J Surg Med. 2020;4(10):887-890.
DOI: 10.28982/josam.792850

Research article
Araştırma makalesi

Journal of Surgery and Medicine
e-ISSN=2602-2079

Psoriatic arthritis is associated with myocardial repolarization dysregulation as assessed by the QTc interval and the Tp-e/QTc ratio

Psöriatik artrit, QTc aralığı ve Tp-e/QTc oranı ile değerlendirilen miyokardiyal repolarizasyon düzensizliği ile ilişkilidir

Hilal Erken Pamukcu [1], Melih Pamukcu [2]

[1] University of Health Sciences, Dışkapı Yıldırım Beyazıt Training & Research Hospital, Department of Cardiology, Ankara, Turkey
[2] University of Health Sciences, Dışkapı Yıldırım Beyazıt Training & Research Hospital, Rheumatology Clinic, Ankara, Turkey

ORCID ID of the author(s)
HEP: 0000-0001-8116-5090
MP: 0000-0002-9129-0503

Corresponding author / Sorumlu yazar:
Hilal Erken Pamukcu
Address / Adres: Dışkapı Yıldırım Beyazıt Eğitim ve Araştırma Hastanesi, Kardiyoloji Kliniği, Dışkapı Ankara, Türkiye
E-mail: hilalerkenn@gmail.com

Ethics Committee Approval: The study protocol was approved by the Ethics Committee of Dışkapı Training and Research Hospital (the approval number of 81/06 at 3/2/2020). All procedures in this study involving human participants were performed in accordance with the 1964 Helsinki Declaration and its later amendments.
Etik Kurul Onayı: Çalışma protokolü Dışkapı Eğitim ve Araştırma Hastanesi Etik Kurulu tarafından onaylandı (3/2/2020 81/06 onay numarası). İnsan katılımcıların katıldığı çalışmalardaki tüm prosedürler, 1964 Helsinki Deklarasyonu ve daha sonra yapılan değişiklikler uyarınca gerçekleştirilmiştir.

Conflict of Interest: No conflict of interest was declared by the authors.
Çıkar Çatışması: Yazarlar çıkar çatışması bildirmemişlerdir.

Financial Disclosure: The authors declared that this study has received no financial support.
Finansal Destek: Yazarlar bu çalışma için finansal destek almadıklarını beyan etmişlerdir.

Published: 10/28/2020
Yayın Tarihi: 28.10.2020

Abstract

Aim: An increased risk of arrhythmia has been shown in patients with psoriatic arthritis (PsA). Tp-e interval and Tp-e/QT ratio are considered useful markers for the assessment of myocardial repolarization heterogeneity. In present study we aim to investigate the characteristics of ventricular repolarization on the basis of QTc interval and Tp-e/QTc ratio in patients PsA.

Methods: This cross-sectional observational study included patients with psoriatic arthritis who were over 18 years of age. A control group was created with healthy volunteers. The diagnosis of psoriatic arthritis were done according to the CASPAR criteria. QT interval, corrected QT (QTc), Tp-e interval, Tp-e/QT ratio and Tp-e/QTc ratio were measured from the 12-lead electrocardiograms (ECG) of the PsA patients and control group.

Results: The study population consisted of 82 patients with PsA and 82 healthy volunteers. According to electrocardiographic parameters the heart rate was similar in both groups, but QT, QTc, Tp-e intervals; Tp-e /QT and Tp-e/ QTc ratios were significantly higher in the PsA group. The median Tp-e/QTc ratio of the patients with PsA was significantly higher than the median Tp-e/QTc ratio of the control group (0.21 (0.20-0.25) vs. 0.18 (0.17-0.19, P<0.001). The correlation analysis showed a weak correlation between Tp-e/QTc ratio and CRP value (r=0.197, P=0.012).

Conclusions: This is the first study to demonstrate the prolongation of the Tp-e/QTc ratio in patients with PsA. Prospective clinical studies to observe clinical importance of this finding particularly with regard to the arrhythmias and sudden death may be more valuable.
Keywords: Psoriatic arthritis, Electrocardiography, Tp-e/QTc ratio, Myocardial repolarization

Öz

Amaç: Psoriatik artritli (PsA) hastalarda aritmi riski artışı olduğu gösterilmiştir. Tp-e aralığı ve Tp-e/QT oranı, miyokardiyal repolarizasyon heterojenitesinin değerlendirilmesinde yararlı belirteçler olarak kabul edilmektedir. Bu çalışmada PsA hastalarında QTc aralığı ve Tp-e/QTc oranı temelinde ventriküler repolarizasyonun özelliklerini incelemeyi amaçladık.

