Kardiologia Polska 2013; 71, 1: 47–52 ISSN 0022–9032

ARTYKUŁ ORYGINALNY / *ORIGINAL ARTICLE*

Heart rate and double product in relation to insulin resistance in patients with hypertension and coronary artery disease

Wojciech Sobiczewski[1], Marcin Wirtwein[2], Edyta Kowalczys[1], Iwona Stopczyńska[1], Andrzej Koprowski[1], Marcin Gruchała[1], Andrzej Rynkiewicz[1]

[1]1st Department of Cardiology, Medical University of Gdansk, Poland
[2]Department of Pharmacology, Medical University of Gdansk, Poland

Abstract

Background: Elevated values of heart rate (HR) and insulin resistance (IR) reflect enhanced sympathetic nervous system activity and may be connected to the development of coronary artery disease (CAD) and diabetes.

Aim: To evaluate the relationship between HR, blood pressure (BP), double product and IR in nondiabetic hypertensive patients with stable CAD.

Methods: There were 73 patients included in the study. Ambulatory BP monitoring was recorded in all patients by a Spacelabs 90207 device. Homeostasis model assessment (HOMA-IR) was used to estimate IR. Double product was calculated by multiplying systolic BP and HR.

Results: In the study population (mean age 67.1 ± 8.4 years, 52% males) there was a positive correlation between HOMA-IR and 24-h double product ($r = 0.35$, $p < 0.01$) and body mass index (BMI) ($r = 0.45$, $p < 0.001$). The receiver operating characteristic analysis of 24-h double product and BMI as predictive markers of IR did not reveal statistical differences between AUC (0.72 ± 0.09 vs. 0.72 ± 0.08, 24-h double product and BMI, respectively, p = NS). The best cut-off points in predicting IR were 8,978 mm Hg/min for 24-h double product and 33.02 kg/m^2 for BMI. There were differences between the non obese (n = 44, mean age 67.9 ± 9.2 years) and obese (n = 29, mean age 65.8 ± 6.9 years) groups in: serum insulin level (7.3 ± 2.3 μU/mL vs. 12.0 ± 7.3 μU/mL, $p < 0.01$), HOMA-IR (1.8 ± 0.7 μU/mL × mmol/L vs. 3.0 ± 2.0 μU/mL × mmol/L, $p < 0.01$), and day systolic BP (128.0 ± 10.8 mm Hg vs. 134.1 ± 10.1 mm Hg, $p < 0.02$).

Conclusions: 24-h double product and BMI may be complementary parameters in the prediction of IR in hypertensive nondiabetics with CAD confirmed by percutaneous coronary interventions in history and/or at least one coronary artery stenosis ≥ 70% in elective coronary angiography.

Key words: double product, insulin resistance, coronary artery disease

Kardiol Pol 2013; 71, 1: 47–52

INTRODUCTION

Increased insulin resistance (IR) is defined as decreased biological response of nutrient to a given concentration of insulin at the target tissue [1]. In physiological conditions, autonomic nervous system activation influences on insulin release but permanent stimulation may lead to hyperinsulinaemia and IR. The gold standard of IR estimation is a hyperinsulinaemic-euglycaemic clamp: a technique that is a complicated invasive procedure used almost exclusively in experimental circumstances. But there is a more common and simpler clinically useful method: homeostasis model assessment (HOMA-IR) is based on fasting plasma glucose and insulin levels [2]. As many studies have revealed, data obtained from a hyperinsulinaemic-euglycaemic clamp correlates well with HOMA-IR.

Address for correspondence:
Marcin Wirtwein, MD, Department of Pharmacology, Medical University of Gdansk, ul. Dębowa 23, 80–211 Gdańsk, Poland, tel/fax: +48 58 349 25 42, e-mail: marwirt@interia.pl; wsob@gumed.edu.pl

Received: 25.03.2012 **Accepted:** 13.09.2012

Heart rate (HR) has been proposed as a simple global index of the autonomic nervous system influence on the heart. Elevated HR may reflect a shift in autonomic balance toward enhanced sympathetic and suppressed vagal tone [3]. In experimental and clinical conditions, it has been established that there is a relationship between insulin level and HR [4]. This association might suggest that there is also a relationship between IR and other markers of enhanced sympathetic tone such as high level of systolic blood pressure (SBP). The most valuable method of simultaneous estimation of both HR and SBP is 24-h blood pressure monitoring (ABPM). 24-h double product (DP) that contains both 24-h HR and 24-h SBP is a better haemodynamic parameter related to autonomic nervous system activation than 24-h HR and 24-h SBP separately, and it is a simple estimator of the heart oxygen consumption [5]. In patients with significant coronary atherosclerosis confirmed in coronary angiography, the influence of the autonomic nervous system on HR and DP may be impaired on the one hand due to decreased coronary blood supply but on the other hand by treatment with HR lowering drugs (e.g. beta-blockers, calcium channel blockers, ivabradine).

