Kardiologia Polska 2016; 74, 3: 274–279; DOI: 10.5603/KP.a2015.0141 ISSN 0022–9032

ARTYKUŁ ORYGINALNY / *ORIGINAL ARTICLE*

Gender-dependent profile of heart rate turbulence parameters in patients after acute myocardial infarction treated invasively

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Abstract

Background and aim: The aim of this study was to determine whether gender is related to differences in heart rate turbulence (HRT) parameters and the authors' own predefined HRT categories comprising turbulence timing (TT) in patients at the early stage of acute myocardial infarction (AMI) treated invasively.

Methods: We analysed 489 consecutive patients (147 females and 342 males, aged 63.9 ± 11.7 years) with AMI admitted to our department and treated invasively on admission to the hospital. On the fifth day after MI 24-h digital Holter recordings were performed to assess HRT, and the following HRT parameters were calculated in all patients using the HRTView program: turbulence onset (TO, %), slope (TS, ms/RR interval), and TT. The following values of HRT parameters were considered abnormal: TO ≥ 0, TS ≤ 2.5, and TT ≥ 10. Based on the abovementioned parameters, the authors defined their own HRT categories (A, B, C): A — comprising three normal parameters, B — one abnormal parameter, C — three abnormal parameters.

Results: TT was significantly later in women than in men: 7.5 ± 3.1 vs. 6.8 ± 3.1 ($p < 0.05$), respectively. When analysing the authors' own predefined HRT categories, significant differences between women and men were present in the occurrence of the category C, including all three abnormal HRT parameters: TO, TS, and TT.

Conclusions: In women worse TT was present and all three abnormal HRT parameters occurred more frequently. HRT in women after AMI is profoundly altered compared to in men. This indicates greater autonomic dysfunction and higher risk for sudden cardiac death in women after AMI.

Key words: heart rate turbulence, women, acute myocardial infarction

Kardiol Pol 2016; 74, 3: 274–279

INTRODUCTION

The high-risk population, e.g. patients who survived sudden cardiac death (SCD) or those with severely reduced left ventricular ejection fraction (LVEF), is easily identifiable, while the majority of patients with myocardial infarction (MI) and intermediate risk of SCD are still a challenge for risk stratification. That is why there is still a clinical need to search for the optimal combination of SCD risk factors, especially in women who are at the highest risk of SCD after MI, in order to improve the prediction of long-term outcomes [1]. Although there are many defined risk stratification parameters, none of them meets all requirements (e.g. high specificity and sensitivity, high-positive predictive value, as well as non-invasive easy to obtain, and cheap feasibility) to be the most appropriate. There is an obvious demand for developing a new risk stratification factors for fatal arrhythmias in post-MI patients who might be in the highest or moderate risk group for SCD. It seems that one of the most convenient and useful methods is the heart rate turbulence (HRT) pattern, which may predict cardiac mortality and identify patients at higher risk for cardiovascular death [2, 3].

The prognostic value of HRT parameters and their combinations in the prediction of total mortality, cardiac mortality, SCD, and fatal arrhythmic events in patients after

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Received: 15.07.2014 **Accepted:** 29.06.2015 **Available as AoP:** 16.07.2015

MI is well-documented [2, 4–10]. In many published studies, HRT was investigated only in terms of such parameters (as their combinations) as turbulence onset (TO) or turbulence slope (TS), and very little attention in the literature has been paid to evaluate turbulence timing (TT) [5, 11, 12].

Although there are well-known risk predictors of major adverse cardiac events (MACE) in the post-MI population (e.g. low LVEF, QRS duration, advanced age, arrhythmias), the analysis of HRT is also a powerful predictor of mortality and MACE in patients after MI [2, 4–6, 13].

According to the study previously provided by us, the category encompassing three abnormal HRT parameters (TO, TS, TT) best distinguished patients at risk of MACE after MI, and TT was a strong independent risk factor for those events [5].

Despite the fact that many studies confirmed the significance and prognostic value of these parameters related to baroreflex sensitivity [14] and describing the acceleration and subsequent deceleration of sinus rate after a single ventricular premature beat [8], these factors have not been fully elucidated with respect to gender.