Yöntemler: Bu kesitsel gözlemsel çalışmaya 18 yaşın üzerinde psoriatik artritli hastalar dahil edildi. Sağlıklı gönüllülerden bir kontrol grubu oluşturuldu. Psoriatik artrit tanısı CASPAR kriterlerine göre yapıldı. PsA hastalarının ve kontrol grubunun 12 kanallı elektrokardiyogramlarından (EKG) QT aralığı, düzeltilmiş QT (QTc), Tp-e aralığı, Tp-e/QT oranı ve Tp-e/QTc oranı ölçüldü.

Bulgular: Çalışma popülasyonu 82 PsA hastası ve 82 sağlıklı gönüllüden oluşuyordu. Elektrokardiyografik parametrelere göre kalp hızı her iki grupta da benzerdi, ancak QT, QTc, Tp-e aralıkları; Tp-e/QT ve Tp-e/QTc oranları PsA grubunda anlamlı olarak yüksekti. PsA'lı hastaların medyan Tp-e/QTc oranı, kontrol grubunun medyan Tp-e/QTc oranından (0,21 (0,20-0,25) ve 0,18 (0,17-0,19, P<0,001) anlamlı olarak daha yüksekti. Korelasyon analizi Tp-e/QTc oranı ile CRP değeri arasında zayıf bir korelasyon gösterdi (r=0,197, P=0,012).

Sonuçlar: Bu çalışma PsA hastalarında Tp-e/QTc oranının uzadığını gösteren ilk çalışmadır. Bu bulgunun özellikle aritmiler ve ani ölüm açısından klinik önemini gözlemlemeye yönelik ileriye dönük klinik çalışmalar daha değerli olabilir.
Anahtar kelimeler: Psoriatik artrit, Elektrokardiyografi, Tp-e/QTc oranı, Miyokardiyal repolarizasyon

How to cite/Atıf için: Pamukcu HE, Pamukcu M. Psoriatic arthritis is associated with myocardial repolarization dysregulation as assessed by the QTc interval and the Tp-e/QTc ratio. J Surg Med. 2020;4(10):887-890.

Introduction

Psoriatic arthritis (PsA) is a chronic inflammatory joint disease and included as one of the spondyloarthropathies (SpA) [1], with a prevalence of PsA in the general population of about 0.1–0.25 % [2,3]. It is known that concomitant cardiovascular risk factors are higher in psoriatic arthritis patients than in the normal population [4,5]. Cardiovascular events have also been shown to be more common in these patients than in the normal population [6].

Cardiovascular disease is the most common cause of death in psoriatic arthritis. It is thought that endothelial dysfunction and arterial stiffness may have an effect on cardiovascular morbidity and mortality in psoriatic arthritis as in many other inflammatory diseases [6].

Some previous studies have reported an increased risk of cardiac arrhythmia in psoriasis [7]. Heart rate variability, which is an indicator of cardiac autonomic regulation, has been shown to decrease in psoriatic arthritis [8]. In another study, it was shown that the Tp-e interval and Tp-e/QT ratio, which are considered the predictors of ventricular arrhythmia, were prolonged in patients with psoriasis vulgaris [9]. In our study, we planned to investigate the characteristics of ventricular repolarization on the basis of QTc interval and Tp-e/QTc ratio in patients with psoriatic arthritis, which can be seen in 6-39% of psoriasis patients [2,3].

Materials and methods

This cross-sectional observational study included patients with psoriatic arthritis who were over 18 years of age who presented to the rheumatology outpatient clinic between February 2020 and June 2020 and were diagnosed with psoriatic arthritis according to the CASPAR criteria [10] and have been treated at least 6 months.

