Kardiologia Polska 2015; 73, 9: 747–752; DOI: 10.5603/KP.a2015.0090 ISSN 0022–9032

ARTYKUŁ ORYGINALNY / *ORIGINAL ARTICLE*

The effect of statin treatment on P-wave characteristics and atrial conduction time

Gulten Tacoy[1], Mehmet Kadri Akboga[2], Cagri Yayla[2], Ahmet Akyel[3], Sedat Turkoglu[1], Murat Ozdemir[1], Ridvan Yalcin[1]

[1]Department of Cardiology, Gazi University Medical Faculty, Ankara, Turkey
[2]Department of Cardiology, Turkiye Yuksek Ihtisas Training and Research Hospital, Ankara, Turkey
[3]Department of Cardiology, Dışkapı Yıldırım Beyazıt Education and Research Hospital, Ankara, Turkey

Abstract

Background and aim: The aim of this study was to evaluate the effect of statin treatment on P-wave morphology, dispersion, and tissue Doppler imaging-derived atrial conduction time (PA-TDI), which are known to be predictors of atrial fibrillation (AF).

Methods: A total of 132 patients with guideline-directed statin indications but no clinical atrial tachyarrhythmias were studied. P-wave duration, P-wave dispersion, and P-wave amplitude on surface 12-lead electrocardiogram and PA-TDI were evaluated before and after three months of statin (either atrovastatin 10–40 mg/d or rosuvastatin 10–20 mg/d) treatment.

Results: Total and low-density lipoprotein cholesterol were significantly reduced after statin therapy. P-wave dispersion significantly decreased from 39.6 ± 9.4 to 36.9 ± 9.6 ms. Statin treatment significantly decreased both the maximum (from 1.5 ± 0.36 to 1.45 ± 0.33 mV, p = 0.001) and the minimum (from 1.07 ± 0.28 to 1.04 ± 0.27 mV, p = 0.01) P-wave amplitude. The PA-TDI value was found to be significantly shorter after statin treatment (121.7 ± 18.7 vs. 118.7 ± 15.8 ms, p = 0.016)

Conclusions: Short-term statin therapy was shown to significantly affect P-wave amplitude, P-wave dispersion, and atrial conduction time in a broad range of patients without any clinical atrial tachyarrhythmia.

Key words: atrial fibrillation, echocardiography, statins, atrial conduction time, P-wave

Kardiol Pol 2015; 73, 9: 747–752

INTRODUCTION

Atrial fibrillation (AF) is the most common sustained arrhythmia observed in clinical practice [1], with increased morbidity and mortality [2]. Ectopic activities originating from the pulmonary veins and surronding venous structures may be the focus of AF [3]. Atrial conduction abnormalities are associated with a higher risk of AF occurence [4]. Patients with paroxysmal AF have been shown to have longer atrial electromechanical delay (AED) than patients with sinus rhythm [5]. P-wave morphology, duration, and dispersion on 12-lead surface electrocardiogram (ECG) and atrial conduction time as depicted by M-mode echocardiography, conventional Doppler, and tissue Doppler echocardiography are noninvasive tools to evaluate atrial conduction properties [6, 7]. AED can be measured from the onset of P-wave on surface ECG to the onset of atrial contraction time determined by tissue Doppler imaging echocardiography (PA-TDI) [8]. De Vos et al. [9] demonstrated that longer PA-TDI interval was associated with new onset AF.

3-hydroxy-3-methylglutaryl coenzyme A reductase inhibitors (i.e. statins) have lipid lowering, anti-inflammatory, and pleiotropic effects [10]. Studies that evaluate the relation between statin use and the occurence of AF have revealed inconsistent results [11, 12]. Accordingly, in this study we aimed to prospectively investigate the effect of short-term statin treatment on P-wave morphology, P-wave dispersion, and PA-TDI as measured by ECG and echocardiography, parameters that have previously been shown to predict the occurence of AF.

