheumatoid; electrocardiography; inflammation; ventricular arrhythmogenesis.

Anahtar sözcükler: Artrit, romatoid; elektrokardiyografi; enflamasyon; ventriküler aritmogenez.