According to the World Health Organisation (WHO) definition, obesity is diagnosed when body mass index (BMI; the weight in kilograms divided by the height in metres squared) is greater than or equal to 30 kg/m^2 [2, 6, 7]. It is the commonest risk factor related to decreased IR that can be connected with high sympathetic nervous system activation in almost all obese subjects. Despite this association, it is clear that sympathetic activation per se cannot fully explain the development of IR in obese patients. The pathogenesis of IR among obese and nonobese subjects is different [8, 9].

The aim of our study was to assess the relationship between IR measured using a simple algorithm of HOMA-IR and 24-h DP in obese and nonobese patients with coronary artery disease (CAD) confirmed in coronary angiography.

METHODS

A total of 73 patients were enrolled in the study. The subjects were referred to the Outpatient Department of Cardiology, Medical University of Gdansk for 24-h ABPM performance. CAD was confirmed by percutaneous coronary interventions in history and/or at least one coronary artery stenosis ≥ 70% in elective coronary angiography. Subjects were diagnosed as nondiabetics according to European Association for the Study of Diabetes standards [10]. Subjects were diagnosed as hypertensive on the basis of the history of hypertensive treatment and ABPM or office blood pressure (BP) values in the hypertension range according to the 2007 ESH-ESC Practice Guidelines for the Management of Arterial Hypertension [11]. Antihypertensive and antianginal treatment were assigned by attending physicians.

ABPM was performed with Spacelabs 90207 (Spacelabs Inc., Richmond, WA, USA). The non-dominant arm was used for the measurement with cuff size adjusted to arm circumference (adult cuff 27–34 cm or large adult cuff 35–44 cm). Patients were instructed to maintain their usual activities but to refrain from strenuous exercise, and to hold the arm by their side during BP measurements. The monitors were programmed to obtain BP readings every 20 minutes between 06:00 and 22:00 and every 30 minutes between 22:00 and 06:00. Daytime ambulatory BP was defined as the period between 08:00 and 22:00 and nighttime as the period between 00:00 and 06:00. Mean 24-h daytime and nighttime values of BP and HR were analysed.

Office BP and HR measurements were performed with an OMRON 905 IT device (Omron Dualtec Automotive Electronics, Inc.). Office HR and BP were obtained two times at 5-minute intervals before ABPM. Mean office HR and BP values of two recordings were analysed.

Blood samples for fasting glucose and insulin level were collected in the morning before BP measurements. Plasma glucose was measured by the glucose oxidase method (Abbott). Plasma insulin was measured by IMx (Abbott) immunoassay. The homeostasis model assessment of IR (HOMA-IR) was used as the method for evaluating IR. HOMA-IR was defined as: fasting insulin (μU/mL) × fasting glucose (mmol/L)/22.5 [12]. According to Lebovitz's classification, subjects with HOMA-IR value ≥ 2.7 μU/mL × mmol/L were diagnosed as insulin resistant [13].

The study protocol was approved by the Ethics Committee of the Medical University of Gdansk.

Statistical analysis

STATISTICA 9.1 software system (Statsoft Inc., Tulsa, OK, USA) was used for analysis. The continuous values are expressed as mean ± standard deviation (SD). Normality of distributions was checked by Shapiro-Wilk test. Student's unpaired *t*- test, U-Mann-Whitney test or χ^2 test were used in the assessment of differences between two groups, as appropriate. The relationships between variables were estimated by Spearman correlation analysis. To explore the relationship between BMI, HR, DP as independent variables and HOMA-IR as dependent variable, the method of linear regression analysis was used. Analysis of the receiver operating characteristic (ROC) was used for predicting IR among study subjects. To define an optimal 24-h DP, HR and BMI cutoff, we computed and searched for the shortest distance on ROC curves. On the basis of the ROC curves, we estimated the ROC area under the curve (AUC) that measured the predictability of IR by 24-h DP, BMI and HR. For all analysis, values of $p < 0.05$ were selected as statistically significant.