Address for correspondence:
Dr Mehmet Kadri Akboga, Turkiye Yuksek Ihtisas Training and Research Hospital, Cardiology Clinic, Sihhiye, Ankara, Turkey, e-mail: mkakboga@yahoo.com
Received: 11.01.2015 **Accepted:** 31.03.2015 **Available as AoP:** 28.04.2015

METHODS

Patients

A total of 132 patients with guideline-directed statin treatment indications were enrolled. Exclusion criteria were the use of statins within the prior six months, moderate to severe valvular heart disease, permanent cardiac pacemaker implantation, and existing atrial arrhythmias such as AF, atrial flutter, or atrial tachycardia documented by ECG or Holter ECG, acute coronary syndrome within the last 12 months, renal or hepatic disease, severe (> 400 mg/dL) hypertriglyceridaemia, anaemia, acute illness, leukocytosis, thrombocytosis, chronic inflammatory disease, and current use of any antiarrhythmic drugs except beta-blockers and calcium channel blockers. Throughout the study the patients recieved the same drugs.

At the baseline evaluation, a fasting lipid panel, a simultaneously recorded 12-lead surface ECG, and transthoracic echocardiography including TDI were obtained. Subsequently the patients were started on statin therapy. The statin type and dose (either atorvastatin 10–40 mg or rosuvastatin 10–20 mg) was left to the physician in charge but the guideline proposed low-density lipoprotein cholesterol (LDL-C) levels were targeted. Patients were seen at the 1st, the 2nd, and finally the 3rd month of statin therapy when the study ended. At the 1st and the 2nd follow-up visits, the patients were clinically examined with respect to compliance with statin therapy, and those who were noncompliant were excluded form further analysis**.** At the end of three months of statin therapy, fasting lipid panel, simultaneously recorded 12-lead surface ECG, and transthoracic echocardiography including TDI were again obtained from each patient. Before study enrollment we suggested to all patients that they adopt lifestyle changes and maintain them until the end of the study.

Electrocardiogram

The 12-lead simultaneous surface ECGs were obtained in the supine position at a paper speed of 50 mm/s and a gain setting of 10 mm/mV. All ECGs were evaluated by a cardiologist who was blinded to the study. The onset of the P-wave was defined as the junction between the isoelectric line and the beginning of P-wave deflection (either negative or positive), and the offset in exactly the same manner. The P-wave duration was depicted as the duration between the onset and offset of the P-wave in each lead. The interval between the longest (Pmax) and the shortest (Pmin) P-wave was defined as the P-wave dispersion. The P-wave amplitude was defined as the vertical distance between the isoelectric line and the peak of the P-wave in each lead. The maximum and the minimum P-wave amplitudes on each 12-lead surface ECG were noted. We measured the P-wave amplitude as an absolute value. After statin treatment maximum and minimum P-wave amplitudes were measured in the same leads.

Transthoracic echocardiography

Transthoracic echocardiography (TTE) (Vivid 7 system, 2.5- to 3.5-MHz transducer, GE-Vingmed Ultrasound AS, Horten, Norway) examination was performed according to the standards of the American Society of Echocardiography by two cardiologists who were blinded to the study protocol [13]. During TTE examination, left ventricular end-diastolic and end-systolic diameters, ejection fraction, interventricular septal and posterior wall thickness, and right heart dimensions were calculated. Transmitral pulsed-wave Doppler velocities were recorded from the apical four-chamber view with the Doppler sample placed between the tips of the mitral leaflets. Early (E) and late (A) wave velocities, E/a ratio, deceleration time, and isovolumetric relaxation time were measured from the mitral inflow profile. The myocardial systolic (Sm), early diastolic (Em), and late diastolic (Am) velocities were obtained at the septal and lateral mitral annulus by using a tissue Doppler sample volume. AED was measured from the lateral mitral annulus by calculating the time interval from the initiation of the P-wave on surface ECG (lead II) to the beginning of the A-wave in tissue Doppler imagings (PA-TDI).

The study was approved by the local Ethics Committee, and informed consent was obtained from all patients before study entry.

Statistical analysis

The data are expressed as the mean ± standard deviation. Continuous variables were tested for normal distribution with Kolmogorov-Smirnov test and compared by means of paired student's t test before and after statin treatment. Categorical data were summarised as percentages and compared using the χ^2 test. In cases of non-normal distribution, Mann Whitney U test or Wilcoxon signed rank test was used. A p value < 0.05 was considered significant. Statistical analyses were performed using SPSS 16.0 Statistical Package for Windows (SPSS Inc, Chicago, Illinois).

