Turkish Journal of Thoracic and Cardiovascular Surgery 2018;26(1):65-72
http://dx.doi.org/doi: 10.5606/tgkdc.dergisi.2018.14819

Original Article / Özgün Makale

The effect of transcatheter aortic valve implantation on Tp-e interval, Tp-e/QT and Tp-e/QTc ratios, and Tp-e dispersion in patients with severe aortic stenosis

Şiddetli aort darlığı olan hastalarda transkateter aort kapak implantasyonunun Tp-e aralığı, Tp-e/QT ile Tp-e/QTc oranları ve Tp-e dispersiyonu üzerine etkisi

Zülkif Tanrıverdi[1], Tuğçe Çöllüoğlu[2], Barış Ünal[3], Hüseyin Dursun[2], Dayimi Kaya[2]

Institution where the research was done:
Dokuz Eylül University, Faculty of Medicine, İzmir, Turkey

Author Affiliations:
[1]Department of Cardiology, Balıklıgöl State Hospital, Şanlıurfa, Turkey
[2]Department of Cardiology, Dokuz Eylül University, Faculty of Medicine, İzmir, Turkey
[3]Department of Cardiology, Cumra State Hospital, Konya, Turkey

ABSTRACT

Background: In this study, we aimed to investigate the effect of transcatheter aortic valve implantation using two types of bioprosthetic valves on novel ventricular repolarization markers including Tp-e, Tp-e/QT and Tpe/QTc ratios, and Tp-ed.

Methods: A total of 61 patients (17 males, 44 females; mean age 78.6±6.5 years; range 55 to 89 years) who underwent transcatheter aortic valve implantation with either a Medtronic CoreValve (n=40) or an Edwards SAPIEN XT valve (n=21) were retrospectively analyzed. The electrocardiographic parameters and left ventricular mass index were calculated prior to the procedure, on postoperative Day 1, and at three months after the procedure.

Results: The Tp-e interval, Tp-e/QT and Tp-e/QTc ratios, Tp-ed, and left ventricular mass index significantly reduced at three months of the procedure, compared to baseline values ($p<0.01$, for all). Similar findings were observed for QT, QTc, and QT dispersion ($p<0.01$, for all). These changes were independent from the types of bioprosthetic valves used. Before the procedure, the left ventricular mass index was positively correlated with the Tp-e ($r=0.350$, $p=0.007$), Tp-e/QT ($r=0.314$, $p=0.015$) and Tp-e/QTc ratios ($r=0.285$, $p=0.029$). In the multivariate analysis, Tp-e interval was found to be independently associated with the left ventricular mass index ($\beta=0.350$, $p=0.007$).

Conclusion: In the present study, the Tp-e interval, Tp-e/QT and Tp-e/QTc ratios, Tp-ed, and left ventricular mass index significantly reduced at three months after transcatheter aortic valve implantation indicating reverse left ventricular remodeling. The effects of two types of bioprosthetic valves on ventricular repolarization markers and left ventricular mass index were similar.

Keywords: Aortic valve stenosis; electrocardiography; heart valve prosthesis; ventricular repolarization markers.

ÖZ

Amaç: Bu çalışmada iki farklı biyoprotez kapak ile yapılan transkateter aort kapak implantasyonunun Tp-e, Tp-e/QT ve Tpe/QTc oranları ve Tp-ed gibi yeni ventriküler repolarizasyon belirteçleri üzerindeki etkisi incelendi.

Çalışma planı: Medtronic CoreValve (n=40) veya Edwards SAPIEN XT (n=21) kapak ile transkateter aort kapak implantasyonu yapılan toplam 61 hasta (17 erkek, 44 kadın; ort. yaş 78.6±6.5 yıl; dağılım 55-89 yıl) retrospektif olarak incelendi. Elektrokardiyografik parametreler ve sol ventrikül kütle indeksi işlemden önce, işlemden bir gün sonra ve işlemden sonra üçüncü ayda hesaplandı.

Bulgular: Tp-e aralığı, Tp-e/QT ile Tp-e/QTc oranları, Tp-ed ve sol ventrikül kütle indeksi başlangıç değerlerine kıyasla, işlemin üçüncü ayında anlamlı ölçüde azaldı (tümü için $p<0.01$). Benzer bulgular QT, QTc ve QT dispersiyonu için gözlendi (tümü için $p<0.01$). Bu değişiklikler, kullanılan biyoprotez kapak türlerinden bağımsızdı. İşlem öncesinde, sol ventrikül kütle indeksi Tp-e ($r=0.350$, $p=0.007$), Tp-e/QT ($r=0.314$, $p=0.015$) ve Tp-e/QTc oranları ($r=0.285$, $p=0.029$) ile pozitif şekilde ilişkili idi. Çok değişkenli analizde, Tp-e aralığı ile sol ventrikül kütle indeksi arasında bağımsız bir ilişki tespit edildi ($\beta=0.350$, $p=0.007$).