Patients with a history of coronary artery disease, moderate to severe valvular heart disease, prior pacemaker insertion, history of dysrhythmia, cardiomyopathy, acute and chronic renal failure, abnormal thyroid function test, abnormal electrolyte values, and those who were on antiarrhythmic drug treatment and using other drugs that can affect Tp-e and QTc intervals such as antiarrhythmic agents, antipsychotic agents, tricyclic antidepressants, and b-blockers were excluded from the study. The study protocol was approved by the Ethics Committee of Dışkapı Training and Research Hospital with the approval number of 81/06 at 3/2/2020, and informed consent was obtained from each patient.

Demographic information of the patients, including age, gender and body mass index (BMI), history of hypertension (HT), hyperlipidemia, and smoking status were recorded. The laboratory tests included complete blood count, erythrocyte sedimentation rate (ESH), C- reactive protein (CRP), fasting glucose level, lipid profile, liver, and kidney and function tests.

Assessment of ECG

The 12-lead ECG recordings were taken at the supine position with a paper speed of 50 mm/sec and voltage of 10 mm/mV using standard ECG system (CardiofaxV model 9320, Nihon Kohden, Tokyo, Japan). QT interval, and Tp-e interval were measured manually. Tp-e/QT ratio and Tp-e/QTc ratio were calculated from these measurements. The onset of P wave was defined as the first visible upward departure from the baseline, and the end of P wave was defined as returning to baseline. QT interval was defined as the time from the onset of the QRS to the point at which T wave returns to baseline. QTc interval was calculated using the Bazett's formula. We used the tangent method to measure Tp-e in leads V2 and V5. The 'tangent' method of measuring Tp-e defines T-peak as the maximum positive or negative deflection of T wave from the isoelectric line. The T-end is defined as the intersection of the isoelectric line with the tangent to the down slope of the T wave. Tp-e/QTc ratio was also calculated because of the uncertainty in the literature regarding whether Tp-e is rate- dependent. All measurements were performed by two independent investigators who were blinded to the clinical status of the subjects. Tp-e measurement was not performed in cases of excessive artifact or T wave flatness.

Statistical analysis

Statistical analysis was performed using SPSS Version 23 (IBM Corp; Armonk, NY, USA) statistical software. Categorical data were presented as numbers and percentages. Continuous variables were presented as mean (standard deviation) when normally distributed, and as median and interquartile ranges otherwise. The Kolmogorov-Smirnov test was performed in order to test the normality of the numerical variables. For variables that were not normally distributed, non-parametric statistical methods were used and Mann-Whitney *U* test was performed in order to compare two independent groups. Student T test was performed in order to compare two independent groups for normally distributed variables. Categorical variables were compared with the x^2 test. Correlation analyses were used to identify the related parameters with the Tp-e/QTc ratio. Pearson's correlation coefficients were used to assess the strength of the relationship between continuous variables and Spearman's correlation analysis for non-continuous and categorical variables. A *P*-value <0.05 was considered statistically significant.

Results

Our study included 82 PsA patients and 82 age and gender-matched controls. The demographic, laboratory and data of patients with and without PsA are presented in Table 1. Patient groups with and without PsA were similar in terms of basal characteristics. Median age of the PsA group was 53.5 (45.7-60.2) years and the median age of the control group was 50 (48-55) years (*P*=0.064).

Patient groups with and without PsA were similar in terms of gender distribution, comorbidities, hypertension hyperlipidemia and smoking status. According to electrocardiographic parameters the heart rate was similar in both groups, but QT, QTc, Tp-e intervals, Tp-e /QT and Tp-e/ QTc ratios were significantly higher in the psoriatic arthritis group. The median Tp-e/QTc ratio of the patients with PsA was 0.21 (0.20-0.25). The median Tp-e/QTc ratio of the control group was 0.18 (0.17-0.19) (*P*<0.001). The electrocardiographic parameters of both the groups are shown in Table 2. Table 3 presents the correlation analyses between the Tp-e/QT ratio and the study

parameters. The single parameter that is associated with the Tp-e/QTc ratio was CRP value (r=0.197, *P*=0.012).