RESULTS

Of the 132 patients enrolled, 38 patients had to be excluded from the final evaluation. Seventeen patients were lost to follow-up, 13 were drug non-compliant, and eight patients stopped statin treatment due to side effects (dyspeptic problems in four, myalgia in two, reversible skin lesions in one, and dizziness in one). As a result, follow-up data were available for 94 patients. The baseline characteristics of the 94 patients are depicted in Table 1.

During a mean follow-up of 109 days, 50 patients received atorvastatin (4 [4.2%] patients 10 mg, 35 [37.2%] patients 20 mg, 11 [11.7%] patients 40 mg) and 44 patients received rosuvastatin (14 [14.8%] patients 10 mg and 30 [31.9%] patients 20 mg).

The fasting lipid profile, 12-lead surface ECG, and echocardiographic findings before and after statin therapy are given in Table 2. Accordingly, there was a significant decrease in total cholesterol (245.4 ± 50.3 vs. 169.9 ± 37.9 mg/dL, p < 0.001), triglyceride (170.8 ± 88.0 vs. 136.2 ± 58.1 mg/dL, p < 0.001), and LDL-C levels (169.8 ± 42.9 vs. 97.4 ± 31.6 mg/dL, p < 0.001). The high-density lipoprotein cholesterol level increased after statin treatment, but the difference was statistically non-significant (45.5 ± 10.4 vs. 46.6 ± 11.6 mg/dL, p = 0.045).

Table 1. Baseline characteristics and laboratory parameters of the study population

Parameters		All patients (n = 94)
Age [years]		55.0 ± 9.3
Female		53 (56.4%)
Hypertension		52 (55.3%)
Diabetes mellitus		24 (25.5%)
Smoking		27 (28.7%)
Family history		9 (9.6%)
Coronary artery disease		30 (31.9%)
Medical therapy:		
RAS blocker		42 (44.7%)
Diuretic		18 (19.1%)
Calcium channel blocker		12 (12.8%)
Beta-blocker		25 (26.6%)
Acetylsalicylate		24 (25.5%)
Clopidogrel		9 (9.5%)
Oral antidiabetic		20 (21.3%)
Atorvastatin	10 mg	4 (4.2%)
	20 mg	35 (37.2%)
	40 mg	11 (11.7%)
Rosuvastatin	10 mg	14 (14.8%)
	20 mg	30 (31.9%)
Creatinine [mg/dL]		0.8 ± 0.1
Haemoglobin [g/dL]		14.0 ± 1.5
Platelet [10^3/mm^3]		249.5 ± 50.1

Data were given as mean ± standard deviation or percentage; RAS — renin–angiotensin system

P-wave dispersion significantly decreased from 39.6 ± 9.4 to 36.9 ± 9.6 ms. Statin treatment significantly decreased both the maximum (from 1.5 ± 0.36 to 1.45 ± 0.33 mV, p = 0.001) and the minimum P-wave amplitude (from 1.07 ± 0.28 to 1.04 ± 0.27 mV, p = 0.01). The PA-TDI value was found to be significantly shorter after statin treatment (121.7 ± 18.7 vs. 118.7 ± 15.8 ms, p = 0.016) (Table 2). The other echocardiographic parameters before and after statin treatment are shown in Table 3. Left ventricular dimensions and Doppler parameters displayed no significant changes after statin treatment.

DISCUSSION

Our study demonstrated that three months of statin treatment with either atorvastatin or rosuvastatin decreased the P-wave amplitude, the P-wave dispersion on the surface ECG, and atrial conduction time as measured by TDI echocardiography in a broad range of patients with statin therapy indications, but not clinical atrial tachyarrhythmia.