Sonuç: Bu çalışmada Tp-e aralığı, Tp-e/QT ile Tp-e/QTc oranları, Tp-ed ve sol ventrikül kütle indeksi transkateter aort kapak implantasyonundan üç ay sonra anlamlı şekilde azalarak, sol ventrikülün yeniden yapılanmasını gösterdi. İki biyoprotez kapağın ventriküler repolarizasyon belirteçleri ve sol ventrikül kütle indeksi üzerindeki etkisi benzerdi.

Anahtar sözcükler: Aort kapak darlığı; elektrokardiyografi; kalp kapak protezi; ventriküler repolarizasyon belirteçleri.

Received: May 08, 2017 *Accepted:* June 30, 2017 *Published online*: January 09, 2018

Correspondence: Zülkif Tanrıverdi, MD. Balıklıgöl Devlet Hastanesi Kardiyoloji Kliniği, 63050 Eyyübiye, Şanlıurfa, Turkey.
Tel: +90 506 - 882 33 35 e-mail: ztverdi@gmail.com

Cite this article as:
Tanrıverdi Z, Çolluoğlu T, Ünal B, Dursun H, Kaya D. The effect of transcatheter aortic valve implantation on Tp-e interval, Tp-e/QT and Tp-e/QTc ratios, and Tp-e dispersion in patients with severe aortic stenosis. Turk Gogus Kalp Dama 2018;26(1):65-72.

©2018 All right reserved by the Turkish Society of Cardiovascular Surgery.

Sudden cardiac death (SCD) due to ventricular arrhythmia is a common event in patients with severe aortic stenosis (AS).[1] Increased wall stress due to pressure overload results in left ventricular hypertrophy (LVH), which impairs coronary blood flow and induces myocardial apoptosis and fibrosis.[2,3] These pathophysiological changes alter the function of ion channels which are responsible for cardiac repolarization, resulting in electrical heterogeneity.[3,4] Therefore, heterogeneous conduction makes the left ventricle more susceptible to arrhythmias, inducing the development of a reentrant circle.[1]

Myocardial repolarization abnormalities can be evaluated by QT interval and T wave changes on surface electrocardiography (ECG).[5] Recent studies have shown that the interval between the peak and the end of the T wave (Tp-e interval) can be used as an index of total dispersion of repolarization.[6] Increased Tp-e interval was reported to be associated with reentrant ventricular arrhythmias, SCD, and mortality.[7] However, Tp-e interval is influenced by the heart rate and body weight.[8] Therefore, Tp-e/QT and Tp-e/QTc (QT corrected) ratios, and Tp-e dispersion (Tp-ed) have been proposed to be better markers of ventricular repolarization.[5,8,9]

Transcatheter aortic valve implantation (TAVI) has been a reliable treatment modality alternative to surgery for high-risk patients with AS.[10,11] It reduces left ventricular (LV) afterload, leading to regression of LV mass (LVM) known as reverse ventricular remodeling.[12,13] Currently, two different bioprosthetic valves are widely used, the Medtronic CoreValve (MCV; Medtronic, Minneapolis, MN, USA) and the Edwards SAPIEN XT valve (ESV; Edwards Lifesciences, Irvine, CA, USA).[14]

Several studies showed that QTd (QT dispersion) increased in patients with AS, while TAVI was found to cause a significant reduction in QTd.[15-17] In addition, Tp-e interval, Tp-e/QT, and Tp-e/QTc ratios were shown to increase in patients with severe AS.[18] However, there is no study investigating the effect of TAVI on these novel markers. In the present study, therefore, we aimed to investigate the effect of TAVI with two types of bioprosthetic valves on repolarization markers and ventricular remodeling.

PATIENTS AND METHODS

Patient selection

All patients who underwent transfemoral TAVI with MCV or ESV between June 2012 and January 2015 in our clinic were retrospectively analyzed. Patients with baseline complete or incomplete bundle brunch block, history of permanent pacemaker (PPM) implantation, chronic treatment with Class I or Class III anti-arrhythmic drugs, non-interpretable ECG data, and patients who had significant coronary artery disease, defined as one or more vessels with stenosis of 50% or more in coronary angiography (CAG), were excluded from the study. In addition, six patients who developed left bundle branch block (LBBB) (MCV n=4 and ESV n=2) and seven patients who required PPM implantation (MCV n=5 and ESV n=2) after TAVI procedure were excluded from the study. Consequently, a total of 61 eligible patients (17 males, 44 females; mean age 78.6±6.5 years; range 55 to 89 years) were included in the study. A written informed consent was obtained from each patient. The study protocol was approved by the institutional Ethics Committee. The study was conducted in accordance with the principles of the Declaration of Helsinki.