Table 1: Basal characteristics of the study participants

	Psoriatic arthritis (n=82)	Control (n=82)	*P*-value
Age (years)	53.5(45.7-60.2)	50(48-55)	0.064
Female Gender, n (%)	47 (57.3)	49 (59.8)	0.874
Diabetes mellitus, n (%)	6 (7.3)	8 (9.8)	0.781
Hypertension, n (%)	25 (30.5)	31 (37.8)	0.410
Hyperlipidemia, n (%)	21 (25.6)	31 (37.8)	0.131
Smoking, n (%)	13 (15.9)	16 (19.5)	0.683
BMI (kg/m^2)	23.3 (1.8)	23.1 (1.7)	0.484
Creatinine (mg/dL)	0.85 (0.3)	0.81 (0.1)	0.303
Potassium (mEq/L)	4.3(4.0-4.5)	4.4(4.1-4.5)	0.129
Fasting glucose (mg/dL)	90 (87-98)	87(86-94)	0.051
AST U/L	20 (16.5-25)	19 (16-23)	0.186
ALT U/L	20 (14.5-25)	20 (12-27)	0.941
TC (mg/dL)	194 (168-218)	181 (169-205)	0.233
Triglyceride (mg/dL)	116 (91-171)	117 (89-181)	0.897
HDL (mg/dL)	51 (46-57)	48 (41-52)	0.547
LDL (mg/dL)	123(108-143)	115 (102-130)	0.285
Hemoglobin (mg/dL)	13.2(12.3-14.2)	13.5 (13.2-13.8)	0.303
WBC	6.7 (5.7-8.1)	6.5 (5.4-8.8)	0.649
ESR (mm/sec)	10 (4.7-17.2)	7.6 (7-8.1)	0.045
CRP (mg/L)	3.8 (1.7-8.9)	3(2-4)	0.028

BMI: body mass index, TC: total cholesterol, WBC: White blood cell count, ESR: erythrocyte sedimentation rate, CRP: C reactive protein

Table 2: Electrocardiographic findings of the study participants

	Psoriatic arthritis (n=82)	Control (n=82)	*P*-value
Heart rate	79.5 (11)	78 (11)	0.720
QT (ms)	390 (378-400)	380 (370-390)	0.007
QTc (ms)	406 (390-428)	399 (380-408)	0.001
Tpe (ms)	90 (80-100)	72 (68-75)	<0.001
Tp-e/QT	0.22 (0.21-0.27)	0.19 (0.17-0.20)	<0.001
Tp-e/ QTc	0.21 (0.20-0.25)	0.18 (0.17-0.19)	<0.001

Table 3: The association between the Tp-e/QTc ratio and variables

Variables	Bivariate correlation	
	r	*P*-value
Age	0.061	0.440
Female gender	-0.018	0.820
Diabetes mellitus	-0.072	0.358
Hypertension	-0.101	0.197
Hyperlipidemia	-0.017	0.827
Smoking	0.088	0.264
BMI	0.039	0.619
WBC	0.023	0.775
ESR	0.100	0.204
CRP	0.197	0.012

BMI: body mass index, WBC: White blood cell count, CRP: C reactive protein, ESR: erythrocyte sedimentation rate

Discussion

The prevalence of cardiovascular diseases is increased in psoriatic arthritis patients and the leading cause of mortality is cardiovascular diseases [6,11-13]. There are studies about the increased risk of arrhythmia in these patients [14]. The Tp-e/QTc ratio is an electrocardiographic parameter and useful in the prediction of ventricular arrhythmias [15-17]. The association between the presence of PsA and Tp-e/QTc prolongation has not been studied before. In the present study, we showed that the Tp-e/QTc ratio is prolonged in psoriatic arthritis patients.

The T-peak to T-end interval (Tp-e) on the 12-lead electrocardiogram (ECG) is in correlation with dispersion of ventricular repolarization and considered as a predictor of ventricular tachyarrhythmia and death [18-20]. The heterogeneity of ventricular repolarization dispersion causes heterogeneity in myocardial refractoriness. This variability provides a predisposition to the ventricular arrhythmias [21]. Tp–e interval is affected by heart rate variability, Tp–e/QTc index is thought to be more meaningful to eliminate this effect [22].

In the present study both QTc and Tp-e/QTc were prolonged in PsA patients. These findings show us that repolarization is delayed and at the same time the distribution of repolarization is impaired. In a previous study it was shown that patients with psoriasis were at higher risk of developing arrhythmia, particularly for those with psoriatic arthritis, independent of traditional cardiovascular risk factors [14]. The underlying mechanism beneath the increased arrhythmic risk in PsA is not known.