Previous studies have shown that statins may prevent AF occurrence in patients with stable angina pectoris and may be effective against ventricular arryhythmias in patients with intracardiac defibrillators [14, 15]. The exact mechanism of the antiarryhythmic effects of statins, if any, have not yet been clearly defined. Inflammation, oxidative stress, and endothelial dysfunction may lead to AF [16]. Statins may prevent atrial remodelling with antiinflammatory and antioxi-

Table 2. The lipid profile, and electrocardiographic and echocardiographic indices before and after statin therapy

	Before	After	P
Heart rate [bpm]	73.5 ± 10.5	73.6 ± 10.9	0.851
PR interval [ms]	151.8 ± 19.6	151.1 ± 18.7	0.243
P-wave dispersion [ms]	39.6 ± 9.4	36.9 ± 9.6	0.014
P-wave amplitude [mV]:			
Maximum	1.5 ± 0.36	1.45 ± 0.33	0.001
Minumum	1.07 ± 0.28	1.04 ± 0.27	0.010
PA-TDI [ms]	121.7 ± 18.7	118.7 ± 15.8	0.016
Total cholesterol [mg/dL]	245.4 ± 50.3	169.9 ± 37.9	< 0.001
LDL-cholesterol [mg/dL]	169.8 ± 42.9	97.4 ± 31.6	< 0.001
HDL-cholesterol [mg/dL]	45.5 ± 10.4	46.6 ± 11.6	0.045
Triglyceride [mg/dL]	170.8 ± 88.0	136.2 ± 58.1	< 0.001

Data are given as mean ± standard deviation; PA-TDI — atrial conduction time determined by tissue Doppler imaging; LDL — low-density lipoprotein; HDL — high-density lipoprotein

Table 3. Echocardiographic findings before and after statin therapy

	Before	After	P
Left atrium [mm]	35.82 ± 4.55	35.31 ± 3.74	0.393
LVEDD [mm]	48.0 ± 2.82	47.5 ± 3.27	0.544
LVESD [mm]	29.12 ± 3.09	28.87 ± 3.94	0.871
LVEF [%]	67.52 ± 8.14	67.95 ± 6.70	0.345
LVEDV [mL/m^2]	113.83 ± 8.40	117.67 ± 17.61	0.607
LVESV [mL/m^2]	33.88 ± 8.97	33.66 ± 11.62	0.955
IVRT [ms]	121.82 ± 24.63	122.89 ± 27.02	0.716
Deceleration time [ms]	173.6 ± 45.89	172.9 ± 48.88	0.883
Mitral annulus E' [m/s]	8.43 ± 2.75	8.13 ± 2.07	0.720
Mitral annulus A' [m/s]	10.75 ± 2.06	10.72 ± 1.99	0.927
Mitral annulus S' [m/s]	7.46 ± 1.62	7.30 ± 1.48	0.344
Lateral annulus E' [m/s]	10.19 ± 3.26	9.93 ± 2.99	0.264
Lateral annulus A' [m/s]	10.80 ± 2.84	11.06 ± 2.66	0.286
Lateral annulus S' [m/s]	7.63 ± 2.11	7.67 ± 2.01	0.858
Tricuspid annulus E' [m/s]	10.88 ± 2.78	10.90 ± 3.22	0.939
Tricuspid annulus A' [m/s]	15.43 ± 3.78	16.0 ± 4.83	0.184
Tricuspid annulus S' [m/s]	11.93 ± 2.89	12.18 ± 3.18	0.348
E wave velocity [m/s]	0.64 ± 0.18	0.66 ± 0.17	0.306
A wave velocity [m/s]	0.69 ± 0.20	0.69 ± 9.25	0.313

Data are given as mean ± standard deviation; LVEDD — left ventricular end-diastolic diameter; LVESD — left ventricular end-systolic diameter; LVEF — left ventricular ejection fraction; LVEDV — left ventricular end-diastolic volume; LVESV — left ventricular end-systolic volume; IVRT — isovolumetric relaxation time

dant effects [16]. The majority of the antiarrythmic effects of statins are evident in high-risk patient populations [17]. It was demonstrated that statins may protect atrial tissue from damage and structural remodelling via sympatholitic activity [18]. Statins may alter ion channel activity and angiotensin II-dependent pathway [18].