That is why the aim of the study was to illustrate gender-specific differences of HRT parameters in the population of patients at the early stage of MI treated invasively.

METHODS

Study population

The study was retrospective and did not need approval of the Local Ethics Committee. The study group comprised 489 consecutive patients (147 females and 342 males, aged 63.9 ± 11.7 years) with acute MI (AMI) admitted to the Silesian Centre for Heart Diseases, Zabrze, Poland in 2004–2007. All patients were treated with percutaneous coronary intervention (PCI) on admission to the hospital.

The inclusion criteria were as follows: MI confirmed by symptoms, electrocardiographic (ECG) changes, and laboratory results (i.e. elevated biomarkers of myocardial necrosis such as troponin and/or creatine phosphokinase-MB isoenzyme), invasive treatment of AMI, survival of at least five days after MI, sinus rhythm during 24-h digital Holter recordings, lack of indications to atrio-ventricular pacing, and accomplished rehabilitation protocol. Patients aged ≥ 80 years, who died before the 24-h Holter monitoring, with atrial fibrillation (either sustained or paroxysmal), or with other non-sinus rhythms that persisted to the end of in-hospital rehabilitation, were excluded from the study. On the fifth day after MI obligatory 24-h digital Holter recordings were carried out with the use of Pathfinder 700 Spacelabs Healthcare.

Patients were qualified to PCI according to the current guidelines of the European Society of Cardiology (ESC) [15, 16].

Patients with confirmed AMI on admission to the hospital were treated as follows: acetylsalicylic acid (ASA) in a dose of 150–325 mg p.o., clopidogrel in a dose of 300–600 mg p.o., and i.v. bolus of unfractioned heparin in a dose of 100 U/kg. Afterwards, they had coronary artery angiograms performed and blood flow through coronary arteries assessed in the Thrombolysis in Myocardial Infarction scale, based on which the decision about further PCI was made. After PCI of the infarct-related artery the patient was monitored on the Intensive Cardiological Care Unit and treated with optimal pharmacotherapy including beta-adrenergic receptor antagonists, angiotensin-converting enzyme inhibitors/angiotensin receptor blockers, ASA, clopidogrel, loop diuretics, spironolactone, and statins.

The profile of patients admitted to hospital is shown in Table 1.

Analysis of HRT

Routine 24-h Holter recordings were performed on the 4–5th day after MI in all patients admitted to the clinic (Pathfinder 700 Spacelabs Medical inc., Hertford, UK) to assess HRT. In order to evaluate HRT sinus rhythm in the routine ECG as well as leading sinus rhythm and the presence of ≥ 5 premature ventricular beats (PVB) during ECG, Holter monitoring was needed [2, 13].

Using the HRTView program the following HRT parameters were calculated:

- A — turbulence onset (TO) defined as the shortening of RR intervals after a compensatory pause. TO is the difference between the sum of two sinus RR intervals immediately following the PVB compensatory pause and the sum of two sinus RR intervals immediately preceding the PVB coupling interval, finally divided by the sum of two sinus RR intervals immediately preceding the coupling interval [8, 17, 18].
- B — the turbulence slope (TS) shows how fast the RR intervals change after the compensatory pause. TS is defined as the maximum positive regression slope assessed over any five consecutive sinus RR intervals within the first 15–20 RR intervals after PVB [8, 17, 19–21].
- C — turbulence timing (TT) defined as the beginning of RR intervals used to calculate TS. It has been reported that TT lies within the first 10 RR intervals after PVB, so TT ≥ 10 was accepted as abnormal [11, 13].

TO ≥ 0% and TS ≤ 2.5 ms/RR interval were considered abnormal and correlated with higher risk of SCD in patients after MI.

Based on the abovementioned parameters the following authors' own HRT categories (A, B, C) were defined: A — comprising three normal (TO, TS, TT normal), B — one abnormal (TO or TS or TT abnormal), and C — three abnormal parameters (TO, TS, and TT abnormal).