Many chronic inflammatory diseases are associated with an increased incidence of atrial and ventricular arrhythmias [23,24]. Many inflammatory markers, such as tumor necrosis factor-alfa, IL-2, IL-6 were associated with cardiac arrhythmias [25].

In previous studies, elevated CRP values were associated with QTc prolongation in healthy individuals [26,27] and in patients with chronic inflammatory arthritis [28]. In our present study, CRP value was associated with Tp-e/ QTc prolongation.

The prolongation of the Tp-e/QTc ratio has been shown in many chronic inflammatory diseases in various studies [29-31]. Our study is the first that shows the prolonged Tp-e/QTc ratio in psoriatic arthritis patients. In psoriatic arthritis, many factors besides inflammation are thought to play a role in repolarization heterogeneity and increased arrhythmia risk. In some studies, patients with psoriasis have been shown to have endothelial dysfunction and coronary microvascular dysfunction [32,33]. It has also been shown that psoriasis is associated with subclinical atherosclerosis [34]. Besides the evidence showing subclinial atherosclerosis, increased comorbid cardiovascular risk factors cause the increase in cardiovascular morbidity and mortality in these patients [34-36]. In psoriatic arthritis, unlike psoriasis vulgaris, the presence of joint involvement in addition to skin findings suggests that the burden of inflammation is heavier than patients with only skin involvement and that cardiovascular risk may be higher [37]. Future studies comparing psoriasis and psoriatic arthritis patients in terms of cardiovascular risk and outcome will illuminate this issue. There are studies showing an increase in arrhythmic events in psoriasis, especially in psoriatic arthritis [14]. It is thought that severe inflammation causes atrial fibrosis, increasing atrial arrhythmia risk and, because of the damage done by vascular inflammation to the artery wall, arrhythmia risk increases due to ischemia and major cardiovascular events [38,39]. In addition, inflammation is thought to increase the risk of arrhythmia by affecting cardiomyocyte electrophysiology on ion channels dysregulation [23].

The detection of prolonged Tp-e and Tp-e/QTc ratios in PsA patients in our study suggests that there is myocardial repolarization heterogeneity and this may play a role in arrhythmic events. However, in our study, there is no clinical data about arrhythmic outcome. In the future, studies supported by ECG data such as Tp-e and Tp-e/QTc ratio, which also include data on arrhythmic outcome, will help to enlighten the pathophysiology of the increased arrhythmia risk in PsA.

Limitations

This study has some limitations. Most importantly, since this study was designed as a single-center and an observational study, we could not achieve any significant evidence about the clinical importance of the prolongation of Tp-e and Tp-e/QTc in PsA patients. Future studies comparing psoriasis patients with and without arthritis may be useful in evaluating the effect of the severity of inflammation on ECG findings.

Conclusion

Our study is the first to demonstrate the prolongation of the Tp-e interval and Tp-e/QTc ratio in patients with psoriatic arthritis. Prospective clinical studies that monitor PsA patients with ECG findings and observe their clinical outcomes, particularly with regard to the arrhythmias and sudden death in follow-up, may be more valuable.