Atrial conduction time can be evaluated with TDI echocardiography. Daubert et al. [7] demonstrated that intraatrial and interatrial conduction delay was associated with a higher risk of AF occurence. De Vos et al. [9] found that left AED was significantly increased in patients with paroxymal AF and determined it as an independent predictor of new-onset AF. In another study, maximum P-wave amplitude and P-wave dispersion were shown to predict the development of AF [19]. Van Beeumen et al. [20] showed that bipolar electrogram voltage decreased after circumferential pulmonary vein isolation for treatment of AF. This finding was related with ablated left atrial tissue area due to decreased depolarisation and electrical capacity, and this was a predictor of AF recurrence. Furthermore, Rader et al. [21] found that preoperative P-wave amplitude was associated with postoperative AF occurrence. In our study we found that P-wave amplitude decreased significantly after statin therapy; therefore, we proposed that statin treatment could alter depolarisation characteristics of the left atrium and be preventive for the development of AF.

In a meta-analysis it was shown that the incidence and recurrence of AF in a high-risk population group was significantly reduced by statin usage [22]. Warita et al. [23] investigated the role of pitavastatin on prevention of AF and they showed that pitavastatin might reduce the incidence of new onset AF in elderly hypertensive patients. Kourliouros et al. [24] showed that the effect of statins on postoperative AF was dose related and higher doses were more effective in the prevention of postoperative AF. Young-Xu et al. [14] found that male patients, hypertensive patients, and patients over 70 years old had the greatest benefit from statins for prevention of AF. Interestingly, they showed that there was no risk reduction of AF in patients who received non-statin cholesterol-lowering agents [14]. They also found that the preventive effects of statins were independent of the reduction in serum cholesterol level. In another study, Humphries et al. [25] investigated statins after successful cardioversion. They showed that statins were closely related with risk reduction in recurrent AF, but this effect was only seen in patients taking concomitant beta-blocker treatment.

In our study we did not evaluate the occurrence of AF but instead we studied surrogate markers of AF in an attempt to disclose the probable mechanisms of statin-induced reduction in AF occurrence, if any, and we found that the markers we studied significantly changed in response to statin therapy. This

is only hypothesis generating and a longer duration of drug exposure in the presence of a control group and 24–48 h Holter ECG recordings in addition would be needed to prove the association of statin therapy with a reduction in AF occurence.

Limitations of the study

The lack of a control group, small sample size, and the rather short (109 days) therapy duration are the obvious study limitations that preclude any firm conclusions as to the possible link between statins and the occurence of AF. All ECGs were evaluated by one cardiologist who was blinded to the study, and therefore we could not evaluate inter- and intraobserver variability in our manuscript.

CONCLUSIONS

In conclusion, we have shown that a short course of statin therapy leads to significant changes in P-wave morphology, P-wave dispersion, and tissue Doppler derived atrial conduction time, which are known to be predictors of AF. Whether this finding would translate to a reduction in the occurence of AF needs to be clarified by further research.

