# The Relationship Between the Presence of Early Repolarization Pattern and the Severity of Coronary Artery Disease in Patients with Stable Coronary Artery Disease

## Stabil Koroner Arter Rahatsızlığı Olan Hastalarda Erken Repolarizasyon Patern Varlığı ile Koroner Arter Hastalığının Ciddiyeti Arasındaki İlişki

**Serhat Karaman[1], Kayıhan Karaman[2], Abuzer Coşkun[3]**

[1] *Department of Emergency Medicine, Gaziosmanpasa University Faculty of Medicine, Tokat, Turkey*
[2] *Department of Cardiology, Gaziosmanpasa University Faculty of Medicine, Tokat, Turkey*
[3] *Department of Emergency Medicine, Sivas State Hospital, Sivas, Turkey*

Yazışma Adresi / *Correspondence:*
**Serhat Karaman**

Gaziosmanpaşa Üniversitesi Tıp Fakültesi, Acil Tıp Ana Bilim Dalı, Tokat 60100, Tokat, Türkiye
T: **+90 505 400 1506** E-mail: **drserhatkaraman@hotmail.com**

Geliş Tarihi / Received : **09.02.2019** Kabul Tarihi / Accepted : **26.02.2019**

**Abstract**

*Objective* Although early repolarization pattern (ERP) has been recognized as a benign variant, in recent studies have shown that it is associated with life-threatening arrhythmias. There are different studies demonstrated that there is a strong relationship between life-threatening arrhythmias and the severity of coronary artery disease (CAD). In this study, we aimed to determine whether Gensini Score (GS) values are higher in patients with ERP.. **( Sakarya Med J 2019, 9(1):112-119 )**

*Materials and Methods* Totally 110 patients with ERP and 90 control subjects were enrolled to the study. GS was calculated for each patient. p values < 0.05 were considered significant

*Results* GS was significantly higher in the ERP patients compared with the control group (19.5±20.2 vs 2.15±4.1; p<0.001). There was significant correlation between GS and age (r=0.507, p<0.001). GS was an independent predictor of ERP in multivariate logistic regression analysis (OR=0.655, 95% CI=0.557-0.769, p<0.001). In addition to, age and low-density lipoprotein cholesterol were independent predictors associated with GS in linear regression analysis (OR=0.370, 95% CI=0.085-0.655, p=0.011 and OR= -0.094, 95% CI= (-0.176-0.013), p=0.024, respectively).

*Conclusions* An increase in the severity of CAD may be shown as a mechanism that leads to the emergence of ERP.

*Keywords* Early repolarization pattern; Gensini score; Stable Coronary Artery Disease

**Öz**

*Amaç* *Erken repolarizasyon paterni (ERP) iyi huylu bir varyant olarak kabul edilmesine rağmen, son zamanlarda yapılan çalışmalarda hayatı tehdit eden aritmilerle ilişkili olduğu gösterilmiştir. Hayatı tehdit eden aritmi ve koroner arter hastalığının (KAH) şiddeti arasında güçlü bir ilişki olduğunu gösteren farklı çalışmalar vardır. Bu çalışmada, ERP'li hastalarda Gensini Skoru (GS) değerlerinin daha yüksek olup olmadığını belirlemeyi amaçladık.* ***( Sakarya Tıp Dergisi 2019, 9(1):112-119 )***

*Gereç ve Yöntemler* *Çalışmaya ERP'li toplam 110 hasta ve 90 kontrol grubu alındı. Her hasta için GS hesaplandı. p< 0.05 anlamlı kabul edildi.*

*Bulgular* *GS, ERP'li hastalarda kontrol grubuna göre anlamlı derecede yüksekti (19.5±20.2 ve 2.15±4.1; p <0.001). GS ile yaş arasında anlamlı ilişki vardı (r= 0.507, p< 0.001). GS, çok değişkenli lojistik regresyon analizinde bağımsız bir ERP belirleyicisidir (OR= 0.655,% 95 CI= 0.557-0.769, p <0.001). Ek olarak, yaş ve düşük yoğunluklu lipoprotein kolesterolü, lineer regresyon analizinde GS ile ilişkili bağımsız öngörücülerdi (OR= 0.370,%95 CI= 0.085-0.655, p= 0.011 ve OR= -0.094,%95 CI= (-0.176 -0.013), sırasıyla p= 0.024).*

