Kardiologia Polska 2015; 73, 8: 637–643; DOI: 10.5603/KP.a2015.0028 ISSN 0022–9032

ARTYKUŁ ORYGINALNY / *ORIGINAL ARTICLE*

Atrial conduction abnormalities in patients with psoriasis vulgaris

Abdurrahman Tasal, Tolga Sinan Guvenc, Seref Kul, Ahmet Bacaksiz, Ercan Erdogan, Osman Sonmez, Emrah Sevgili, Didem Dizman, Nahide Onsun, Nuray K. Ay

Department of Cardiology, Bezmialem Vakif University, Istanbul, Turkey

Abstract

Background: Psoriasis vulgaris is one of the most common chronic inflammatory skin disorders. Patients with psoriasis are at risk of developing atrial fibrillation (AF). The electromechanical delay (EMD) is the time interval from the onset of the P wave on surface electrocardiography (ECG) to the beginning of the A wave. Prolonged atrial EMD is an independent risk factor for the development of AF.

Aim: This study investigated the intra- and interatrial EMD in patients with psoriasis.

Methods: This study included 85 adults with psoriasis vulgaris (Group 1) and 46 age- and sex-matched healthy individuals (Group 2). ECGs were obtained from all subjects, and atrial EMD variables were calculated. Results are reported as means ± standard deviations and percentages. Continuous variables were analysed using Student's t-test. A p-value < 0.05 was considered statistically significant.

Results: Interatrial electromechanical delay (IA-EMD) and intra-left atrial electromechanical delay (ILA-EMD) were significantly longer in the psoriasis group compared with controls. A correlation analysis between psoriasis severity (PASI score) and the atrial conduction parameters revealed a significant positive correlation between PASI and IA-EMD ($r = 0.261$, $p < 0.001$). In addition, there was a positive correlation between high-sensitivity C-reactive protein (hsCRP) and IA-EMD ($p = 0.022$).

Conclusions: The atrial conduction time was longer in patients with psoriasis vulgaris and it correlated with the severity of disease and hsCRP. Since the association between delayed conduction and AF is known, the measurement of intra-atrial conduction times could be a practical tool to estimate the AF risk in these patients.

Key words: atrial fibrillation, psoriasis vulgaris, electromechanical delay

Kardiol Pol 2015; 73, 8: 637–643

INTRODUCTION

Psoriasis vulgaris is a chronic inflammatory skin disorder characterised by lesions affecting the skin, scalp, nails, and joints [1]. Although the pathogenesis of psoriasis is still not completely understood, inflammatory processes and oxidative stress are considered as the most important mechanisms for disease development and maintenance [2]. In addition to dermal and arthritic manifestations, psoriasis is associated with a multitude of cardiovascular (CV) disorders, including premature atherosclerosis, cerebrovascular disease, and stroke [3]. The development of CV complications is attributed to the systemic inflammation associated with psoriasis [2]. Recently, it was also shown that psoriasis patients have a tendency for atrial fibrillation (AF) [4]. AF is the most common type of arrhythmia, with a frequency of 1% in the general population, and it is associated with embolic stroke and increased CV morbidity and mortality [5]. Multifactorial, chronic inflammation is an important contributor to the development and maintenance of AF, and myocardial infiltration with inflammatory cells had been demonstrated in AF patients previously [6]. Since psoriasis is also a chronic inflammatory condition, it is possible that myocardial inflammation and associated conduction heterogeneity within the atrial myocardium could be the pathophysiological links explaining the association between psoriasis and AF.

Prolonged intra- and interatrial conduction times are markers of abnormal conductivity within the atrium and of

Address for correspondence:
Dr Seref Kul, Bezmialem Vakif University, Department of Cardiology, Adnan Menderes Bulvari (Vatan caddesi) 34093, Fatih Istanbul, Turkey, e-mail: doktorseref@hotmail.com
Received: 16.03.2014 **Accepted:** 27.11.2014 **Available as AoP:** 03.02.2015

vulnerability to AF [7]. A simple measurement for evaluating the intra- and interatrial conduction times is electromechanical delay (EMD). Measuring the EMD using echocardiography is a promising tool for evaluating the risk of AF in certain risk groups [8]. Since psoriasis is characterised by an increased incidence of AF, perhaps an elevated EMD in these patients could aid clinical risk assessment. Beyond the clinical evaluation, demonstration of an abnormal EMD in psoriatic patients might lead to a better understanding of the pathophysiological substrates that lead to an increased incidence of AF, since EMD is a marker for abnormal intra-atrial conductivity.