Electrocardiography

Standard 12-lead ECG was recorded at 25 mm/s paper speed and 10 mm/mV amplitude in the supine position before TAVI on post-TAVI Day 1 and three months post-TAVI for each patient. To increase the accuracy of the measurements, calipers and magnifying lenses were used. All ECG recordings were scanned and transferred to the digital platform. The QT interval was measured from the beginning of the QRS complex to the end of T wave and was QTc for the heart rate using the Bazett's formula: $QTc = QT/\sqrt{RR}$.[5] The QTd was defined as the difference between the maximum and minimum QT interval of the 12 leads.[19] Tp-e interval was defined as the interval from the peak of a T wave to the end of T wave. Measurements of Tp-e interval were performed from the precordial leads, lead V2 was selected for measuring.[20] If a U wave followed the T wave, the T wave offset was measured as the nadir between the T and U waves.[7,21] When the T wave was negative or biphasic, the end point of the T wave was regarded, as when the trace returned to the baseline. T wave with an amplitude smaller than 1.5 mm were excluded from the analysis. Tp-e dispersion was defined as the difference between maximum and minimum Tp-e interval in the precordial leads.[5] In patients with atrial fibrillation, QT and Tp-e were measured in five consecutive beats, and the mean value was calculated.[22] Tp-e/QT and Tp-e/QTc ratios were calculated as the ratio of Tp-e to the corresponding QT and QTc interval in lead V2.

Echocardiography

Standard comprehensive transthoracic echocardiography was performed for each patient before

TAVI and was repeated on post-TAVI Day 1 and at three months after TAVI. All echocardiograms and Doppler measurements were performed according to the current recommendations.[23] The following measurements were obtained: left ventricle end-diastolic diameter (LVEDD), left ventricle end-systolic diameter (LVESD), interventricular septum diameter (IVSD), posterior wall diameter (PWD), LVEF, transvalvular pressure gradients, and aortic valve area (AVA). The LVM was calculated using the Devereux formula:[24] 0.8× [1.04× (LVEDD+IVSD+PWD)3 - (LVEDD)3] + 0.6 g and LVM was divided by body surface area to obtain the LVM index (LVMI).

Statistical analysis

Statistical analysis was performed using the IBM SPSS for Windows version 22.0 software (IBM Corp., Armonk, NY, USA). Continuous data were expressed in mean ± standard deviation (SD), while categorical data were expressed in percentages. Comparisons of continuous data were based on the Student's t-test for parametric variables and Mann-Whitney U test for non-parametric variables. Categorical data were compared using the chi-square test. Analysis of variance was performed for the comparison of repeated measurements using ANOVA for parametric variables and Friedman with Bonferroni corrections for non-parametric variables. Correlation analysis was used to investigate the associations between ECG and echocardiographic parameters. To avoid collinearity in evaluating the multivariate model, independent variables were tested for inter-correlation. Variables which showing a significant association with LVMI were entered in the multivariate linear regression analysis to determine independent predictors of LVMI. A p value of <0.05 was considered statistically significant.

RESULTS

In total, MCV was implanted to 40 (65.6%) and ESV to 21 (34.4%) patients. The mean post-TAVI length of hospitalization was 4.5±1.6 days, and no ventricular arrhythmia attack was observed in patients until discharge. Baseline characteristic of patients are listed in Table 1.

Table 1. Baseline characteristics of the study population (n=61)

	n	%	Mean±SD
Age (year)			78.6±6.5
Gender			
Male	17		
Female	44		
Body mass index (kg/m^2)			25.4±4.1
Hypertension	51	83.6	
Diabetes mellitus	19	31.1	
Previous cardiac surgery	11	18	
Society of thoracic surgery score			8.3±4.8
Logistic EuroSCORE			33.9±14.5
EuroSCORE II			10.2±7.3
Interventricular septum (cm)			1.5±0.2
Posterior wall (cm)			1.3±0.2
Maximum gradient (mmHg)			76.6±17.6
Mean gradient (mmHg)			48.0±11.4
Aortic valve area (cm^2)			0.5±0.1
Left ventricular ejection fraction			53.5±14.5
Medtronic CoreValve			
23	1	1.6	
26	14	23	
29	18	29.5	
31	7	11.5	
Edwards Sapien XT			
23	5	8.2	
26	15	24.6	
29	1	1.6	

SD: Standard deviation.