*Sonuçlar* *KAH'nın ciddiyetindeki artış, ERP'nin ortaya çıkmasına yol açan bir mekanizma olarak gösterilebilir.*

*Anahtar Kelimeler* *Erken repolarizasyon patern; Gensini skoru; Stabil Koroner Arter Hastalığı*

## INTRODUCTION

Early repolarization pattern (ERP) is characterized by a J-point (between the end of the QRS complex and the beginning of the ST segment) elevation of at least one mm in two contiguous leads with a "notching" type appearance or a "slurring" in the inferior, lateral, or inferolateral leads on a surface electrocardiogram (ECG).[1] It has been recognized as a benign variant, especially in healthy young male subjects, although recent studies have shown that ERP is associated with an increased risk of cardiovascular death and sudden cardiac death (SCD).[2-5] However, few studies have investigated the relationship between atherosclerotic heart disease and ERP. These studies have investigated the association between ERP and the risk of ventricular tachycardia (VT)/ventricular fibrillation (VF) and SCD in patients with acute coronary syndrome and have suggested that ERP might increase the risk of VT/VF.[6,7]

Different studies confirmed a strong and consistent association between life-threatening arrhythmias and coronary artery disease (CAD).[8,9] Gensini Score (GS) is used to determine the extent and severity of CAD. GS is a scoring system which is primarily calculated based on the involved artery, the extent of atherosclerosis, and the existence of collateral.[10] The aim of this study was to determine whether the GS value is higher in patients with ERP compared patients without ERP.

## MATERIALS and METHODS

### Study Design

The present study is a cross-sectional descriptive study.

### Study Population

A total of 200 patients with stable angina pectoris (110 patients with ERP [55 men; mean age, 60.0±8.0 years] and 90 patients without ERP in the control group [41 men; mean age, 58.4±8.9]) who were admitted to the cardiology clinic and emergency services department from March 2012 to December 2017 were included in the study. The patients were included in the study when they presented to the cardiology clinic for the first-line control. All patients recruited for the study had objective signs of ischemia (treadmill exercise or myocardial single-photon emission computed tomography [SPECT]) and were referred for coronary angiography. Patients presenting with acute myocardial infarction, a history of PCI or coronary artery bypass grafting, end-stage renal disease, cardiomyopathies, moderate to severe valvular heart diseases, heart failure, atrial fibrillation, complete right or left bundle branch block, channelopathies, including long QT syndrome, Brugada Syndrome, and malignancy were excluded from the study. Patients taking medications known to have effect on QT interval, such as tricyclic antidepressants, β-blockers, calcium channel blockers or antiarrhythmics were also excluded. Patients with hypertension were under the treatment of angiotensin converting enzyme inhibitors or angiotensin receptor blockers. Approval was obtained from the local ethics committee for this study, and complied with the Declaration of Helsinki.

### Electrocardiographic and Echocardiographic Examination

Twelve-lead ECGs were obtained from all subjects in a supine position at a paper speed of 25 mm/sec and a calibration of 10 mm/mV. ECGs were obtained by the same investigator using the same ECG recorder (Nihon Kohden ECG-9020K, Japan). Baseline ECGs were simultaneously evaluated in random order by two experienced cardiologists for the presence of ERP. ERP was defined as elevation of the J point (ie, the junction of the QRS complex and ST-segment) by 0.1 mV above the baseline with either QRS slurring or notching in the inferior leads (II, III, and aVF), the lateral leads (I, aVL, and V4 to V6), or both.

All echocardiography examinations (General Electric Vivid S5, Milwaukee, WI, USA) were performed by an experienced cardiologist in all subjects using a 2.5–3.5 MHz transducer in the left decubitus position. Left ventricular ejection fraction (LVEF) was assessed using Simpson's method.

### Coronary Angiography and Severity and Extent of Coronary Artery Disease

All patients underwent selective right and left coronary angiography through the right femoral artery using the standard Judkins technique with a MEGALIX Cat plus 125/40/90-121 GW model angiography device (Siemens Artis Zee, Forchheim, Germany). Iohexol 350/100 was used as a contrast agent, and approximately 6–8 ml of the contrast medium was injected manually for each exposure. The coronary arteries were imaged in the right and left oblique position using cranial and caudal angulations. The severity and extent of coronary atherosclerosis in patients was assessed using the GS, which grades the narrowing of the lumen of the coronary arteries as 1 for 1%-25% narrowing, 2 for 26%-50% narrowing, 4 for 51%-75% narrowing, 8 for 76%-90% narrowing, 16 for 91%-99% narrowing, and 32 for total occlusion. This score is then multiplied by a factor that takes into account the importance of the lesion's position in the coronary arterial tree; for example: 5 for the left main coronary artery, 2.5 for the proximal left anterior descending (LAD) coronary artery or proximal left circumflex (LCX) coronary artery, 1.5 for the mid-region of LAD, and 1 for the distal LAD or mid-distal region of the LCX. The GS was expressed as the total of the scores for all coronary arteries.