To avoid artefacts or inappropriate classification of T-wave as a PVB, we used filters that excluded from HRT analysis RR intervals that fulfilled the baseline criteria defined below:

- RR intervals shorter than 300 ms;
- RR intervals longer than 2000 ms;

Table 1. The profile of patients admitted to the hospital

	Women (n = 147)	Men (n = 342)	P
Age [years]	63 ± 11	59 ± 10	0.65
Hypertension	96 (65.3%)	162 (47.4%)*	< 0.05
Hyperlipidaemia	83 (56.5%)	161 (47.1%)	0.057
Type 2 diabetes mellitus	57 (38.8%)	81 (23.7%)*	< 0.05
Smokers	70 (47.6%)	217 (63.5%)*	< 0.05
MI (anterior wall)	67 (45.6%)	143 (41.8%)	0.44
MI (inferior wall)	43 (29.3%)	124 (36.3%)	0.13
Non-ST-elevation MI	30 (20.4%)	55 (16.1%)	0.24
LVEF 5 days after MI	45 ± 7	44 ± 7	0.94
TIMI before PCI < 3	127 (86.4%)	309 (90.3%)	0.19
TIMI after PCI < 3	19 (12.9%)	40 (11.6%)	0.70
Cardiogenic shock during admission	4 (2.7%)	15 (4.4%)	0.38
Total mortality during 30-month follow-up	11 (7.5%)	27 (7.9%)	0.87

*p < 0.05 between men and women; MI — myocardial infarction; LVEF — left ventricular ejection fraction; PCI — percutaneous coronary interventions; TIMI — Thrombosis In Myocardial Infarction

— > 200 ms difference when comparing to a previous sinus beat;
— > 20% difference when comparing to a reference interval (the mean value of five last sinus beats).

Statistical analysis

All parameters were expressed as means ± standard deviation. The Kruskal-Wallis test was used to evaluate the differences in HRT parameters between groups, while the qualitative data analysis was performed using the Pearson χ^2 test and results were shown as percentages. Independent predictors of death were identified in the multivariate Cox-regression model and expressed as hazard ratios (HR) with 95% confidence intervals (CI). Regression models were developed after the inclusion of all parameters with univariate association with all-cause mortality, and then the backward stepwise variable selection method was applied. Two similar models were constructed: model 1 with abnormal TO, TS, and TT and model 2 — substituted by the category C. The other covariates for all-cause mortality adjustment included into both models were: male gender, cardiogenic shock on admission to hospital, hypertension, diabetes mellitus, smoking, incomplete revascularisation, glomerular filtration rate (GFR) < 60 mL/min/1.73 m^2, advanced age over 70 years, multivessel coronary artery disease, and LVEF < 35%.

Logistic regression was used to select clinical parameters independently associated with abnormal TO, TS, and TT and finally with category C.

All tests were double-sided. The p value < 0.05 was considered statistically significant. All analyses were performed using the software package Statistica (version 6.1, StatSoft Inc., Tulsa, OK, USA).

RESULTS

Within 30.8 ± 15.0 months of follow-up 38 (7%) patients died. Clinical characteristics of patients are presented in Table 1. Hypertension and diabetes mellitus type 2 were significantly more frequent in women, while smokers were more frequent among men. Hyperlipidaemia was more often observed in women than in men, although statistically insignificant. There was no difference in the frequency of ST elevation myocardial infarction (STEMI) and non-STEMI between groups divided according to the gender. In the group of women, the most frequent electrocardiographic localisation was the anterior wall, while in the group of men it was the inferior wall. In both localisations of MI there were no important differences between the examined groups.

Comparison of HRT between women and men

HRT parameters in all patients were as follows: mean TO –0.7 ± 2.3%, mean TS 6.1 ± 6.4 ms/RR interval, mean TT 7.1 ± 3.1. Abnormal value of TO (≥ 0%) was observed in 184 patients, TS (≤ 2.5 ms/RRinterval) in 155 patients and TT (≥ 10) in 140 patients.