Table 2. Clinical, electrocardiographic and echocardiographic variables before and after transcatheter aortic valve implantation

Variables	Preoperative-TAVI	Postoperative-TAVI 1st day	Postoperative-TAVI 3rd month
	Mean±SD	Mean±SD	Mean±SD
Systolic blood pressure (mmHg)	113.8±13.3	118.3±10.1	117.1±10.6
Diastolic blood pressure (mmHg)	65.7±9.6	64.0±7.8	67.5±9.1
Heart rate (min)	77.4±14.1	81.1±13.5	77.7±12.7
QT (ms)	384.5±43.8	383.4±45.9	368.3±39.0*
QT corrected (ms)	435.2±46.1	441.3±37.2	416.3±41.9*
QT dispersion (ms)	48.6±34.9	38.2±23.0	32.4±17.7*
T peak-to-end time (ms)	88.3±21.7	86.1±17.4	74.0±18.3*
T peak-to-end time/QT	0.23±0.05	0.23±0.04	0.20±0.04*
T peak-to-end time/QT corrected	0.20±0.04	0.20±0.04	0.18±0.04*
Tp-e dispersion (ms)	27.0±16.4	23.1±15.7	13.4±10.3*
Left ventricular mass index (g/m^2)	155.0±41.8	154.7±41.3	132.9±33.8*

TAVI: Transcatheter aortic valve implantation; SD: Standard deviation; * p<0.01 versus pre-transcatheter aortic valve implantation.

Clinical, ECG, and echocardiographic parameters of the study group are shown in Table 2. Compared to pre-TAVI values, no significant difference was found for Tp-e, Tp-e/QT, Tp-e/QTc, Tp-ed and LVMI at post-TAVI Day 1 (p>0.05, for all). However, Tp-e (88.3±21.7 vs 74.0±18.3, p<0.01), Tp-e/QT (0.2±0.1 vs 0.2±0.0, p<0.01), Tp-e/QTc (0.2±0.0 vs 0.2±0.0, p<0.01), Tp-ed (27.0±16.4 vs 13.4±10.3, p<0.01), and LVMI (155.0±41.8 vs 132.9±33.8, p<0.01) significantly reduced after three months from TAVI, compared to pre-TAVI values (Figure 1). Similar findings were observed for QT, QTc, and QTd values. Systolic blood pressure, diastolic blood pressure, and heart rate did not significantly change during the follow-up period.

Comparison of ECG and echocardiographic parameters between MCV and ESV groups are listed in Table 3. QT, QTc, QTd, Tp-e, Tp-e/QT, Tp-e/QTc, Tp-ed and LVMI values were similar in both groups (p>0.05, for all).

In the correlation analysis, LVMI was positively correlated with Tp-e (r=0.350, p=0.007), Tp-e/QT (r=0.314, p=0.015), and Tp-e/QTc (r=0.285, p=0.029) before TAVI (Figure 2). However, no significant correlation was found between LVMI and QT parameters.

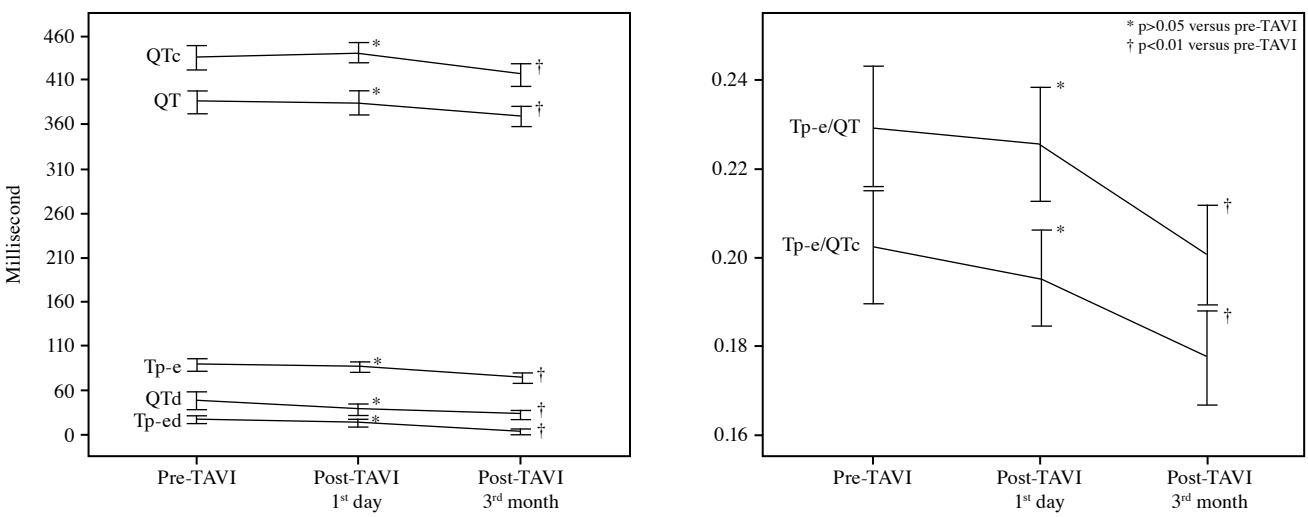


Figure 1. QT and Tp-e interval measurements at pre-TAVI, post-TAVI day 1 and three months post-TAVI.