### Statistical Analysis

All tests were performed by using PASW Statistics (SPSS 18.0 for Windows, Inc., Chicago, IL, USA). Continuous variables were expressed as mean±standard deviation, and the qualitative variables were expressed as a percentage or ratio. Compliance of the variables with normal distribution was assessed using the Kolmogorow-Smirnov test. Continuous variables were compared between groups using either the Student's t-test or the Mann–Whitney U test, depending on the variable's compliance with the normal distribution. For the qualitative variables, the chi-square test was used. Pearson correlation test was used for exploring the correlation state of some variables. Multivariate linear regression analysis was used to analyze independent predictors of GS. Multivariate logistic regression analysis was used to identify the independent predictors of ERP and independent variables that differed significantly in the univariate analyses ($p < 0.1$). A p value of less than 0.05 was considered statistically significant.

## RESULTS

Demographic and other characteristics of the ERP and control groups are shown in Table 1. There was no statistically significant difference between the ERP and the control groups with respect to age, gender, glucose, creatinine,

**Table-1: Baseline characteristics, laboratory and echocardiographic parameters of the study groups**

| | | | |
|---|---|---|---|
| Age, years | 60.2±8.1 | 58.8±8.9 | 0.225 |
| Male, n (%) | 69 (62.7) | 42 (46.7) | 0.059 |
| Systolic BP, mmHg | 110.2±11.4 | 111.±9.4 | 0.330 |
| Creatinin, mg/dL | 0.88±0.39 | 0.88±0.35 | 0.868 |
| Glucose, mg/dL | 92.8±9.4 | 90.1±8.4 | 0.451 |
| Total cholesterol, mg/dL | 168.2±66.7 | 174±50.9 | 0.462 |
| LDL cholesterol, mg/dL | 100.2±40.4 | 106.2±36.0 | 0.269 |
| LVEF, (%) | 63.5±4.7 | 63.3±5.2 | 0.458 |
| Magnesium, mg/dL | 2.18±0.43 | 2.12±0.38 | 0.329 |
| Calcium, mg/dL | 9.28±0.65 | 9.17±0.53 | 0.198 |
| Sodium, mmol/L | 138.7±4.1 | 138.9±4.2 | 0.716 |
| Potassium, mmol/L | 4.52±0.54 | 4.38±0.60 | 0.084 |
| Gensini score | 19.5±20.2 | 2.15±4.1 | <0.001 |

BP: blood pressure; LDL: low-density lipoprotein; LVEF: left ventricular ejection fraction. Numerical variables with a normal distribution were presented as mean±standard deviation, or n (%).

LVEF, total cholesterol, and low-density lipoprotein cholesterol values. In addition, there was no significant difference between the groups in terms of electrolytes, such as sodium, potassium, calcium, and magnesium (Table 1). GS was significantly higher in the ERP patients compared with the control group (19.5±20.2 vs 2.15±4.1; p<0.001) (Fig. 1). Pearson's correlation test was used to determine the variables associated with the GS. Pearson's correlation analysis revealed positive relationship between GS and age (r=0.507, p<0.001), male sex (r=0.215, p=0.002). However, there was no correlation among GS, systolic blood pressure, serum glucose, serum creatinin, total cholesterol, and LDL cholesterol.

Univariate and multivariate logistic regression analyses for the predictors of ERP in the study population are presented in Table 2. In the univariate analysis, potassium level and GS were associated with ERP. Variables with a p value less than 0.1 in univariate analysis were included in the multivariate model. Multivariate logistic regression analysis demonstrated that the GS was an independent predictor of ERP (OR=0.654, 95% CI=0.556-0.769, p<0.001). In addition to, age and LDL cholesterol were independent predictors associated with GS in linear regression analysis (OR= 0.370, 95% CI=0.085-0.655, p=0.011 and OR= -0.094, 95% CI= (-0.176-0.013), p=0.024, respectively) (Table 3).