Therefore, this study assessed atrial conduction abnormalities in psoriasis vulgaris patients and examined the relationship between these parameters and the severity of psoriasis vulgaris.

Table 1. Disease specific characteristics of patients with psoriasis vulgaris

Parameters	Mean ± standard deviation
Mean age of onset of psoriasis [years]	21.1 ± 8.7 (range: 7–35)
Mean duration of disease [years]	11.8 ± 7.0 (range: 3–26)
Mean PASI score	4.1 ± 4.2 (range: 0.2–18.4)
Mean PSI score	1.7 ± 0.9 (range: 0–3)
Mean affected BSA [%]	8.2 ± 12.5 (range: 0–90)
Mean NAPSI score	22.2 ± 21.2 (range: 0–93)

PASI — psoriasis area and severity index; PSI — psoriasis severity index; BSA — body surface area; NAPSI — nail psoriasis severity index

METHODS

Patients

The study population included 85 consecutive psoriasis patients older than 18 years, in whom diagnosis had been established by biopsy in our dermatology department, and who had been followed for at least three years (Group 1); 46 age- and sex-matched healthy subjects served as a control group (Group 2). Subjects with hypertension, diabetes mellitus, coronary artery disease (CAD), lung diseases, pulmonary hypertension, valvular heart disease, liver or kidney disease, collagen vascular diseases, rhythms other than sinus, any CV drug use, abnormal thyroid function, or abnormal serum electrolyte values were excluded from the study. Patients with co-existing psoriatic arthritis (diagnosed by a rheumatologist) were excluded due to possible concomitant effects on heart rhythm. All subjects provided informed consent, and the study was approved by the local ethics committee.

The demographic characteristics, disease duration, and drug history of each patient were recorded. The body mass index (BMI) of all subjects was calculated as body weight (in kg) divided by height squared (in m^2). Obesity was defined as a BMI ≥ 30 kg/m^2. Biochemical variables such as fasting glucose levels and the lipid panel were recorded. The serum high-sensitivity C-reactive protein (hsCRP) level was obtained using the nephelometric method and a Dade Behring Cardio Phase kit.

Evaluation of patient disease activity

The diagnosis of psoriasis vulgaris was based on a dermatologist's diagnosis or the description of characteristic lesions, with subsequent pathological confirmation. The mean duration of the disease was 11.8 ± 7.0 (range 3–26) years. Clinical severity was assessed using the psoriasis area and severity index (PASI) [9]. The PASI assesses four body regions: the head, trunk, and upper and lower extremities. For each region, the surface area involved is graded from 0 to 6, and each of the three variables (erythema, thickness, and scaling of the plaques) is graded from 0 to 4. The scores from each region are summed to give a total PASI score ranging from 0 to 72. The psoriasis severity index (PSI) was also used to evaluate clinical signs (erythema, thickness, and scaling) on a scale of 0 (absent) to 3 (severe) [10]. The affected body surface area (BSA) was also evaluated. The nail psoriasis severity index (NAPSI), a simple numeric tool for evaluating nail psoriasis, was used to quantify the degree of nail changes [11]. The NAPSI was assessed separately for each fingernail and toenail. Each nail is evaluated for the presence or absence of nail matrix disease (pitting, leukonychia, red spots in the lunula, and nail plate crumbling) and nail bed disease (oil drop/salmon patch discoloration, onycholysis, nail bed hyperkeratosis, and splinter haemorrhage). The sum of the scores for all of the nails constitutes the patient's NAPSI. Nail involvement was said to be present in patients with a NAPSI score ≥ 1. The disease-specific characteristics (age of onset, duration of psoriasis, mean PASI, mean PSI, mean NAPSI, and number of patients with nail involvement) of the psoriasis patients are summarised in Table 1. Most of the patients had mild psoriasis and received local treatment.