References

1. Feld J, Weiss G, Rosner I, Rozenbaum M, Laor A, Rimar D, et al. Electrocardiographic findings in psoriatic arthritis: a case-controlled study. J Rheumatol. 2008;35(12):2379-82. Epub 2008/10/10. doi: 10.3899/jrheum.080314. PubMed PMID: 18843776.
2. Shbeeb M, Uramoto KM, Gibson LE, O'Fallon WM, Gabriel SE. The epidemiology of psoriatic arthritis in Olmsted County, Minnesota, USA, 1982-1991. J Rheumatol. 2000;27(5):1247-50. Epub 2000/05/17. PubMed PMID: 10813295.
3. Wilson FC, Icen M, Crowson CS, McEvoy MT, Gabriel SE, Kremers HM. Incidence and clinical predictors of psoriatic arthritis in patients with psoriasis: a population-based study. Arthritis Rheum. 2009;61(2):233-9. Epub 2009/01/30. doi: 10.1002/art.24172. PubMed PMID: 19177544; PubMed Central PMCID: PMCPMC3061343.
4. Husni ME, Mease PJ. Managing comorbid disease in patients with psoriatic arthritis. Current rheumatology reports. 2010;12(4):281-7. doi: 10.1007/s11926-010-0112-3. PubMed PMID: 20589485.
5. Horreau C, Pouplard C, Brenaut E, Barnetche T, Misery L, Cribier B, et al. Cardiovascular morbidity and mortality in psoriasis and psoriatic arthritis: a systematic literature review. Journal of the European Academy of Dermatology and Venereology : JEADV. 2013;27 Suppl 3:12-29. doi: 10.1111/jdv.12163. PubMed PMID: 23845149.
6. Jamnitski A, Symmons D, Peters MJ, Sattar N, McInnes I, Nurmohamed MT. Cardiovascular comorbidities in patients with psoriatic arthritis: a systematic review. Annals of the rheumatic diseases. 2013;72(2):211-6. doi: 10.1136/annrheumdis-2011-201194. PubMed PMID: 22532629.
7. Markuszeski L, Bissinger A, Janusz I, Narbutt J, Jedrzejowska AS, Zalewska A. Heart rate and arrhythmia in patients with psoriasis vulgaris. Arch Med Res. 2007;38(1):64-9. Epub 2006/12/19. doi: 10.1016/j.arcmed.2006.08.004. PubMed PMID: 17174725.
8. Gaydukova I, Rebrov A, Nikitina N, Poddubnyy D. Decreased heart rate variability in patients with psoriatic arthritis. Clinical rheumatology. 2012;31(9):1377-81. doi: 10.1007/s10067-012-2015-3. PubMed PMID: 22673792.
9. Arisoy A, Karaman K, Karayakali M, Demirelli S, Seckin HY, Celik A, et al. Evaluation of ventricular repolarization features with novel electrocardiographic parameters (Tp-e, Tp-e/QT) in patients with psoriasis. Anatol J Cardiol. 2017;18(6):397-401. Epub 2017/12/20. doi: 10.14744/AnatolJCardiol.2017.7901. PubMed PMID: 29256874; PubMed Central PMCID: PMCPMC6282903.
10. Taylor W, Gladman D, Helliwell P, Marchesoni A, Mease P, Mielants H, et al. Classification criteria for psoriatic arthritis: development of new criteria from a large international study. Arthritis Rheum. 2006;54(8):2665-73. Epub 2006/07/28. doi: 10.1002/art.21972. PubMed PMID: 16871531.
11. Kibari A, Cohen AD, Gazitt T, Bitterman H, Lavi I, Feldhamer I, et al. Cardiac and cardiovascular morbidities in patients with psoriatic arthritis: a population-based case control study. Clinical rheumatology. 2019;38(8):2069-75. Epub 2019/04/03. doi: 10.1007/s10067-019-04528-y. PubMed PMID: 30937638.
12. Tam LS, Tomlinson B, Chu TT, Li M, Leung YY, Kwok LW, et al. Cardiovascular risk profile of patients with psoriatic arthritis compared to controls--the role of inflammation. Rheumatology (Oxford). 2008;47(5):718-23. Epub 2008/04/11. doi: 10.1093/rheumatology/ken090. PubMed PMID: 18400833.
13. Han C, Robinson DW, Jr., Hackett MV, Paramore LC, Fraeman KH, Bala MV. Cardiovascular disease and risk factors in patients with rheumatoid arthritis, psoriatic arthritis, and ankylosing spondylitis. J Rheumatol. 2006;33(11):2167-72. Epub 2006/09/19. PubMed PMID: 16981296.