QTc: QT corrected; Tp-e: T peak-to-end time; QTd: QT dispersion; Tp-ed: Tp-e dispersion; Pre-TAVI: Preoperative-transcatheter aortic valve implantation; Post-TAVI: Postoperative-transcatheter aortic valve implantation.

Table 3. Comparison of the effects of two bioprosthetic valves on electrocardiographic and echocardiographic variables

Variables	Preoperative-TAVI			Postoperative-TAVI 1st day			Postoperative-TAVI 3rd month		
	MCV (n=40)	ESV (n=21)	*p*	MCV (n=40)	ESV (n=21)	*p*	MCV (n=40)	ESV (n=21)	*p*
QT (ms)	392.8±48.8	379.0±35.4	NS	385.3±45.5	388.5±52.4	NS	360.9±45.3	365.0±39.4	NS
QTc (ms)	444.1±51.6	425.9±47.5	NS	441.9±46.4	448.1±39.4	NS	410.7±45.7	408.8±48.3	NS
QTd (ms)	55.5±37.6	35.7±14.0	NS	42.1±25.5	36.7±27.8	NS	35.2±18.2	27.9±15.8	NS
Tp-e (ms)	90.3±23.2	82.5±13.7	NS	86.3±17.4	86.3±19.8	NS	74.7±20.6	72.2±11.3	NS
Tp-e/QT	0.2±0.1	0.2±0.0	NS	0.2±0.0	0.2±0.1	NS	0.2±0.0	19.9±3.3	NS
Tp-e/QTc	0.2±0.0	0.2±0.0	NS	0.2±0.0	0.2±0.0	NS	0.2±0.0	0.2±0.0	NS
Tp-ed (ms)	28.3±16.8	23.3±11.5	NS	22.9±16.1	25.2±13.6	NS	14.0±12.2	12.2±4.6	NS
LVMI (g/m^2)	157.9±41.1	145.7±45.5	NS	157.3±40.4	145.8±45.5	NS	134.5±31.8	129.9±37.9	NS

TAVI: Transcatheter aortic valve implantation; MCV: Medtronic CoreValve; ESV: Edwards Sapien Valve; ms: milliseconds; QTc: QT corrected; QTd: QT dispersion; Tp-e: T peak-to-end time; Tp-ed: Tp-e dispersion; LVMI: left ventricular mass index; NS: Non-significant.

To assess independent association between LVMI and repolarization markers, multivariate linear regression analysis was performed including QT, QTc, QTd, Tp-e, Tp-e/QT, Tp-e/QTc, Tp-ed. Tp-e interval (β=0.350, p=007) was the only independent predictor of LVMI (Table 4).

DISCUSSION

The main findings of our study were as follows: *(i)* Tp-e and QT intervals, and LVMI were significantly reduced three months after TAVI indicating reverse ventricular electrical and structural remodeling; *(ii)* the effects of two different types of bioprosthetic valves on ventricular remodeling were similar; and *(iii)* Tp-e, Tp-e/QT and Tp-e/QTc were positively correlated with LVMI, and Tp-e interval was independently associated with LVMI. To the best of our knowledge, this is the first study demonstrating the effect of TAVI on Tp-e interval, Tp-e/QT, Tp-e/QTc ratios, and Tp-ed.

Myocardial ischemia is an important determinant responsible for repolarization changes and electrical heterogeneity.[25] Therefore, to evaluate the effect of TAVI on the ventricular repolarization parameters more clearly, we attentively ruled out the possibility of coronary artery disease in all our patients. In our study, CAG was performed to all patients before TAVI procedure. Patients who had ≥50% stenosis in any vessel were excluded from the study to increase the credibility of our study and prevent bias.