RESULTS

The basic characteristics of the study population are presented in Table 1. The population was divided according to the WHO definition into: a nonobese subgroup defined by BMI < 30 kg/m^2 and an obese subgroup with BMI ≥ 30 kg/m^2.

No significant differences in terms of age or sex were observed between the subgroups. Similarly, there were no statistical differences between the studied subgroups in mean values of office and ABPM (except daytime SBP), HR or DP (Table 2). There was a higher waist circumference in obese than in nonobese patients. The subgroups did not differ in fasting glucose levels, although insulin levels were significantly lower in the nonobese than the obese group. Insulin resistance defined by Lebovitz's classification was observed three times more often in the obese subgroup than the nonobese (Table 1). In the total group, there was a significant relationship between HOMA-IR and office, 24-h, daytime and nighttime DP (Table 3). Table 4 presents correlations between HOMA-IR and office, 24-h, day and night DP in obese subjects. In the nonobese group, we did not observe a significant relationship between HOMA-IR and office or ABPM DP. Between the nonobese and obese groups, there was no significant difference in pharmacological antihypertensive treatment.

Table 1. Basic characteristic of the study group

	Total (n = 73)	Nonobese (n = 44)	Obese (n = 29)	P
Male [%]	52.0	54.5	48.3	NS
Age [years]	67.1 ± 8.4	67.9 ± 9.2	65.8 ± 6.9	NS
Body mass index [kg/m^2]	29.5 ± 4.2	26.7 ± 2.0	33.8 ± 2.8	< 0.001
Waist circumference [cm]		98.3 ± 12.1	104.6 ± 16.3	< 0.02
Glucose level [mmol/L]	5.4 ± 0.6	5.3 ± 0.5	5.5 ± 0.6	NS
Insulin level [μU/mL]	9.2 ± 5.5	7.3 ± 2.6	12.0 ± 7.3	< 0.01
HOMA-IR [μU/mL × mmol/L]	2.3 ± 1.5	1.8 ± 0.7	3.0 ± 2.0	< 0.01
HOMA-IR > 2.7 [μU/mL × mmol/L, %]	17.8	9.1	31.3	< 0.02
Antihypertensive drugs [%]:				
Beta-blockers	73.0	68.9	75.2	NS
Calcium channel blockers	20	18.4	21.6	NS
ACE-inhibitor	61	58.2	62.4	NS
ARB	11	10.8	11.7	NS
Diuretics	18	16.7	18.9	NS

HOMA-IR — homeostasis model assessment of insulin resistance; ACE — angiotensin converting enzyme; ARB — inhibitor for angiotensin receptor type 2

Table 2. 24-h, day, night and office blood pressure, heart rate and double product values in the study group

	Total	BMI < 30	BMI ≥ 30	P
24-h SBP [mm Hg]	126.1 ± 10.1	124.4 ± 10.3	128.7 ± 9.2	NS
24-h DBP [mm Hg]	71.5 ± 8.6	71.4 ± 8.8	71.7±8.5	NS
24-h HR [bpm]	66.5 ± 9.0	66.4 ± 9.1	66.5 ± 9.1	NS
24-h DP [mm Hg/min]	8382 ± 1324	8264 ± 1362	8560 ± 1267	NS
Day SBP [mm Hg]	130.4 ± 10.9	128.0 ± 10.8	134.1 ± 10.1	< 0.02
Day DBP [mm Hg]	75.1 ± 9.2	74.9 ± 9.4	75.4 ± 9.1	NS
Day HR [bpm]	70.3 ± 10.3	70.2 ± 10.4	70.4 ± 10.3	NS
Day DP [mm Hg/min]	9164 ± 1568	8982 ± 1614	9440 ± 1480	NS
Night SBP [mm Hg]	117.1 ± 11.9	116.9 ± 12.9	117.4 ± 10.5	NS
Night DBP [mm Hg]	63.9 ± 9.1	64.2 ± 90.4	63.4 ± 8.7	NS
Night HR [bpm]	59.2 ± 8.4	59.2 ± 8.5	59.1 ± 8.5	NS
Night DP [mm Hg/min]	6945 ± 1266	6947 ± 1340	6943 ± 1172	NS
Office SBP [mm Hg]	145.7 ± 18.6	143.1 ± 17.9	149.7 ± 19.2	NS
Office DBP [mm Hg]	81.1 ± 10.5	80.0 ± 10.6	82.9 ± 10.3	NS
Office HR [bpm]	68.6 ± 13.1	67.6 ± 13.7	70.1 ± 12.2	NS
Office DP [mm Hg/min]	9977 ± 2231	9640 ± 2145	10787 ± 2299	NS