Echocardiographic evaluation

All echocardiographic examinations were performed using a Philips EnVisor C echocardiography platform (Philips Medical Systems, Andover, MA, USA) and a 3.5-MHz transducer. All patients were examined in the left lateral and supine positions using precordial M-mode, two-dimensional, Doppler, and tissue Doppler echocardiography. Patients with diastolic dysfunction were excluded from the study. One-lead electrocardiogram (ECG) was recorded continuously during echocardiography. Data were recorded from the average of three cardiac cycles. The left ventricular end-diastolic (LVED), left ventricular end-systolic (LVES), and left atrial end-systolic diameters were measured from M-mode in the parasternal long-axis views. The left ventricular ejection fraction (LVEF) was measured using the Teichholz formula. Left atrial volume was measured by tracing the maximum volume of the left atrium during systole in the

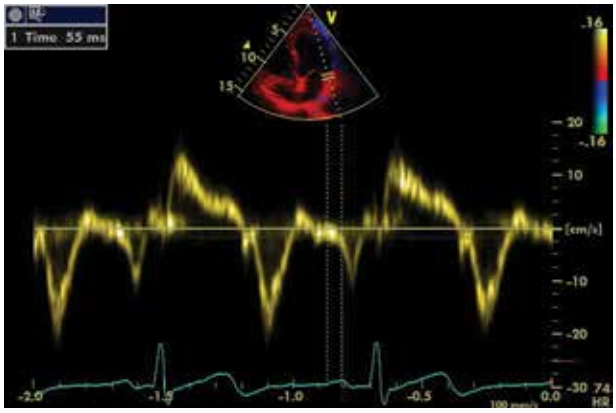

Figure 1. Measurement of the PA interval with tissue Doppler imaging, which defined the time interval from the onset of P wave on the surface electrocardiogram to the beginning of the atrial contraction A wave

apical four-chamber view. Tissue Doppler echocardiography was performed at transducer frequencies of 3.5 MHz, adjusting the spectral pulsed Doppler signal filters to a Nyquist limit of 15–20 cm/s and using the minimal optimal gain. The monitor sweep speed was set at 100 mm/s to optimise the spectral display of the myocardial velocities. The pulsed Doppler sample volume was placed in the middle segment of the lateral left and right atrial walls and in the interatrial septum just above the oval fossa in the apical four-chamber view. Time intervals from the onset of the P wave on the surface ECG to the beginning of the A wave (PA) representing EMD were obtained from the right atrial lateral wall and left atrial lateral and septal walls and named PA right, PA lateral, and PA septal, respectively. The measured PA intervals are shown in Figure 1. The timing of mechanical activation of each reference point depends on the distances from these points to the sinus node.

- The difference between PA lateral and PA septal was defined as the intra-left atrial electromechanical delay (ILA-EMD).
- The difference between PA right and PA septal was defined as the intra-right atrial electromechanical delay (IRA-EMD).
- The difference between PA lateral and PA right was defined as the inter-atrial electromechanical delay (IA-EMD).

Statistical analysis

Continuous variables were expressed as mean ± standard deviation and categorical variables as percentages. The categorical and continuous variables between the two groups were compared using the χ^2 test and Student's t-test, respectively. Correlations between atrial conduction time variables and clinical variables were computed using the Pearson correlation test. The potential interaction between psoriasis and obesity, as well as the potential effect of medications on the intra- and interatrial conduction times were examined using univariate analysis. A p-value < 0.05 was considered statistically significant. SPSS 17.0 for Windows was used for the statistical analysis.