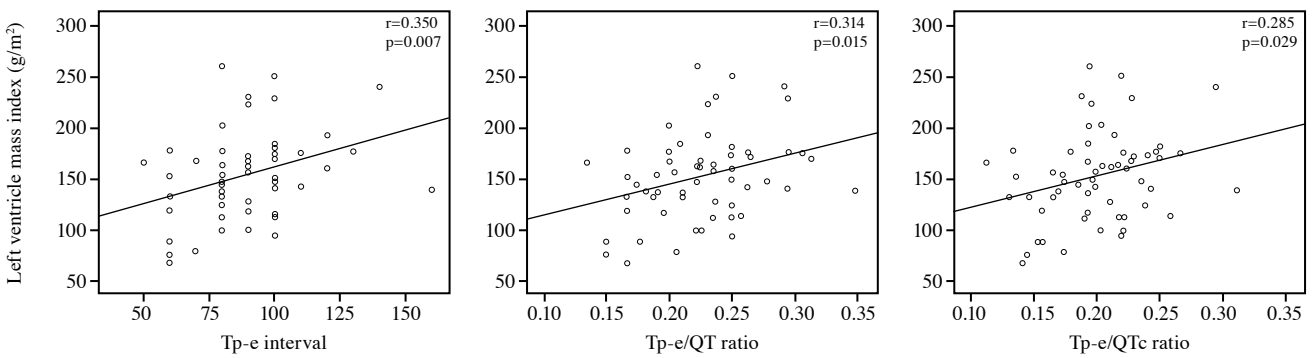


Figure 2. Correlation analysis between left ventricular hypertrophy index and Tp-e interval, Tp-e/QT and Tp-e/QTc ratio before TAVI.
Tp-e: T peak-to-end time; Tp-e/QT: T peak-to-end time/OT; Tp-e/QTc: T peak-to-end time/QT corrected; TAVI: Postoperative-transcatheter aortic valve implantation.

Table 4. Multivariate linear regression analysis showing independent predictor of left ventricular mass index

	Unstandardized coefficients		Standardized coefficients		
	B	SE	β	t	*p*
Tp-e interval	0.729	0.258	0.350	2.821	0.007

B: Unstandardized regression coefficient; SE: Standard error; β: Standardized β coefficient.

In patients with AS, LVH is a common adaptive mechanism due to pressure overload. This hypertrophic remodeling is a pathological and complex process resulting in structural and functional disturbance.[12,13] In addition, LVH prolongs action potential duration and increases inhomogeneity of ventricular repolarization, leading to an arrhythmogenic state.[26] After TAVI, LV afterload is reduced, and LVM regresses. Previous studies have shown that the regression of LVM occurs mainly within the first six months after TAVI.[14,27] This process is called as reverse remodeling of LV. In this study, the significant reduction in LVMI indicating reverse ventricular structural remodeling was observed at the end of three months of follow-up. As TAVI improves LV functions with structural remodeling, it is probable that it may also induce reverse ventricular electrical remodeling.

The QT measurement is a non-invasive marker of ventricular repolarization heterogeneity. Several studies have shown that QTd increased in patients with AS[15] and the increased QTd value significantly reduced after TAVI.[16,17] Similar to these studies, we also found significant reductions in QT, QTc, and QTd after three months from TAVI. However, in these studies, only QT parameters were used, and no information about the novel repolarization markers.

Recently, Tp-e, Tp-e/QT, Tp-e/QTc ratios, and Tp-ed have been proposed to be a better marker of ventricular repolarization. In this study, we firstly showed that TAVI led to significant reduction in Tp-e, Tp-e/QT, Tp-e/QTc ratios, and Tp-ed within a three-month period indicating reverse ventricular electrical remodeling. In addition, LVMI significantly reduced at post-TAVI three months and it was positively correlated with Tp-e, Tp-e/QT, and Tp-e/QTc. Moreover, Tp-e interval was independently associated with LVMI. Previous studies have also reported that increased LVM is associated with prolonged Tp-e intervals.[18,28] We, therefore, can conclude that the most probable reason for the improvement in repolarization markers is the regression of LVM by TAVI. Unlike Tp-e intervals, we found no significant correlation between LVMI and QT parameters. Therefore, we believe that Tp-e intervals are more sensitive than QT intervals for evaluating myocardial repolarization.

Another possible mechanism which may explain the effect of TAVI on repolarization markers is the recovery of autonomic functions. It has been reported that patients with severe AS have increased sympathetic nervous system activity and decreased sympathetic baroreflex gain.[29] Dumonteil et al.[29] proposed that autonomic dysfunction may provide further explanation for the high incidence of sudden death and mortality observed in these patients. They also showed normalization of sympathetic nervous system activity and restoration of arterial baroreflex gain after TAVI. Increased sympathetic activity was demonstrated to increase the QT interval.[30,31] Thus, it can be postulated that autonomic dysfunction may trigger ventricular repolarization abnormalities and prolongation of Tp-e. After TAVI, mechanical obstruction significantly decreases and cardiac output increases.[12-14] This induces improvement of autonomic dysfunctions, which may be one cause of the reduction in Tp-e interval.