14. Chiu HY, Chang WL, Huang WF, Wen YW, Tsai YW, Tsai TF. Increased risk of arrhythmia in patients with psoriatic disease: A nationwide population-based matched cohort study. J Am Acad Dermatol. 2015;73(3):429-38. Epub 2015/07/21. doi: 10.1016/j.jaad.2015.06.023. PubMed PMID: 26188627.
15. Rautaharju PM. A farewell to QT dispersion. Are the alternatives any better? J Electrocardiol. 2005;38(1):7-9. Epub 2005/01/22. doi: 10.1016/j.jelectrocard.2004.09.017. PubMed PMID: 15660341.
16. 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(6):567-74. Epub 2008/09/16. doi: 10.1016/j.jelectrocard.2008.07.016. PubMed PMID: 18790499.
17. Kors JA, Ritsema van Eck HJ, van Herpen G. The meaning of the Tp-Te interval and its diagnostic value. J Electrocardiol. 2008;41(6):575-80. Epub 2008/10/29. doi: 10.1016/j.jelectrocard.2008.07.030. PubMed PMID: 18954608.
18. 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(3):301-8. Epub 2011/04/23. doi: 10.1016/j.jelectrocard.2011.03.004. PubMed PMID: 21511064.
19. Rosenthal TM, Masvidal D, Abi Samra FM, Bernard ML, Khatib S, Polin GM, et al. Optimal method of measuring the T-peak to T-end interval for risk stratification in primary prevention. Europace. 2018;20(4):698-705. Epub 2017/03/25. doi: 10.1093/europace/euw430. PubMed PMID: 28339886.
20. Erikssen G, Liestol 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(2):85-94. Epub 2012/04/28. doi: 10.1111/j.1542-474X.2012.00493.x. PubMed PMID: 22537325; PubMed Central PMCID: PMCPMC6932507.
21. Xia Y, Liang Y, Kongstad O, Liao Q, Holm M, Olsson B, et al. In vivo validation of the coincidence of the peak and end of the T wave with full repolarization of the epicardium and endocardium in swine. Heart Rhythm. 2005;2(2):162-9. Epub 2005/04/27. doi: 10.1016/j.hrthm.2004.11.011. PubMed PMID: 15851290.
22. Hidayet S, Demir V, Turan Y, Gurel G, Tasolar MH. Evaluation of Tp-e interval, Tp-e/QT ratio, and Tp-e/QTc ratio in patients with Behcet's disease. Anatol J Cardiol. 2019;22(2):85-90. Epub 2019/08/04. doi: 10.14744/AnatolJCardiol.2019.70019. PubMed PMID: 31375650; PubMed Central PMCID: PMCPMC6735444.
23. Lazzerini PE, Capecchi PL, Acampa M, Galeazzi M, Laghi-Pasini F. Arrhythmic risk in rheumatoid arthritis: the driving role of systemic inflammation. Autoimmun Rev. 2014;13(9):936-44. Epub 2014/05/31. doi: 10.1016/j.autrev.2014.05.007. PubMed PMID: 24874445.
24. Sgreccia A, Morelli S, Ferrante L, Perrone C, De Marzio P, De Vincentiis G, et al. QT interval and QT dispersion in systemic sclerosis (scleroderma). J Intern Med. 1998;243(2):127-32. Epub 1998/05/05. PubMed PMID: 9566641.
25. Guo Y, Lip GY, Apostolakis S. Inflammation in atrial fibrillation. J Am Coll Cardiol. 2012;60(22):2263-70. Epub 2012/12/01. doi: 10.1016/j.jacc.2012.04.063. PubMed PMID: 23194937.
26. Kazumi T, Kawaguchi A, Hirano T, Yoshino G. C-reactive protein in young, apparently healthy men: associations with serum leptin, QTc interval, and high-density lipoprotein-cholesterol. Metabolism. 2003;52(9):1113-6. Epub 2003/09/25. doi: 10.1016/s0026-0495(03)00184-7. PubMed PMID: 14506615.
27. Kim E, Joo S, Kim J, Ahn J, Kim J, Kimm K, et al. Association between C-reactive protein and QTc interval in middle-aged men and women. Eur J Epidemiol. 2006;21(9):653-9. Epub 2006/10/31. doi: 10.1007/s10654-006-9034-9. PubMed PMID: 17072542.