Conflict of interest: *none declared*

References

1. Kannel WB, Abbott RD, Savage DD, McNamara PM. Epidemiological features of chronic atrial fibrillation: the Framingham study. N Engl J Med, 1982; 306: 1018–1022.
2. Benjamin EJ, Wolf PA, D'Agostino RB et al. Impact of atrial fibrillation on the risk of death: The Framingham Heart Study. Circulation, 1998; 98: 946–952.
3. Haïssaguerre M, Jaïs P, Shah DC et al. Spontaneous initiation of atrial fibrillation by ectopic beats originating in the pulmonary veins. N Engl J Med, 1998; 339: 659–666.
4. Shimizu A, Fukatani M, Tanigawa M et al. Intraatrial conduction delay and fragmented atrial activity in patients with paroxymal atrial fibrillation. Jpn Circ J, 1989; 9: 1023–1030
5. Cui QQ, Zhang W, Wang H et al. Assessment of atrial electromechanical coupling and influential factors in nonrheumatic paroxymal atrial fibrillation. Clin Cardiol, 2008; 31: 74–78. doi: 10.1002/clc.20162.
6. Deniz A, Sahiner L, Aytemir K et al. Tissue doppler echocardiography can be a useful technique to evaluate atrial conduction time. Cardiol J, 2012; 19: 487–493. doi: 10.1002/clc.20162.
7. Daubert JC, Pavin D, Jauvert G, Mabo P. Intra and interatrial conduction delay: Implications for cardiac pacing. Pacing Clin Electrophysiol, 2004; 27: 507–525.
8. Ozer N, Yavuz B, Can I et al. Doppler tissue evaluation of intra-atrial and interatrial electromechanical delay and comparison with P-wave dispersion in patients with mitral stenosis. J Am Soc Echocardiogr, 2005; 18: 945–948.
9. De Vos CB, Weijs B, Crijns HJ et al. Atrial tissue Doppler imaging for prediction of new-onset atrial fibrilation. Heart, 2009; 95: 835–840. doi: 10.1136/hrt.2008.148528.
10. Pedersen TR, Wilhelmsen L, Faergeman O et al. Follow-up study of patients randomized in the Scandinavian simvastatin survival study (4S) of cholesterol lowering. Am J Cardiol, 2000; 86: 257–262.
11. Faunchier L, Pierre B, de Labriolle A et al. Antiarrhythmic effect of statin therapy and atrial fibrillation a meta-analysis of randomized controlled trials. J Am Coll Cardiol, 2008; 51: 828–835. doi: 10.1016/j.jacc.2007.09.063.
12. Liu T, Li L, Koratzopoulos P et al. Statin use and development of atrial fibrillation: a systematic review and meta-analysis of randomized controlled clinical trials and obsevational studies. Int J Cardiol, 2008; 126: 160–170.
13. Lang RM, Bierig M, Devereux RB et al. Recommendations for chamber quantification: a report from the American Society of Echocardiography's Guidelines and Standards Committee and the Chamber Quantification Writing Group, developed in conjunction with the European Association of Echocardiography, a branch of the European Society of Cardiology. Am J Soc Echocardiogr, 2005; 18: 1440–1463.
14. Young-Xu Y, Jabbour S, Goldberg R et al. Usefulness of statin drugs in protecting against atrial fibrillation in patients with coronary artery disease. Am J Cardiol, 2003; 92: 1379–1383.
15. Mitchell LB, Powell JL, Gillis AM et al. Are lipid-lowering drugs als antiarrhythmic drugs? An analysis of the Antiarrhythmics versus Implantable Defibrillators (AVID) trial. J Am Coll Cardiol, 2003; 42: 81–87.
16. Korantzopoulos P, Kolettis T, Siogas K, Goudevenos J. Atrial fibrillation and electrical remodelling: the potential role of inflammation and oxidative stress. Med Sci Monit, 2003; 9: 225–229.
17. De Sutter J, Tavernier R, De Buyzere M et al. Lipid lowering drugs and recurrences of life-threatening ventricular arrhythmias in high-risk pateints. J Am Coll Cardiol, 2000; 36: 766–772.
18. Pellegrini CN, Vittinghoff E, Lin F et al. Statin use is associated with lower risk of atrial fibrillation in women with coronary disease: The HERS Trial. Heart, 2009; 95: 704–708. doi: 10.1136/hrt.2008.154054.
19. Dogan A, Kahraman H, Ozturk M, Avsar A. P-wave dispersion and left atrial appendage function for predicting recurrence after conversion of atrial fibrillation and relation of P-wave dispersion to appendage function. Echocardiography, 2004; 21: 523–530.
20. Van Beeumen K, Houben R, Tavernier R et al. Changer in P-wave area and P-wave duration after circumferential pulmonary vein isolation. Europace, 2010; 12: 798–804. doi: 10.1093/europace/eup410.
21. Rader F, Costantini O, Jarrett C et al. Quantitative electrocardiography for predicting postoperative atrial fibrillation after cardiac surgery. J Electrocardiol, 2011; 44: 761–767. doi: 10.1016/j.jelectrocard.2010.12.005.
22. Faunchier L, Pierre B, de Labriolle A et al. Antiarrhythmic effect of statin therapy and atrial fibrillation a meta-analysis of randomized controlled trials. J Am Coll Cardiol, 2008; 51: 828–835. doi: 10.1016/j.jacc.2007.09.063.
23. Warita S, Kawasaki M, Tanaka R et al. Effects of pitavastatin on cardiac structue and function and on prevention of atrial fibrillation in elderly hypertensive patients. Circ J, 2012; 76: 2755–2762.
24. Kourliouros A, De Souza A, Roberts N et al. Dose-related effect of statins on atrial fibrillation after cardiac surgery. Ann Thorac Surg, 2008; 85: 1515–1520. doi: 10.1016/j.athoracsur.2008.01.040.
25. Humphries KH, Lee M, Sheldon R et al. Statin use and recurrence of atrial fibrillation after successful cardioversion. Am Heart J, 2007; 154: 908–913.