In another study, Yayla et al.[18] showed that the Tp-e/QTc ratio had a significant positive correlation with mean aortic gradient ($r=0.192$, $p=0.049$). This result indicates that the ventricular repolarization anomalies increase in parallel with the severity of aortic stenosis. However, we did not find a correlation between Tp-e intervals and transaortic gradients. This may be due to our small sample of size, compared to the aforementioned study.

The self-expandable MCV and balloon-expandable ESV are structurally different. The former has been associated with higher conduction disorders and higher PPM requirement.[32,33] Although the conduction disorders related to transcatheter aortic valves are well-known, the effects of different bioprostheses on myocardial repolarization have not been studied yet. In our study, the structural difference of the bioprostheses did not lead to any difference on the ECG myocardial repolarization markers. However, it should be noted that we excluded those patients who developed LBBB or required PPM implantation after TAVI in this study. Nevertheless, the incidence of LBBB ($p>0.05$) and PPM requirement ($p>0.05$) were not different between the MCV and ESV groups. Further large-scale studies are, therefore, required to elucidate the effect of two different types of bioprosthetic valves on ventricular repolarization markers more clearly.

Although we found novel and significant findings, the small sample size was the main limitation of this study. Also, although none of the patients had ventricular arrhythmia until discharge, the patients were not prospectively followed for ventricular arrhythmia. A long-term Holter recording would be, thus, better to evaluate the incidence of ventricular arrhythmias in these patients. Therefore, prospective studies with more participants are needed to evaluate the impact of the improvements in novel repolarization markers on clinical outcomes after TAVI.

In conclusion, transcatheter aortic valve implantation caused significant reductions in the Tp-e interval, Tp-e/QT, and Tp-e/QTc ratios, Tp-ed, and left ventricular mass index in a short period of time, indicating reverse electrical and structural left ventricular remodeling. The effects of two different types of bioprosthetic valves on the repolarization markers were similar.

Declaration of conflicting interests

The authors declared no conflicts of interest with respect to the authorship and/or publication of this article.

Funding

The authors received no financial support for the research and/or authorship of this article.