BMI — body mass index; SBP — systolic blood pressure; DBP — diastolic blood pressure; HR — heart rate; DP — double product

Table 3. Correlations between HOMA-IR and office, 24-h, day and night blood pressure parameters in total group

	r	p
Office SBP	0.16	NS
Office HR	0.33	0.005
Office DP	0.40	< 0.001
24-h SBP	0.14	NS
24-h HR	0.32	0.005
24-h DP	0.45	0.02

SBP — systolic blood pressure; HR — heart rate; DP — double product; r — correlation coefficient

Table 4. Correlation between HOMA-IR and office, 24-h, day, night DP in obese subjects

	r	p
Office DP	0.6	< 0.001
24-h DP	0.61	< 0.01
Day DP	0.43	< 0.01
Night DP	0.58	< 0.001

DP — double product; r — correlation coefficient

In the total group of patients there was a positive correlation between HOMA-IR and BMI ($r = 0.45$, $p < 0.001$), as well as between HOMA-IR and waist circumference ($r = 0.49$, $p < 0.001$). Moreover, in the total group, after adjustment for sex and age, HOMA-IR was independently related to 24-h DP (β 0.3, $p < 0.001$) as well as to 24-h HR (β 0.3, $p < 0.01$) in stepwise multiple linear regression model. In obese patients, there was a positive correlation between HOMA-IR and BMI ($r = 0.63$, $p < 0.001$) and 24-h DP ($r = 0.51$, $p <$ < 0.01), as well as between HOMA-IR and waist circumference ($r = 0.64$, $p < 0.001$). But in nonobese patients, there was a positive correlation only between HOMA-IR and BMI ($r = 0.36$, $p < 0.01$) and 24-h DP ($r = 0.47$, $p < 0.001$), but not between HOMA-IR and waist circumference.

In the assessment of IR, among office and ABPM values of HR, BP and DP the ROC analysis revealed the highest AUC value for 24-h DP and for 24-h HR. Furthermore, there was a higher AUC value for 24-h DP than for 24-h HR ($0.72 \pm$ $\pm$ 0.09 vs. 0.60 ± 0.08, $p < 0.05$). However, we did not observe any significant differences of AUC value between 24-h DP and BMI (0.72 ± 0.09 vs. 0.72 ± 0.08, p = NS). Based on the shortest distance on the ROC curve (corresponding to the largest sum sensitivity and specificity), optimal cut-offs were: 8,978 mm Hg/min for 24-h DP and 33.02 kg/m^2 for BMI (Fig. 1). In the patients without insulin resistance (HOMA--IR < 2.7 μU/mL × mmol/L), there was no subject with 24-h DP and BMI values over the cut-off points, and in this group there were 25.9% subjects with only one value (24-h DP or BMI) over the cut-off point.

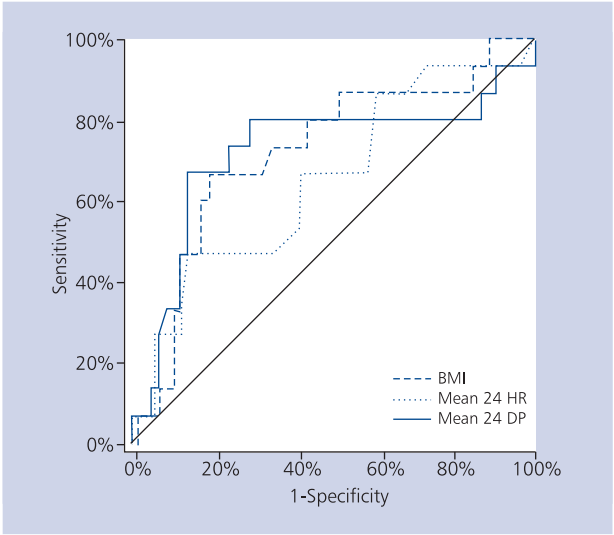

Figure 1. ROC curves in assessment of insulin resistance in total study group; BMI — body mass index; HR — heart rate; DP — double product

DISCUSSION

The results of our study provide several new findings on the relationship between IR and DP in a cohort of hypertensive patients with coronary atherosclerosis confirmed by angiography.