TT was significantly later in women than in men: 7.5 ± 3.1 vs. 6.8 ± 3.1 (p < 0.05), respectively. Among the remaining HRT parameters no significant gender-related differences were noticed: TO (–0.6 ± 2.7 vs. –0.7 ± 2.4%, p = NS) and TS (5.6 ± 5.6 vs. 6.4 ± 6.7 ms/RR interval, p = NS). When analysing the authors' own predefined HRT categories, significant differences between women and men were present in the occurrence of category C including all three abnormal HRT parameters: TO, TS, and TT. The percentage of women with this category was significantly greater than the percentage of men (Table 2). However, the logistic

Table 2. Comparison of heart rate turbulence (HRT) between women and men

HRT	Women (n = 147)	Men (n = 342)	P
Mean TO [%]	–0.6 ± 2.7	–0.7 ± 2.4	0.27
Mean TS [ms/RR interval]	5.6 ± 5.6	6.4 ± 6.7	0.39
Mean TT	7.5 ± 3.1	6.9 ± 3.1	< 0.05
TO > 0%	62 (42.2%)	122 (35.6%)	0.17
TS ≤ 2.5 ms/RR interval	44 (29.9%)	111 (32.5%)	0.58
TT ≥ 10	47 (31.9%)	93 (27.2%)	0.28
TO, TS, and TT normal	51 (34.7%)	135 (39.5%)	0.32
TO, TS, or TT abnormal	54 (36.7%)	106 (31%)	0.21
TO, TS, and TT abnormal	18 (12.2%)	19 (5.6%)	0.01

TO — turbulence onset; TS — turbulence slope; TT — turbulence timing

regression analysis did not show that gender is an independent risk factor for abnormal TO, TS, or TT. Nevertheless, it was proven that mean heart rate is an independent risk factor for abnormal TT, TS, and category C.

The assessment of independent risk factors of death

The independent risk factors of death in the studied population were: cardiogenic shock on admission to hospital — HR 16.16 (95% CI 7.466–34.976, $p < 0.05$), hypertension — HR 2.2 (95% CI 1.074–4.638, $p < 0.05$), GFR < 60 mL/min/1.73 m^2 — HR 2.33 (95% CI 1.184–4.604, $p < 0.05$), LVEF < 35% — HR 3.17 (95% CI 1.553–6.494, $p < 0.05$), abnormal TT (model 1) — HR 3.742 (95% CI 1.829–7.659, $p < 0.05$), and category C (model 2) — HR 2.193 (95% CI 1.020–4.715, $p < 0.05$). None of the above-mentioned parameters was gender-dependent (NS).

DISCUSSION

Previously, we have evaluated the utility of HRT as a risk factor in post-MI patients and showed that TT is significantly worse in patients with MACE than in patients without such events: (8.87 ± 3.08 vs. 6.57 ± 2.93, $p < 0.001$). We have revealed new evidence that three abnormal HRT parameters (including TT) best differentiated patients at risk of MACE after MI [5]. The most important finding of our current study in post-MI patients was the gender-related difference in the HRT category comprising all three abnormal HRT parameters (TS, TO, TT). According to our knowledge such a finding has not been reported so far. When we analysed only TO and TS we stated that there is no significant gender-related difference between women and men in these HRT parameters (TO: –0.6 ± 2.7 vs. –0.7 ± 2.4, p = NS and TS: 5.6 ± 5.6 vs. 6.4 ± 6.7 ms/RR interval, p = NS). Similar conclusions were presented in numerous previous studies in healthy and post-MI patients [4, 3, 12, 22–25]. Moreover, Schwab et al. [24] investigated gender-related differences in TO and TS as well as the correlation between these parameters and age or heart rate in 95 healthy individuals, and they did not reveal any gender-related difference in the abovementioned HRT parameters. Furthermore, the results of another study conducted by the same authors in healthy volunteers showed that there was no gender-related difference regarding TO or TS values. These authors reported that the abnormal TO was observed in 24% of the population (18% of women vs. 29% of men, p = 0.28), while the incidence of pathological TS was reported in 5% of patients (5% of women vs. 6% of men, p = 0.87) [12]. However, they confirmed that an increased heart rate before a ventricular premature contraction (VPC) was associated with a decreased TO ($p < 0.0001$), but again there was no significant difference between women and men (p = 0.28). Similarly, Cygankiewicz et al. [7] confirmed the lack of correlation between the gender and TS value; however, the authors showed that TS was useful in predicting total mortality in various groups of patients. Recently, Hoshida et al. [4] prospectively enrolled 313 consecutive post-MI HRT-positive patients (HRT positive when both TO and TS were abnormal) and they showed no significant gender-related differences between the positive and negative HRT groups (p = 0.47). What is more, during 1190 ± 441 days of follow-up they demonstrated that the HRT-positive outcome was the most significant predictor for the endpoint — defined as overall cardiac mortality and fatal arrhythmic events (HR 5.7, 95% CI 2.1–15.9, p = 0.0008) with no gender differences. It is also worth mentioning that Davies et al. [25] calculated baroreflex sensitivity in correlation to HRT and blood pressure in a group of 45 patients with congestive heart failure, and they showed no significant differences in HRT between women and men.