REFERENCES

1. Sorgato A, Faggiano P, Aurigemma GP, Rusconi C, Gaasch WH. Ventricular arrhythmias in adult aortic stenosis: prevalence, mechanisms, and clinical relevance. Chest 1998;113:482-91.
2. Chambers J. The left ventricle in aortic stenosis: evidence for the use of ACE inhibitors. Heart 2006;92:420-3.
3. Krémer R. Arrhythmias in the natural history of aortic stenosis. Acta Cardiol 1992;47:135-40.
4. Rials SJ, Friehling TD, Marinchak RA, Kowey PR. Potassium channels in cardiac arrhythmias: focus on antiarrhythmic drug action. Prog Clin Biol Res 1990;334:111-21.
5. 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.
6. 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.
7. Panikkath R, Reinier K, Uy-Evanado A, Teodorescu C, Hattenhauer J, Mariani R, et al. Prolonged Tpeak-to-tend interval on the resting ECG is associated with increased risk of sudden cardiac death. Circ Arrhythm Electrophysiol 2011;4:441-7.
8. 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.
9. 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.
10. Leon MB, Smith CR, Mack M, Miller DC, Moses JW, Svensson LG, et al. Transcatheter aortic-valve implantation for aortic stenosis in patients who cannot undergo surgery. N Engl J Med 2010;363:1597-607.
11. Dagdelen S, Karabulut H, Senay S, Akyol A, Toraman F, Cagıl, et al. Transcatheter aortic valve implantation in patients with high-risk aortic stenosis: a clinical follow-up. Turk Gogus Kalp Dama 2011;19:495-502.
12. Fairbairn TA, Steadman CD, Mather AN, Motwani M, Blackman DJ, Plein S, et al. Assessment of valve haemodynamics, reverse ventricular remodelling and myocardial fibrosis following transcatheter aortic valve implantation compared to surgical aortic valve replacement: a cardiovascular magnetic resonance study. Heart 2013;99:1185-91.
13. Vizzardi E, D'Aloia A, Fiorina C, Bugatti S, Parrinello G, De Carlo M, et al. Early regression of left ventricular mass associated with diastolic improvement after transcatheter aortic valve implantation. J Am Soc Echocardiogr 2012;25:1091-8.
14. Dursun H, Tanriverdi Z, Colluoglu T, Kaya D. Effect of transcatheter aortic valve replacement on P-wave duration, P-wave dispersion and left atrial size. J Geriatr Cardiol 2015;12:613-7.
15. Ducceschi V, Sarubbi B, D'Andrea A, Liccardo B, Briglia N, Carozza A, et al. Increased QT dispersion and other repolarization abnormalities as a possible cause of electrical instability in isolated aortic stenosis. Int J Cardiol 1998;64:57-62.
16. Kasapkara HA, Ayhan H, Durmaz T, Keleş T, Sari C, Baştuğ S, et al. Short-term effect of transcatheter aortic valve implantation on QT dispersion. Turk J Med Sci 2015;45:615-8.
17. Erkan H, Hatem E, Ağaç MT, Korkmaz L, Gökdeniz T, Aykan AÇ, et al. Effect of transcatheter aortic valve implantation on QT dispersion in patients with aortic stenosis. J Geriatr Cardiol 2014;11:286-90.
18. Yayla Ç, Bilgin M, Akboğa MK, Gayretli Yayla K, Canpolat U, Dinç Asarcikli L, et al. Evaluation of Tp-E Interval and Tp-E/QT Ratio in Patients with Aortic Stenosis. Ann Noninvasive Electrocardiol 2016;21:287-93.
19. Day CP, McComb JM, Campbell RW. QT dispersion: an indication of arrhythmia risk in patients with long QT intervals. Br Heart J 1990;63:342-4.
20. Karaman K, Altunkaş F, Çetin M, Karayakali M, Arısoy A, Akar I, et al. New markers for ventricular repolarization in coronary slow flow: Tp-e interval, Tp-e/QT ratio, and Tp-e/QTc ratio. Ann Noninvasive Electrocardiol 2015;20:338-44.
21. Nuis RJ, Turgut G, van der Boon RM, van Mieghem NM, Nauta ST, Serruys PW, et al. Defective recovery of QT dispersion following transcatheter aortic valve implantation: frequency, predictors and prognosis. J Geriatr Cardiol 2015;12:482-8.
22. 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.
23. Zamorano JL, Badano LP, Bruce C, Chan KL, Gonçalves A, Hahn RT, et al. EAE/ASE recommendations for the use of echocardiography in new transcatheter interventions for valvular heart disease. J Am Soc Echocardiogr 2011;24:937-65.
24. Devereux RB, Alonso DR, Lutas EM, Gottlieb GJ, Campo E, Sachs I, et al. Echocardiographic assessment of left ventricular hypertrophy: comparison to necropsy findings. Am J Cardiol 1986;57:450-8.
25. Yoshimura M, Matsumoto K, Watanabe M, Yamashita N, Sanuki E, Sumida Y. Influence of exercise on QT dispersion

in hypertensive patients with left ventricular hypertrophy without coronary artery disease. Jpn Circ J 1999;63:881-4.

26. Oikarinen L, Nieminen MS, Viitasalo M, Toivonen L, Wachtell K, Papademetriou V, et al. Relation of QT interval and QT dispersion to echocardiographic left ventricular hypertrophy and geometric pattern in hypertensive patients. The LIFE study. The Losartan Intervention For Endpoint Reduction. J Hypertens 2001;19:1883-91.
27. La Manna A, Sanfilippo A, Capodanno D, Salemi A, Cadoni A, Cascone I, et al. Left ventricular reverse remodeling after transcatheter aortic valve implantation: a cardiovascular magnetic resonance study. J Cardiovasc Magn Reson 2013;15:39.
28. Darbar D, Cherry CJ, Kerins DM. QT dispersion is reduced after valve replacement in patients with aortic stenosis. Heart 1999;82:15-8.
29. Dumonteil N, Vaccaro A, Despas F, Labrunee M, Marcheix B, Lambert E, et al. Transcatheter aortic valve implantation reduces sympathetic activity and normalizes arterial spontaneous baroreflex in patients with aortic stenosis. JACC Cardiovasc Interv 2013;6:1195-202.
30. Marfella R, Gualdiero P, Siniscalchi M, Carusone C, Verza M, Marzano S, et al. Morning blood pressure peak, QT intervals, and sympathetic activity in hypertensive patients. Hypertension 2003;41:237-43.
31. Zipes DP. The long QT interval syndrome. A Rosetta stone for sympathetic related ventricular tachyarrhythmias. Circulation 1991;84:1414-9.
32. Siqueira D, Abizaid A, Arrais M, Sousa JE. Transcatheter aortic valve replacement in elderly patients. J Geriatr Cardiol 2012;9:78-82.
33. Gül M, Yıldırım A, Uslu N, Eksik A, Erol MK, Uyarel H, et al. The outcomes of transcatheter aortic valve implantation with Edwards SAPIEN or CoreValve devices: Single-center experience in Turkey. Turk Gogus Kalp Dama 2012;20:434-41.