RESULTS

The demographic characteristics of the groups are summarised in Table 2. There were no significant differences in terms of age, gender, cigarette smoking, family history of CAD, or BMI. Although the psoriasis patients and controls had similar BMI values, more subjects within the psoriasis group were obese (29.9% vs. 10.6%, p = 0.012). The mean systolic and diastolic blood pressures and heart rate were similar between groups. Biochemical data, including serum glucose, low-density lipoprotein cholesterol, and triglyceride levels, were not significantly different between the

Table 2. Demographic characteristics of the study population

	Group 1 (psoriatic; n = 85)	Group 2 (control; n = 46)	P
Age [years]	34.5 ± 6.5	34.9 ± 11.3	0.54
Gender (female/male)	39/46	26/20	0.24
Current smoker	37.6% (32/85)	41.3% (19/46)	0.47
Body mass index [kg/m^2]	26.0 ± 4.5	24.8 ± 3.4	0.13
Office systolic BP [mm Hg]	114.6 ± 13.2	117.5 ± 9.8	0.19
Office diastolic BP [mm Hg]	68.6 ± 11.4	71.9 ± 8.2	0.08
Heart rate [bpm]	73.8 ± 10.9	76.1 ± 8.1	0.23
Fasting glucose [mg/dL]	90.5 ± 10.3	88.6 ± 8.3	0.4
Serum LDL-C [mg/dL]	104.6 ± 29.3	107.7 ± 25.2	NS
Serum triglycerides [mg/dL]	123.4 ± 51.4	121.6 ± 55.1	NS
hsCRP [mg/L]	1.5 ± 0.3	0.3 ± 0.3	< 0.001

Data were presented as mean ± standard deviation; BP — blood pressure; LDL-C — low-density lipoprotein cholesterol; hsCRP — high sensitive C-reactive protein; NS — statistically non-significant

Table 3. Echocardiographic measurements of the study population

	Group 1 (psoriatic; n = 85)	Group 2 (control; n = 46)	P
Two-dimensional echocardiography			
LVESD [cm]	3.9 ± 0.8	3.7 ± 0.5	NS
LVEDD [cm]	4.6 ± 0.5	4.4 ± 0.6	NS
LV ejection fraction [%]	64.6 ± 4.2	65.2 ± 4.6	NS
LA [cm^3]	32.8 ± 7.5	31.1 ± 7.6	NS
Doppler echocardiography			
E [cm/s]	79.7 ± 14.8	80.2 ± 16.2	NS
A [cm/s]	59.1 ± 12.3	60.4 ± 13.0	NS
EDT [ms]	166.8 ± 41.4	163.7 ± 38.9	NS
IVRT [ms]	86.3 ± 12.5	84.6 ± 14.9	NS
Tissue Doppler echocardiography			
PA lateral [ms]	107.54 ± 11.25	77.43 ± 13.82	< 0.001
PA septum [ms]	88.47 ± 13.58	65.08 ± 13.57	< 0.001
PA right [ms]	76.08 ± 12.86	52.60 ± 11.72	< 0.001
ILA-CT	19.07 ± 8.98	12.34 ± 7.16	< 0.001
IRA-CT	12.38 ± 13.87	12.47 ± 14.27	0.97
IA-CT	31.45 ± 14.95	24.82 ± 14.45	0.01

Data were presented as mean ± standard deviation; LVESD — left ventricular end-systolic diameter; LVEDD — left ventricular end-diastolic diameter; LV — left ventricular; LA — left atrial volume; E — peak mitral valve flow velocity during early rapid filling phase; A — peak mitral valve flow velocity during atrial contraction; EDT — deceleration time of early phase of mitral valve flow; IVRT — isovolumetric relaxation time; ILA-CT — intra-left atrial conduction time; IRA-CT — intra-right atrial conduction time; IA-CT — inter-atrial conduction time; NS — statistically non-significant