Cite this article as: Tacoy G, Akboga MK, Yayla C et al. The effect of statin treatment on P-wave characteristics and atrial conduction time. Kardiol Pol, 2015; 73: 747–752. doi: 10.5603/KP.a2015.0090.

Wpływ leczenia statynami na morfologię załamka P i czas przewodzenia przedsionkowego

Gulten Tacoy[1], Mehmet Kadri Akboga[2], Cagri Yayla[2], Ahmet Akyel[3], Sedat Turkoglu[1], Murat Ozdemir[1], Ridvan Yalcin[1]

[1]Department of Cardiology, Gazi University Medical Faculty, Ankara, Turcja
[2]Department of Cardiology, Turkiye Yuksek Ihtisas Training and Research Hospital, Ankara, Turcja
[3]Department of Cardiology, Dışkapı Yıldırım Beyazıt Education and Research Hospital, Ankara, Turcja

Streszczenie

Wstęp i cel: Celem niniejszego badania była ocena wpływu leczenia statynami na morfologię i dyspersję załamka P oraz czas przewodzenia przedsionkowego określonego w obrazowaniu metodą doplera tkankowego (PA-TDI) uznawany za czynnik predykcyjny migotania przedsionków (AF).

Metody: Przeanalizowano dane 132 chorych ze wskazaniami do stosowania statyn, zgodnie z wytycznymi terapeutycznymi, ale bez klinicznych objawów tachyarytmii przedsionkowej. Przed rozpoczęciem terapii i po 3-miesięcznym leczeniu statynami (atorwastatyna 10–40 mg/d. lub rosuwastatyna 10–20 mg/d.) oceniano czas trwania, dyspersję i amplitudę załamka P w powierzchniowym 12-odprowadzeniowym elektrokardiogramie oraz określano czas PA-TDI.

Wyniki: Stężenie cholesterolu całkowitego i cholesterolu frakcji LDL było istotnie niższe po leczeniu statynami. Nastąpiło istotne zmniejszenie dyspersji załamka P z 39,6 ± 9,4 do 36,9 ± 9,6 ms. Terapia statynami spowodowała znamienną redukcję maksymalnej (z 1,5 ± 0,36 do 1,45 ± 0,33 mV; p = 0,001) i minimalnej amplitudy załamka P (z 1,07 ± 0,28 do 1,04 ± 0,27 mV; p = 0,01). Czas PA-TDI był istotnie krótszy po leczeniu statynami (121,7 ± 18,7 vs. 118,7 ± 15,8 ms; p = 0,016)

Wnioski: Wykazano, że krótkotrwała terapia statynami istotnie wpływa na amplitudę i dyspersję załamka P oraz czas przewodzenia przedsionkowego w zróżnicowanej grupie chorych bez objawowej tachyarytmii przedsionkowej.

Słowa kluczowe: migotanie przedsionków, echokardiografia, statyny, czas przewodzenia przedsionkowego, załamek P

Kardiol Pol 2015; 73, 9: 747–752

Adres do korespondencji:
Dr Mehmet Kadri Akboga, Turkiye Yuksek Ihtisas Training and Research Hospital, Cardiology Clinic, Sihhiye, Ankara, Turkey, e-mail: mkakboga@yahoo.com
Praca wpłynęła: 11.01.2015 r. **Zaakceptowana do druku:** 31.03.2015 r. **Data publikacji AoP:** 28.04.2015 r.