**Table-2: Univariate and multivariate analysis for the predictors of ERP**

| | Univariate analysis | | Multivariate analysis* | |
|---|---|---|---|---|
| | OR (95% CI) | p | OR (95% CI) | p |
| Age | 0.980 (0.948-1.013) | 0.224 | - | - |
| Magnesium | 0.709 (0.356-1.412) | 0.328 | - | - |
| Calcium | 0.735 (0.460-1.174) | 0.198 | - | - |
| Sodium | 1.013 (0.946-1.084) | 0.714 | - | - |
| Potassium | 0.649 (0.396-1.063) | 0.086 | 0.729 (0.385-1.380) | 0.331 |
| Gensini score | 0.655 (0.557–0.769) | <0.001 | 0.654 (0.556-0.769) | <0.001 |

CI: confidence interval; ERP: early repolarization pattern; OR: odds ratio.
*Variables with a p < 0.1 in univariate analysis were included in the multivariate model.

**Table 3: Linear regression analysis for the predictors of Gensini score**

| | Linear regression analysis | |
|---|---|---|
| | OR (95% CI) | p |
| Male sex | 3.370 (-1,543-8.282) | 0.178 |
| Age | 0.370 (0.085-0.655) | 0.011 |
| Total cholesterol | 0.040 (-0.019-0.100) | 0.183 |
| LDL cholesterol | -0.094 (-0.176-0.013) | 0.024 |

CI: confidence interval; LDL: low-density lipoprotein; OR: odds ratio.

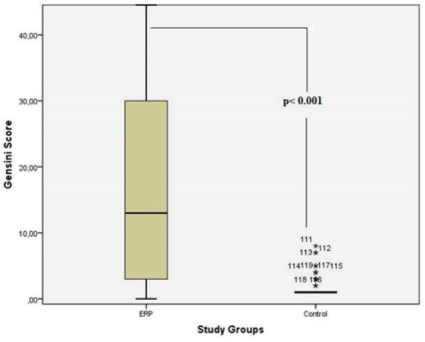


Figure 1: Mean Gensini values between the study groups

## DISCUSSION

In the present study, it was demonstrated that the GS is increased in patients with ERP when compared to those in subjects without ERP. In addition, GS was an independent predictor of ERP. Thus, these findings suggest that an increase in the GS might worsen the stage of myocardial repolarization.

ERP is characterized by a prominent elevation of the J-point between the end of the QRS complex and the beginning of the ST segment on surface ECG.1 ERP has a range of 1-13% in the general population, depending on age (predominant in young adults), race (predominant in black populations), and sex (predominant in males).[11] Although ERP is considered a benign ECG finding, current studies have shown that it increases the risk of ventricular arrhythmia and SCD.[12-14] The underlying pathophysiologic mechanism of ERP remains elusive and most likely has a complex structure. Experimental studies suggest that J-point elevation indicates increased transmural distribution of myocardial repolarization which contributes a susceptibility to ventricular arrhythmias.[15] The second possible mechanism is the dysregulation of the autonomic nervous system, and autonomic tonus may play an important role in the formation of VTs associated with ERP. The reason for this is that many events have been shown to occur during an increased vagal tone, such as while sleeping or post-meal.[16]

Under certain conditions known to affect the distribution of myocardial repolarization, such as ischemic heart disease, patients with ERP may be at an increased risk of arrhythmia.[17] Atherosclerosis is a systemic disease associated with inflammation, and there is a strong relationship between atherosclerosis and endothelial dysfunction. Endothelial dysfunction may lead to fluctuations in microvascular resistance. Atherosclerotic CAD caused by endothelial dysfunction may lead to ischemia and, in turn, to circulatory disturbances at the microvascular level during the ongoing process.[18,19] Myocardial repolarization parameters may be affected by ischemia as a result of the extent and severity of CAD.8 The GS was established to expose the severity and extent of coronary atherosclerosis. Many studies have demonstrated the relationship between myocardial repolarization and coronary atherosclerosis; however, to our knowledge, there are not yet sufficient data about the relationship between ERP and the severity of coronary atherosclerosis.