groups. Echocardiographic variables, including the LVED and LVSD diameters and left atrial volume, were similar between groups. The LVEF and diastolic function variables were also similar between the two groups. In tissue Doppler echocardiographic examination, PA right duration was similar, while PA lateral and PA septal differed significantly between the two groups. IA-EMD (PA lateral–PA right) and ILA-EMD (PA lateral–PA septum) were significantly longer in the psoriasis group compared with the control group (Table 3). No relationship was found between obesity and the atrial conduction times, and the presence of obesity had no significant interaction with psoriasis with respect to IA-EMD and ILA-EMD (p for interaction 0.076 for ILA-EMD and 0.33 for IA-EMD). In addition, the medications used by the psoriasis patients did not have a significant effect on IA-EMD or ILA-EMD, while tumour necrosis factor alpha (TNF-α) blockers significantly affected IRA-EMD ($p = 0.018$, no significant interaction with other drugs). However, IRA-EMD was not significantly different between the groups, even when the patients on TNF-α therapy were excluded ($p = 0.579$). Correlation analysis between parameters of psoriasis severity (PASI score) and atrial conduction revealed a significant positive correlation between PASI and IA-EMD ($r = 0.261$, $p < 0.001$; Fig. 2). In addition, there was a positive correlation between hsCRP and IA-EMD ($p = 0.022$). The intra- and inter-observer variabilities for conventional Doppler- and tissue Doppler imaging (TDI)-derived variables (PA lateral, PA septal, and PA tricuspid) ranged from 3% to 7%.

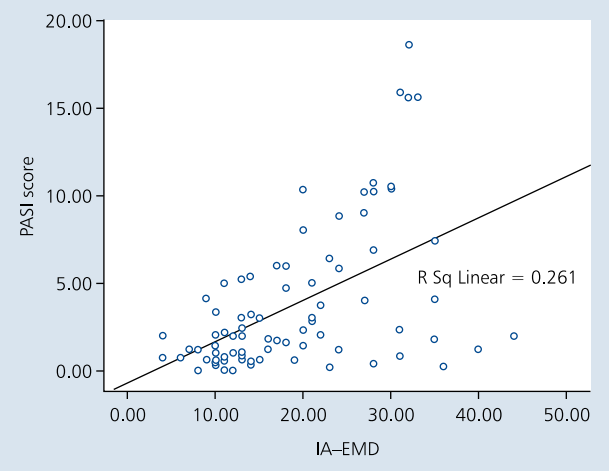

Figure 2. Scatter plot of the psoriasis area and severity index (PASI) score against inter-atrial electromechanical delay (IA-EMD) [ms] ($r = 0.256$, $p = 0.005$)

DISCUSSION

This study investigated the atrial conduction times in patients with psoriasis vulgaris using TDI. The major finding was that the intra-atrial and left atrial conduction times were prolonged

in these patients. We also found correlations between IA-EMD with both the PASI score and hsCRP levels, which suggests that disease activity and the severity of the inflammatory reaction contributed to the altered intra-atrial conduction.

Psoriasis vulgaris is a chronic inflammatory skin disease affecting nearly 3% of the global population or roughly 125 million people worldwide [12]. The skin lesions in psoriasis are characterised by epidermal hyperproliferation, abnormal keratinocyte differentiation, T-lymphocyte infiltration, and increased cytokine expression [1]. Inflammatory cytokines such as TNF-α, interferon gamma, and interleukin 2 are important factors in lesion development [13]. In addition to skin or joint involvement, these patients are prone to accelerated atherosclerosis, with an increase in CV complications [4, 12, 14–17]. Therefore, psoriasis can be regarded as a chronic systemic inflammatory disorder, such as rheumatoid arthritis and systemic lupus erythematosus, rather than an isolated skin disease [1].

Diverse aetiological factors can lead to development of AF in patients; these factors include genetic susceptibility [18], diseases associated with pressure or volume overload in the ventricles [19], CAD [20], and cardiac toxins [21, 22]. In addition, recent studies have shown an association between chronic inflammation and AF. Patients with AF show increased blood CRP levels [23] and infiltration of the atrial myocardium with inflammatory cells [24]. Interestingly, Ahlehoff et al. [4] recently identified psoriasis as a potential causative factor for AF, and the incidence of AF correlated with disease severity. This increased incidence of AF might also partly explain the increased tendency for stroke in psoriasis patients [4, 25–28].