We have shown that HR and DP were significantly related to IR only in obese patients with confirmed CAD despite the widespread use of HR lowering drugs. HOMA-IR as a parameter of IR was higher in obese subjects than in nonobese, but there was no difference between HR, SBP and DP. Comparable values of BP and HR may result from similar pharmacological treatment in both groups. This fact indicates that simple haemodynamic parameters modified by pharmacological treatment (BP, HR or DP) related to autonomic nervous system activity are not useful indicators for IR in nonobese and hypertensive patients with CAD.

HR is a simple marker of autonomic influence on the heart. The relationship between sympathetic nerve activity and insulin secretion is a complex of differential sympathetic effects on pancreatic α- and β-adrenergic receptors. Adrenergic stimuli markedly blunt insulin secretion, mainly via α-receptors, but catecholamines in low concentrations potentially stimulate insulin secretion by activating β-receptors [14]. Results from our study suggest that in nondiabetic subjects with CAD, there is a significant relationship between HR, DP (office as well as ABPM) and IR. However there is no significant relationship between SBP and IR.

The relationship between BMI and HOMA-IR has been well documented in previous studies [15]. ROC analysis has revealed that 24-h DP and BMI were factors predicting IR in our study group of nondiabetic hypertensives with confirmed coronary atherosclerosis. According to our results, a combination of elevated BMI and 24-h DP may improve prediction

of IR at least in nondiabetic hypertensives with CAD. Moreover, the absence of coexisting elevated values of BMI and DP may exclude risk of IR. On the one hand, the relation between obesity and IR is not present in all obese subjects. But on the other hand, nonobese, nondiabetic, healthy individuals can be insulin resistant, and type 2 diabetes occurs also in nonobese patients [16]. It would be interesting to examine this relationship in normotensive subjects without significant coronary atherosclerosis.

An important finding of our study was that office and ABPM values (except daytime SBP) did not differ between the nonobese group and the obese group. The same observation has been reported in other studies [17]. Surprisingly, we did not observe a significant correlation between ABPM values and HOMA-IR in the whole study group, nonobese and obese subjects. According to previous studies, the relationship between serum insulin level or IR and BP values is obscure.

An important strength of this study is that in the nonobese and obese subgroups, age, gender and fasting serum glucose did not differ. Thus, the homogeneity of our study population minimises any potential confounding influence of these variables on the relationships we described.

Limitations of the study

The limitation of our study is that all recruited patients were hypertensive and received antihypertensive treatment which could influence the autonomic nervous system, BP, HR and IR. However, there was no significant difference in the pharmacological treatment of hypertension between the nonobese and the obese group. The fact that we used a surrogate measure of IR such as HOMA-IR index, instead of the gold standard euglycaemic hyperinsulinaemic clamp, could have an influence on our results. However, other studies which did use a clamp technique have also reported an association between HR and IR [1].

CONCLUSIONS

24-h DP calculated as SBP times HR can be a valuable parameter in predicting IR in hypertensive nondiabetics with CAD confirmed by percutaneous coronary interventions in history and/or at least one coronary artery stenosis ≥ 70% in elective coronary angiography. In nondiabetics with hypertension and coronary atherosclerosis, 24-h DP may be a good simple parameter indicating the IR. Moreover, 24-h DP and BMI may be complementary parameters in evaluating the risk of IR.