The QT interval encompasses both depolarization and repolarization, whereas the Tpeak and Tend (Tp-e) interval represents transmural dispersion of myocardial repolarization.[20,21] Previous studies have investigated the relationship between CAD and myocardial repolarization. It has been shown that a prolonged QT dispersion (QTd) is associated with an increased risk of dangerous ventricular arrhythmias as in patients with CAD.[22] Goodhart et al.[23] found that revascularization in chronic total occlusion (CTO) resulted in a decrease in QTd, which was sustained at six months and also associated with improved global left ventricular function. On the other hand, in a study by Cetin et al.[9], it was found that the Tp-e interval decreased after successful percutaneous CTO revascularization. These studies suggest that patients with increased CAD severity have an increased electrical imbalance in their myocardium.

J waves, including ERP, are associated with the occurrence of lethal arrhythmic events in patients with Brugada syndrome, ischemic heart disease, and hypertrophic cardiomyopathy.[24,25] Recently, some researchers have investigated the relationship between ERP and cardiovascular events. Tikkanen et al.[26] have shown that patients with ERP have a higher risk of having ischemic events and ischemic VF. In another study, Rudic et al.[6] have suggested that ERP seems to be associated with VTs in the setting of acute myocardial infarction. In addition to this information, ERP may point to peri-infarction block, which would indicate latent ischemic heart disease. Therefore, ERP should prompt

clinicians to consider latent ischemic heart disease before attributing the J-point elevation to a primary electrical abnormality.[27] However, there is currently no study investigating the relationship between CAD severity and ERP.

### Study Limitations

Our study had some limitations. First, our study was single centered and included a small number of patients. Therefore, statistical power was limited. The results should be verified in a larger prospective cohort study. Second, because we did not have other ambulatory Holter measures, such as heart rate variability and heart rate turbulence, we could not investigate the relationship between the autonomic nervous system and ERP. Third, we did not have data on cardiac event rates for this study because we could not follow the study population for prospective arrhythmic events. Further comprehensive studies should be conducted with a larger number of patients and a longer follow-up time to increase the consistency of our results.

### Conclusion

The increased severity of CAD is associated with an increased risk of ventricular arrhythmias. An increase in the severity of CAD may be indicative of a mechanism that leads to the formation of ERP by causing deterioration in the distribution of ventricular repolarization.

Potential Conflict of Interest
No potential conflict of interest relevant to this article was reported.
Sources of Funding
There were no external funding sources for this study.
Study Association
This study is not associated with any thesis or dissertation work.