Measures of electromechanical heterogeneity across the atria during depolarisation are indicators of an increased risk for AF [7]. Recently, Bacaksiz et al. [29] showed that patients with psoriasis vulgaris had greater P wave dispersion, indicating increased electrical heterogeneity and an increased risk for AF. The time delay between electrical activation of the atria (the beginning of the P wave) and mechanical activation (initiation of tissue Doppler signals) was previously established as a method to calculate the atrial EMD [30]. In addition, TDI-based measurements of the atrial EMD correlate with P wave dispersion [8]. This abnormal atrial conductivity in psoriasis patients, as demonstrated by this study and previous reports, could explain the increased incidence of AF in these patients [29]. Measuring the atrial conduction time could be useful for estimating the risk of AF in psoriasis patients, since such patients are usually young and lack conventional AF risk factors [4]. Although our results did not directly demonstrate an increased AF risk in psoriatic patients with prolonged atrial conduction time, it is reasonable to investigate asymptomatic AF episodes in these patients considering the potential consequences of unrecognised AF.

Although there is still no direct evidence, we believe that chronic inflammation is the best potential explanation linking psoriasis vulgaris with increased AF incidence and the abnormal conductivity patterns in the atrial myocardium. Chronic inflammation is considered as the pathophysiological basis that causes increased CV morbidity, including premature atherosclerosis, in psoriatic patients [31]. Some of our findings also suggest inflammation as the potential mechanism for abnormal atrial EMD. We observed that the degree of impaired left atrial conductivity correlated positively with the PASI score. Higher PASI scores indicated more severe psoriasis, and previous observations suggest that the PASI score correlated with serum levels of systemic inflammatory cytokines, such as TNF-α and interleukin 6 [32]. A positive correlation between hsCRP levels and IA-EMD was also noted in the present study ($p = 0.022$). Although these findings suggest that the degree of abnormality in atrial conductivity increases with the degree of inflammation, our results do not imply causality, and further studies are needed to resolve this subject.

Limitations of the study

A major limitation of this study was its cross-sectional design. In addition, since all of the patients were in sinus rhythm during the study, we did not perform a Holter examination to investigate the presence of atrial arrhythmias. In other words, the value of a prolonged EMD in predicting future arrhythmic events in patients with psoriasis has not been evaluated. Moreover, we do not have data from before and after therapy initiation to determine whether this treatment affects the atrial conduction time. Although we found an association with disease severity and the degree of increase in atrial conduction times, the majority of patients had a PASI score less than 10 and were therefore classified as having mild disease. For these reasons, large-scale, long-term follow-up prospective studies, preferably including patients with severe disease, are required to establish the predictive value of atrial conduction variables for the development of AF in patients with psoriasis.

CONCLUSIONS

The intra- and interatrial EMD were longer in patients with psoriasis vulgaris compared with the controls. We also demonstrated that the inter-atrial EMD was significantly correlated with the disease activity score (PASI) and hsCRP. These results suggest that prolongation of the atrial conduction time might help explain the increased AF incidence in these patients, and inflammatory processes might be the cause of this increase. Further long-term prospective studies are needed to clarify the clinical utility and prognostic importance of EMD in patients with psoriasis vulgaris.