28. Lazzerini PE, Acampa M, Capecchi PL, Hammoud M, Maffei S, Bisogno S, et al. Association between high sensitivity C-reactive protein, heart rate variability and corrected QT interval in patients with chronic inflammatory arthritis. Eur J Intern Med. 2013;24(4):368-74. Epub 2013/03/23. doi: 10.1016/j.ejim.2013.02.009. PubMed PMID: 23517852.
29. Yayla C, Yayla ME, Yayla KG, Ilgen U, Akboga MK, Duzgun N. The Assessment of Tp-e Interval and Tp-e/QT Ratio in Patients With Systemic Sclerosis. Arch Rheumatol. 2016;31(2):139-44. Epub 2016/01/28. doi: 10.5606/ArchRheumatol.2016.5753. PubMed PMID: 29900933; PubMed Central PMCID: PMCPMC5827829.
30. Demirtas K, Yayla C, Yuksel M, Acar B, Unal S, Ertem AG, et al. Tp-e interval and Tp-e/QT ratio in patients with celiac disease. Rev Clin Esp. 2017;217(8):439-45. Epub 2017/10/11. doi: 10.1016/j.rce.2017.09.001. PubMed PMID: 28992960.
31. Sivri S, Çelik M . Effect of anti-TNFα treatment on Tp-e interval and Tp-e/QT ratio in patients with ankylosing spondylitis: A case-control study. J Surg Med. 2020;4(3):182-5.
32. Osto E, Piaserico S, Maddalozzo A, Forchetti G, Montisci R, Famoso G, et al. Impaired coronary flow reserve in young patients affected by severe psoriasis. Atherosclerosis. 2012;221(1):113-7. Epub 2012/01/13. doi: 10.1016/j.atherosclerosis.2011.12.015. PubMed PMID: 22236480.
33. Balci DD, Balci A, Karazincir S, Ucar E, Iyigun U, Yalcin F, et al. Increased carotid artery intima-media thickness and impaired endothelial function in psoriasis. Journal of the European Academy of Dermatology and Venereology : JEADV. 2009;23(1):1-6. Epub 2008/08/16. doi: 10.1111/j.1468-3083.2008.02936.x. PubMed PMID: 18702627.
34. Yiu KH, Yeung CK, Zhao CT, Chan JC, Siu CW, Tam S, et al. Prevalence and extent of subclinical atherosclerosis in patients with psoriasis. J Intern Med. 2013;273(3):273-82. Epub 2012/09/26. doi: 10.1111/joim.12002. PubMed PMID: 23003220.
35. Gonzalez-Juanatey C, Llorca J, Amigo-Diaz E, Dierssen T, Martin J, Gonzalez-Gay MA. High prevalence of subclinical atherosclerosis in psoriatic arthritis patients without clinically evident cardiovascular disease or classic atherosclerosis risk factors. Arthritis Rheum. 2007;57(6):1074-80. Epub 2007/08/01. doi: 10.1002/art.22884. PubMed PMID: 17665475.
36. Gladman DD, Ang M, Su L, Tom BD, Schentag CT, Farewell VT. Cardiovascular morbidity in psoriatic arthritis. Annals of the rheumatic diseases. 2009;68(7):1131-5. Epub 2008/08/14. doi: 10.1136/ard.2008.094839. PubMed PMID: 18697777.
37. Ahlehoff O, Gislason GH, Charlot M, Jorgensen CH, Lindhardsen J, Olesen JB, et al. Psoriasis is associated with clinically significant cardiovascular risk: a Danish nationwide cohort study. J Intern Med. 2011;270(2):147-57. Epub 2010/12/01. doi: 10.1111/j.1365-2796.2010.02310.x. PubMed PMID: 21114692.
38. Saba S, Janczewski AM, Baker LC, Shusterman V, Gursoy EC, Feldman AM, et al. Atrial contractile dysfunction, fibrosis, and arrhythmias in a mouse model of cardiomyopathy secondary to cardiac-specific overexpression of tumor necrosis factor-{alpha}. Am J Physiol Heart Circ Physiol. 2005;289(4):H1456-67. Epub 2005/06/01. doi: 10.1152/ajpheart.00733.2004. PubMed PMID: 15923312.
39. Dave J, Ahlman MA, Lockshin BN, Bluemke DA, Mehta NN. Vascular inflammation in psoriasis localizes to the arterial wall using a novel imaging technique. J Am Acad Dermatol. 2014;70(6):1137-8. Epub 2014/03/20. doi: 10.1016/j.jaad.2013.12.043. PubMed PMID: 24641984; PubMed Central PMCID: PMCPMC4024331.

This paper has been checked for language accuracy by JOSAM editors.
The National Library of Medicine (NLM) citation style guide has been used in this paper.