Furthermore, Jeron et al. [3] reported that the pathological TO (> 0%) and pathological TS (< 2.5 ms/RR) were calculated at 29.59% and 27.55%, respectively, in a group of 196 post-MI patients without any significant gender impact. Analogically, Li-na et al. [6] reported that the HRT-positive outcome (HR 5.01, 95% CI 1.33–18.85, p = 0.017) had significant asso-

ciation with the study endpoint, defined as cardiac mortality, while the impact of gender was not assessed in this study [6].

As was mentioned above, our present study focused on the relationship of gender and all three abnormal HRT parameters (including TT) in post-MI patients. We showed that worse TT and all three abnormal HRT parameters in women in comparison with men indicated greater autonomic dysfunction, and therefore women can be at higher risk of SCD after MI.

The differences in TT values for predicting total mortality in various groups of patients depending on gender are still unknown, which is why its predictive value and also pathophysiological thresholds are not clearly determined. Thus, further investigation in this matter is required. It seems reasonable to compare gender-related differences in TT values in the healthy population and post-MI patients, and in particular, to validate the additional risk stratification criterion (higher TT in women). The value of the suggested approach for comprehensive assessment of risk stratification also requires further confirmation in larger cohorts.

Our findings shed new light on the predictive role of gender-related differences in HRT parameters and their value as an additional factor for risk stratification in post-MI patients.

Limitations of the study

The performed analysis is retrospective. The methods used in this manuscript do not allow for such an analysis in patients without or with only few PVBs.

CONCLUSIONS

Independent risk factors of death in patients after MI are: cardiogenic shock, hypertension, renal dysfunction, LVEF < 35%, abnormal TT, and category C (all three abnormal HRT parameters). In women, worse TT was present, and all three abnormal HRT parameters occurred more frequently; however, there were no gender-related changes among these parameters. HRT in women after AMI is profoundly altered compared to men. This may indicate the greater autonomic dysfunction and higher risk for SCD in women after AMI.

Conflict of interest: *Beata Średniawa, MD, PhD, consultant Medtronic Bakken Research Centre, lectures fee Reynolds Medical Diagnostyka Kardiologiczna.*