Conflict of interest: *none declared*

References

1. Schön MP, Boehncke WH. Psoriasis. N Engl J Med, 2005; 352: 1899–1912.
2. Balta I, Balta S, Demirkol S, et al. Aortic arterial stiffness is a moderate predictor of cardiovascular disease in patients with psoriasis vulgaris. Angiology, 2014; 65: 74–78.
3. Mehta NN, Yu Y, Pinnelas R et al. Attributable risk estimate of severe psoriasis on major cardiovascular events. Am J Med, 2011; 124: 775.e1–e6.
4. Ahlehoff O, Gislason GH, Jørgensen CH, et al. Psoriasis and risk of atrial fibrillation and ischaemic stroke: a Danish Nationwide Cohort Study. Eur Heart J, 2012; 33: 2054–2064.
5. Turpie AG. Rivaroxaban as an oral anticoagulant for stroke prevention in atrial fibrillation. Ther Clin Risk Manag, 2014; 10: 197–205. eCollection 2014. Review.
6. Güvenç TS, Ilhan E, Hasdemir H et al. A novel explanation for the cause of atrial fibrillation seen in atherosclerotic coronary artery disease: "downstream inflammation" hypothesis. Med Hypotheses, 2010; 74: 665–667.
7. Perzanowski C, Ho AT, Jacobson AK. Increased P-wave dispersion predicts recurrent atrial fibrillation after cardioversion. J Electrocardiol, 2005; 38: 43–46.
8. Özer 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. Louden BA, Pearce DJ, Lang W et al. A Simplified Psoriasis Area Severity Index (SPASI) for rating psoriasis severity in clinic patients. Dermatol Online J, 2004; 10: 7.
10. Amornpinyokeit N, Asawanonda P. 8-Methoxypsoralen cream plus targeted narrowband ultraviolet B for psoriasis. Photodermatol Photoimmunol Photomed, 2006; 22: 285–289.
11. Rich P, Scher RK. Nail Psoriasis Severity Index: a useful tool for evaluation of nail psoriasis. J Am Acad Dermatol, 2003; 49: 206–212.
12. Prodanovich S, Kirsner RS, Kravetz JD et al. Association of psoriasis with coronary artery, cerebrovascular, and peripheral vascular diseases and mortality. Arch Dermatol, 2009; 145: 700–703.
13. Elder JT, Bruce AT, Gudjonsson JE et al. Molecular dissection of psoriasis: integrating genetics and biology. J Invest Dermatol, 2010; 130: 1213–1226.
14. Rico T, Marchione R, Kirsner RS. Vascular disease in psoriasis. J Invest Dermatol, 2009; 129: 2327.
15. Flammer AJ, Ruschitzka F. Psoriasis and atherosclerosis: two plaques, one syndrome? Eur Heart J, 2012; 33: 1989–1991.
16. Patel RV, Shelling ML, Prodanovich S et al. Psoriasis and vascular disease–risk factors and outcomes: a systematic review of the literature. J Gen Intern Med, 2011; 26: 1036–1049.
17. Daudén E, Castañeda S, Suárez C et al. [Integrated approach to comorbidity in patients with psoriasis]. Actas Dermosifiliogr, 2012; 103: 1–64.
18. Darbar D, Herron KJ, Ballew JD et al. Familial atrial fibrillation is a genetically heterogeneous disorder. J Am Coll Cardiol, 2003; 41: 2185–2192.
19. Krahn AD, Manfreda J, Tate RB et al. The natural history of atrial fibrillation: incidence, risk factors, and prognosis in the Manitoba Follow-Up Study. Am J Med, 1995; 98: 476–484.
20. Liberthson RR, Salisbury KW, Hutter AM Jr et al. Atrial tachyarrhythmias in acute myocardial infarction. Am J Med, 1976; 60: 956–960.
21. Ettinger PO, Wu CF, De La Cruz C Jr. et al. Arrhythmias and the "Holiday Heart": alcohol-associated cardiac rhythm disorders. Am Heart J, 1978; 95: 555–562.
22. Woeber KA. Thyrotoxicosis and the heart. N Engl J Med, 1992; 327: 94–98.
23. Yao SY, Chu JM, Chen KP et al. Inflammation in lone atrial fibrillation. Clin Cardiol, 2009; 32: 94–98.
24. Chen MC, Chang JP, Liu WH et al. Increased inflammatory cell infiltration in the atrial myocardium of patients with atrial fibrillation. Am J Cardiol, 2008; 102: 861–865.
25. Gelfand JM, Neimann AL, Shin DB et al. Risk of myocardial infarction in patients with psoriasis. JAMA, 2006; 296: 1735–1741.
26. Gelfand JM, Dommasch ED, Shin DB et al. The risk of stroke in patients with psoriasis. J Invest Dermatol, 2009; 129: 2411–2418.
27. Brauchli YB, Jick SS, Miret M et al. Psoriasis and risk of incident myocardial infarction, stroke or transient ischaemic attack: an inception cohort study with a nested case–control analysis. Br J Dermatol, 2009; 160: 1048–1056.
28. Kimball AB, Guerin A, Latremouille-Viau D et al. Coronary heart disease and stroke risk in patients with psoriasis: retrospective analysis. Am J Med, 2010; 123: 350–357.
29. Bacaksiz A, Erdogan E, Tasal A et al. Electrocardiographic P–wave characteristics in patients with psoriasis vulgaris. Ups J Med Sci, 2013; 118: 35–41.
30. 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.
31. Coumbe AG, Pritzker MR, Duprez DA. Cardiovascular risk and psoriasis: beyond the traditional risk factors. Am J Med, 2014; 127: 12–18.
32. Shimizu A, Fukatani M, Tanigawa M et al. Intra-atrial conduction delay and fragmented atrial activity in patients with paroxysmal atrial fibrillation. Jpn Circ J, 1989; 53: 1023–1030.