## References

1. *Macfarlane PW, Antzelevitch C, Haissaguerre M, Huikuri HV, Potse M, Rosso R, et al. The early repolarization pattern: A Consensus Paper. J Am Coll Cardiol. 2015;66:470-477.*
2. *Klatsky AL, Oehm R, Cooper RA, Udaltsova N, Armstrong MA. The early repolarization normal variant electrocardiogram: Correlates and consequences. Am J Med. 2003;115:171-177.*
3. *Haïssaguerre M, Derval N, Sacher F, Jesel L, Deisenhofer I, de Roy L, et al. Sudden cardiac arrest associated with early repolarization. N Engl J Med. 2008;358:2016-2023.*
4. *Rosso R, Kogan E, Belhassen B, Rozovski U, Scheinman MM, Zeltser D, et al. J-point elevation in survivors of primary ventricular fibrillation and matched control subjects: Incidence and clinical signifcance. J Am Coll Cardiol. 2008;52:1231-1238.*
5. *Patel RB, Ilkhanoff L, Ng J, Chokshi M, Mouchli A, Chacko SJ, et al. Clinical characteristics and prevalence of early repolarization associated with ventricular arrhythmias following acute ST elevation myocardial infarction. Am J Cardiol. 2012;110:615-620.*
6. *Rudic B, Veltmann C, Kuntz E, Behnes M, Elmas E, Konrad T, et al. Early repolarization pattern is associated with ventricular fibrillation in patients with acute myocardial infarction. Heart Rhythm. 2012;9:1295-1300.*
7. *Ali Diab O, Abdel-Hafez Allam RM, Mohamed HG, Mohamed TR, Abel-Hafeez Khalid S. Early repolarization pattern is associated with increased risk of early ventricular arrhythmias during acute ST segment elevation myocardial infarction. Ann Noninvasive Electrocardiol. 2015;20:474-480.*
8. *Taşolar H, Ballı M, Çetin M, Otlu YÖ, Altun B, Bayramoğlu A. Effects of the coronary collateral circulation on the Tp-e interval and Tp-e/QT ratio in patients with stable coronary artery disease. Ann Noninvasive Electrocardiol. 2015;20(1):53-61.*
9. *Cetin M, Zencir C, Cakici M, Yildiz E, Tasolar H, Balli M, et al. Effect of a successful percutaneous coronary intervention for chronic total occlusion on parameters of ventricular repolarization. Coron Artery Dis. 2014;25(8):705-712.*
10. *Gensini GG. The coronary artery disease scoring and retrieval system of Gensini. In: Gensini GG, Editor. Coronary arteriography. New York, NY: Futura Pub. Co.;1975. p.271-274.*
11. *Miyazaki S, Shah AJ, Haïssaguerre M. Early repolarization syndrome – a new electrical disorder associated with sudden cardiac death –. Circ J. 2010;74(10):2039-2044.*
12. *Gussak I, Antzelevitch C. Early repolarization syndrome: clinical characteristics and possible cellular and ionic mechanisms. J Electrocardiol. 2000;33:299-309.*
13. *Shu J, Zhu T, Yang L, Cui C, Yan GX. ST-segment elevation in the early repolarization syndrome, idiopathic ventricular fibrillation, and the Brugada syndrome: cellular and clinical linkage. J Electrocardiol. 2005;38:26-32.*
14. *Furukawa Y, Yamada T, Morita T, Iwasaki Y, Kawasaki M, Kikuchi A, et al. Early repolarization pattern associated with sudden cardiac death: long-term follow up in patients with chronic heart failure. J Cardiovasc Electrophysiol. 2013;24:632-639.*
15. *Benito B, Guasch E, Rivard L, Nattel S. Clinical and mechanistic issues in early repolarization of normal variants and lethal arrhythmia syndromes. J Am Coll Cardiol. 2010;56:1177-1186.*
16. *Takagi M, Aihara N, Takaki H, Taguchi A, Shimizu W, Kurita T, et al. Clinical characteristics of patients with spontaneous or inducible ventricular fibrillation without apparent heart disease presenting with J wave and ST segment elevation in inferior leads. J Cardiovasc Electrophysiol. 2000;11:844-848.*
17. *Naruse Y, Tada H, Harimura Y, Hayashi M, Noguchi Y, Sato A, et al. Early repolarization is an independent predictor of occurrences of ventricular fibrillation in the very early phase of acute myocardial infarction. Circ Arrhythm Electrophysiol. 2012;5:506-513.*
18. *Thenappan T, Ali Raza J, Movahed A. Aortic atheromas: current concepts and controversies-a review of the literature. Echocardiography. 2008;25:198-207.*
19. *Laurent S, Cockcroft J, Van Bortel L, et al. Expert consensus document on arterial stiffness: methodological issues and clinical applications. Eur Heart J. 2006;27:2588-2605.*
20. *Antzelevitch C. T peak-T end interval as an index of transmural dispersion of repolarization. Eur J Clin Invest. 2001;31:555-557.*
21. *Karaman K, Karayakali M, Arisoy A, Akar I, Ozturk M, Yanik A, et al. Is There any Relationship Between Myocardial Repolarization Parameters and the Frequency of Ventricular Premature Contractions? Arq Bras Cardiol. 2018;110(6):534-541.*
22. *Zareba W, Moss AC, le Cessie S. Dispersion of ventricular repolarization and arrhythmic cardiac death in coronary artery disease. Am J Cardiol. 1994;74:550-553.*
23. *Goodhart DM, Hubacek J, Anderson TJ, Duff H, Barbeau G, Ducas J, et al. TOSCA investigators. Effect of percutaneous coronary intervention of nonacute total coronary artery occlusions on QT dispersion. Am Heart J. 2006;151:529.e1-529.e6.*
24. *Antzelevitch C, Yan GX, Ackerman MJ, Borggrefe M, Corrado D, Guo J, et al. J-wave syndromes expert consensus conference report: emerging concepts and gaps in knowledge. J Arrhythm. 2016;32(5):315-339.*
25. *Tsuda T, Hayashi K, Konno T, Sakata K, Fujita T, Hodatsu A, et al. J waves for predicting cardiac events in hypertrophic cardiomyopathy. JACC Clin Electrophysiol. 2017;3(10):1136-1142.*
26. *Tikkanen JT, Wichmann V, Junttila MJ, Rainio M, Hookana E, Lappi OP, et al. Association of early repolarization and sudden cardiac death during an acute coronary event. Circ Arrhythm Electrophysiol. 2012;5:714¬-718.*
27. *Antzelevitch C, Yan GX. J wave syndromes. Heart Rhythm. 2010;7:549-558.*