References

1. Adabag AS, Therneau TM, Gersh BJ et al. Sudden death after myocardial infarction. JAMA, 2008; 300: 2022–2029. doi: 10.1001/jama.2008.553.
2. Bauer A, Malik M, Schmidt G et al. Heart rate turbulence: standards of measurement, physiological interpretation, and clinical use: International Society for Holter and Noninvasive Electrophysiology Consensus. J Am Coll Cardiol, 2008; 52: 1353–1365. doi: 10.1016/j.jacc.2008.07.041.
3. Jeron A, Kaiser T, Hengstenberg C et al. Association of the heart rate turbulence with classic risk stratification parameters in post-myocardial infarction patients. Ann Noninvasive Electrocardiol, 2003; 8: 296–301.
4. Hoshida K, Miwa Y, Miyakoshi M et al. Simultaneous assessment of T-wave alternans and heart rate turbulence on holter electrocardiograms as predictors for serious cardiac events in patients after myocardial infarction. Circ J, 2013; 77: 432–438.
5. Cebula S, Sredniawa B, Kowalczyk J et al. The significance of heart rate turbulence in predicting major cardiovascular events in patients after myocardial infarction treated invasively. Ann Noninvasive Electrocardiol, 2012; 17: 230–240. doi: 10.1111/j.1542-474X.2012.00510.x.
6. Li-na R, Xin-hui F, Li-dong R et al. Ambulatory ECG-based T-wave alternans and heart rate turbulence can predict cardiac mortality in patients with myocardial infarction with or without diabetes mellitus. Cardiovasc Diabetol, 2012; 11: 104.
7. Cygankiewicz I, Zareba W, Vazquez R et al. Heart rate turbulence predicts all-cause mortality and sudden death in congestive heart failure patients. Heart Rhythm, 2008; 5: 1095–1102. doi: 10.1016/j.hrthm.2008.04.017.
8. Schmidt G, Malik M, Barthel P et al. Heart-rate turbulence after ventricular premature beats as a predictor of mortality after acute myocardial infarction. Lancet, 1999; 353: 1390–1396.
9. Exner DV, Kavanagh KM, Slawnych MP et al. Noninvasive risk assessment early after a myocardial infarction the REFINE study. J Am Coll Cardiol, 2007; 50: 2275–2284.
10. Sulimov V, Okisheva E, Tsaregorodtsev D. Non-invasive risk stratification for sudden cardiac death by heart rate turbulence and microvolt T-wave alternans in patients after myocardial infarction. Europace, 2012; 14: 1786–1792. doi: 10.1093/europace/eus238.
11. Bonnemeier H, Wiegand UK, Friedlbinder J et al. Reflex cardiac activity in ischemia and reperfusion: Heart rate turbulence in patients undergoing direct percutaneous coronary intervention for acute myocardial infarction. Circulation, 2003; 108: 958–964.
12. Schwab JO, Eichner G, Veit G et al. Influence of basic heart rate and sex on heart rate turbulence in healthy subjects. Pacing Clin Electrophysiol, 2004; 27: 1625–1631.
13. Watanabe MA, Schmidt G. Heart rate turbulence: a 5-year review. Heart Rhythm, 2004; 1: 732–738.
14. Lin LY, Lai LP, Lin JL et al. Tight mechanism correlation between heart rate turbulence and baroreflex sensitivity: sequential autonomic blockade analysis. J Cardiovasc Electrophysiol, 2002; 13: 427–431.
15. Erhardt L, Herlitz J, Bossaert L et al. Task Force on the management of chest pain. Eur Heart J, 2002; 23: 1153–1176.
16. Patrono C, Bachmann F, Baigent C et al. The Task Force on the Use of Antiplatelet Agents in Patients with Atherosclerotic Cardiovascular Disease of the European Society of Cardiology. Eur Heart J, 2004; 25: 166–181.
17. Schneider R, Barthel P, Watanabe M. Heart rate turbulence on Holter. In: Malik M, Camm JA eds. Dynamic electrocardiography. Blackwell Futura Publishing, London 2004: 190–193.
18. Barthel P, Schneider R, Bauer A et al. Risk stratification after acute myocardial infarction by heart rate turbulence. Circulation, 2003; 108: 1221–1226.
19. Writing Committee of The Task Force. Heart rate variability. Standards of measurement, physiological interpretation, and clinical use. Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology. Eur Heart J, 1996; 17: 354–381.
20. Moore RK, Groves DG, Barlow PE et al. Heart rate turbulence and death due to cardiac decompensation in patients with chronic heart failure. Eur J Heart Fail, 2006; 8: 585–590.
21. Guzik P, Schmidt G. A phenomenon of heart-rate turbulence, its evaluation, and prognostic value. Card Electrophysiol Rev, 2002; 6: 256–261.
22. Lee KT, Lai WT, Chu CS et al. Effect of electrophysiologic character of ventricular premature beat on heart rate turbulence. J Electrocardiol, 2004; 37: 41–46.