Zaburzenia przewodzenia przedsionkowego u chorych na łuszczycę zwyczajną

Abdurrahman Tasal, Tolga Sinan Guvenc, Seref Kul, Ahmet Bacaksiz, Ercan Erdogan, Osman Sonmez, Emrah Sevgili, Didem Dizman, Nahide Onsun, Nuray K. Ay

Department of Cardiology, Bezmialem Vakif University, Istanbul, Turcja

Streszczenie

Wstęp: Łuszczyca zwyczajna jest jedną z najczęstszych przewlekłych zapalnych chorób skóry. U pacjentów z łuszczycą występuje ryzyko migotania przedsionków (AF). Opóźnienie elektromechaniczne (EMD) to odstęp czasowy od początku załamka P w elektrokardiogramie (EKG) powierzchniowym do początku załamka A.

Cel: W niniejszym badaniu oceniono wewnątrz- i międzyprzedsionkowe EMD u chorych na łuszczycę zwyczajną.

Metody: Do badania włączono 85 dorosłych pacjentów z łuszczycą zwyczajną (Grupa 1) oraz 46 dopasowanych pod względem wieku i płci zdrowych osób (Grupa 2). U wszystkich uczestników wykonano badanie EKG I obliczono przedsionkowe zmienne EMD. Wyniki przedstawiono jako średnie ± odchylenie standardowe i wartości procentowe. Do analizy zmiennych ciągłych zastosowano test t Studenta. Wartość $p < 0,05$ przyjęto za istotną statystycznie.

Wyniki: Międzyprzedsionkowe opóźnienie elektromechaniczne (IA-EMD) i opóźnienie elektromechaniczne w obrębie lewego przedsionka (ILA-EMD) były istotnie dłuższe w grupie chorych na łuszczycę niż w grupie kontrolnej. Analiza korelacji między stopniem ciężkości łuszczycy (skala PASI) a parametrami przewodzenia przedsionkowego wykazała istotną dodatnią korelację między PASI a IA-EMD ($r = 0,261$; $p < 0,001$). Ponadto stwierdzono dodatnią korelację między stężeniem białka C oznaczanego metodą wysokoczułą (hsCRP) a IA-EMD ($p = 0,022$).

Wnioski: U pacjentów z łuszczycą zwyczajną czas przewodzenia przedsionkowego był dłuższy i korelował ze stopniem ciężkości choroby oraz stężeniem hsCRP. Z uwagi na to, że związek między opóźnieniem przewodzenia i AF jest znany, pomiar czasu przewodzenia wewnątrzprzedsionkowego mógłby być praktycznym narzędziem służącym do oceny ryzyka AF u tych chorych.

Słowa kluczowe: migotanie przedsionków, łuszczyca zwyczajna, opóźnienie elektromechaniczne

Kardiol Pol 2015; 73, 8: 637–643

Adres do korespondencji:
Dr Seref Kul, Bezmialem Vakif University, Department of Cardiology, Adnan Menderes Bulvari (Vatan caddesi) 34093, Fatih Istanbul, Turkey, e-mail: doktorseref@hotmail.com
Praca wpłynęła: 16.03.2014 r. **Zaakceptowana do druku:** 27.11.2014 r. **Data publikacji AoP:** 03.02.2015 r.