23. Grimm W, Sharkova J, Christ M et al. Heart rate turbulence following ventricular premature beats in healthy controls. Ann Noninvasive Electrocardiol, 2003; 8: 127–131.
24. Schwab JO, Eichner G, Shlevkov N et al. Impact of age and basic heart rate on heart rate turbulence in healthy persons. Pacing Clin Electrophysiol, 2005; 28 (suppl. 1): S198–S201.
25. Davies LC, Francis DP, Ponikowski P et al. Relation of heart rate and blood pressure turbulence following premature ventricular complexes to baroreflex sensitivity in chronic congestive heart failure. Am J Cardiol, 2001; 87: 737–742.

Cite this article as: Średniawa B, Mitręga KA, Cebula S et al. Gender-dependent profile of heart rate turbulence parameters in patients after acute myocardial infarction treated invasively. Kardiol Pol, 2016; 74: 274–279. doi: 10.5603/KP.a2015.0141.

Różnice turbulencji rytmu zatokowego w zależności od płci chorych po zawale serca leczonych inwazyjnie

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Streszczenie

Wstęp: Doniesienia naukowe ostatnich lat dotyczące turbulencji rytmu zatokowego (HRT) potwierdziły znaczenie prognostyczne tego parametru związanego z odruchem z baroreceptorów także u chorych z zawałem serca (MI) leczonych nowoczesnymi metodami farmakologicznymi i przezskórną interwencją wieńcową. Dotychczas niewielu badaczy określało wartości HRT, wyodrębniając płcie.

Cel: Celem niniejszej pracy było określenie zależności między płcią a parametrami i kategoriami własnymi HRT u chorych z MI leczonych inwazyjnie przy przyjęciu.

Metody: Cyfrowe 24-godzinne monitorowanie holterowskie wykonano średnio w 5. dobie MI u 147 kobiet i 342 mężczyzn z MI (śr. wiek: 63,9 ± 11,7 roku; śr. frakcja wyrzutowa: 44,9 ± 7,7%). Za pomocą oprogramowania HRTView obliczono: początek (TO, %), nachylenie (TS, ms/odstęp RR) oraz pozycję (TT) turbulencji. TO ≥ 0%, TS ≤ 2,5 ms/odstęp RR i TT ≥ 10 przyjęto za nieprawidłowe, definiując na tej podstawie kategorie HRT własne (A, B, C) zawierające TO, TS i TT. Kategoria A obejmowała trzy prawidłowe parametry, B — jeden z trzech nieprawidłowy, C — trzy nieprawidłowe.

Wyniki: W grupie kobiet w porównaniu z grupą mężczyzn zaobserwowano istotnie późniejszy TT wynoszący odpowiednio 7,5 ± 3,1 i 6,8 ± 3,1 ($p < 0,05$). Obie grupy nie różniły się statystycznie między sobą w zakresie TO i TS, które u kobiet wynosiły: –0,58 ± 2,1 i 5,6 ± 5,6 (p = NS), a u mężczyzn odpowiednio: –0.7 ± 2,4 i 6,4 ± 6,7 (p = NS). Analizując kategorie własne, największe różnice w zależności od płci stwierdzono w przypadku kategorii C obejmującej trzy nieprawidłowe parametry HRT: TO, TS i TT. W grupie kobiet wystąpiła ona u 12,2%, a w grupie mężczyzn u 5,6% ($p < 0,05$).

Wnioski: Największe różnice w HRT w zależności od płci obserwuje się w kategorii obejmującej trzy nieprawidłowe parametry HRT, która występuje częściej u kobiet. Może to świadczyć o zwiększonym ryzyku zgonu po MI u kobiet z taką kategorią HRT.

Słowa kluczowe: turbulencja rytmu zatokowego, kobiety, zawał serca

Kardiol Pol 2016; 74, 3: 274–279

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Praca wpłynęła: 15.07.2014 r. **Zaakceptowana do druku:** 29.06.2015 r. **Data publikacji AoP:** 16.07.2015 r.