Conflict of interest: *none declared*

References

1. Facchini FS, Stoohs RA, Reaven GM. Enhanced sympathetic nervous system activity. The linchpin between insulin resistance, hyperinsulinemia, and heart rate. Am J Hypertens, 1996; 9: 1013–1017.
2. DeFronzo RA, Gunnarsson R, Bjorkman O et al. Effects of insulin on peripheral and splanchnic glucose metabolism in non-insulin-dependent (type II) diabetes mellitus. J Clin Invest, 1985; 76: 149–155.
3. Festa A, D'Agostino R, Jr., Hales CN et al. Heart rate in relation to insulin sensitivity and insulin secretion in nondiabetic subjects. Diabetes Care. 2000; 23: 624–628.
4. Cabou C, Cani PD, Campistron G et al. Central insulin regulates heart rate and arterial blood flow: an endothelial nitric oxide synthase-dependent mechanism altered during diabetes. Diabetes, 2007; 56: 2872–2877.
5. Laslett LJ, Paumer L, Amsterdam EA. Increase in myocardial oxygen consumption indexes by exercise training at onset of ischemia in patients with coronary artery disease. Circulation, 1985; 71: 958–962.
6. Gray DS, Fujioka K. Use of relative weight and body mass index for the determination of adiposity. J Clin Epidemiol, 1991; 44: 545–550.
7. Report of a WHO Consultation. Obesity: preventing and managing the global epidemic. World Health Organ Tech Rep Ser, 2000; 894: I-XII, 1–253.
8. Caro JF. Clinical review 26: Insulin resistance in obese and nonobese man. J Clin Endocrinol Metab, 1991; 73: 691–695.
9. Kahn BB, Flier JS. Obesity and insulin resistance. J Clin Invest, 2000; 106: 473–481.
10. Ryden L, Standl E, Bartnik M et al. Guidelines on diabetes, pre-diabetes, and cardiovascular diseases: executive summary. The Task Force on Diabetes and Cardiovascular Diseases of the European Society of Sardiology (ESC) and of the European Association for the Study of Diabetes (EASD). Eur Heart J, 2007; 28: 88–136.
11. Mancia G, De Backer G, Dominiczak A et al. 2007 ESH-ESC practice guidelines for the management of arterial hypertension: ESH-ESH Task Force on the management of arterial hypertension. J Hypertens, 2007; 25: 1751–1762.
12. Matthews DR, Hosker JP, Rudenski AS et al. Homeostasis model assessment: insulin resistance and beta-cell function from fasting plasma glucose and insulin concentrations in man. Diabetologia, 1985; 28: 412–419.
13. Lebovitz HE. Treating hyperglycemia in type 2 diabetes: new goals and strategies. Cleve Clin J Med, 2002; 69: 809–820.
14. Miller RE. Pancreatic neuroendocrinology: peripheral neural mechanisms in the regulation of the Islets of Langerhans. Endocr Rev, 1981; 2: 471–494.
15. Vogeser M, Konig D, Frey I et al. Fasting serum insulin and the homeostasis model of insulin resistance (HOMA-IR) in the monitoring of lifestyle interventions in obese persons. Clin Biochem, 2007; 40: 964–968.
16. Sinaiko A. Obesity, insulin resistance and the metabolic syndrome. J Pediatr (Rio J), 2007; 83: 3–4.
17. de la Sierra A, Ruilope M. Ambulatory blood pressure in obesity. CMReJournal, 2009; 2: 31–36.

Związek między częstością rytmu serca i podwójnym iloczynem a insulinoopornością u pacjentów z nadciśnieniem tętniczym i chorobą wieńcową

Wojciech Sobiczewski[1], Marcin Wirtwein[2], Edyta Kowalczys[1], Iwona Stopczyńska[1], Andrzej Koprowski[1], Marcin Gruchała[1], Andrzej Rynkiewicz[1]

[1]I Klinika Kardiologii, Gdański Uniwersytet Medyczny, Gdańsk

[2]Katedra Farmakologii, Gdański Uniwersytet Medyczny, Gdańsk

Streszczenie

Wstęp: Częstość rytmu serca (HR) oraz insulinooporność są wykładnikami wzmożonej aktywności współczulego układu nerwowego i mogą istotnie wpływać na rozwój choroby wieńcowej i cukrzycy.

Cel: Celem badania była ocena zależności między HR, wartościami ciśnienia tętniczego krwi, podwójnego iloczynu a insulinoopornością u pacjentów bez cukrzycy, z nadciśnieniem tętniczym i chorobą wieńcową.

Metody: Do badania włączono 73 osób; 24-godzinny pomiar ciśnienia tętniczego wykonano z zastosowaniem aparatów SpacLabs 90207. Ocenę isulinooporności przeprowadzono przy użyciu wskaźnika insulinooporności (HOMA-IR). Podwójny iloczyn wyliczono jako iloczyn wartości skurczowego ciśnienia tętniczego i HR.

Wyniki: W badanej grupie (średni wiek 67,1 ± 8,4 roku, 52% mężczyzn) zaobserwowano dodatnią korelację między HOMA-IR i 24-godzinną wartością podwójnego iloczynu ($r = 0,35$; $p < 0,01$) i wskaźnikiem masy ciała (BMI; $r = 0,45$; $p < 0,001$). Analiza ROC 24-godzinnego podwójnego iloczynu i BMI nie wykazała statystycznie istotnej różnicy między AUC (0,72 ± ± 0,09 *vs*. 0,72 ± 0,08, 24-godzinny podwójny iloczyn i BMI, odpowiednio, p = NS). W analizie ROC insulinooporności punkt odcięcia dla 24-godzinnego podwójnego iloczynu wynosił 8978 mm Hg/min, natomiast dla BMI 33,02 kg/m^2. Zaobserwowano również istotne różnice między pacjentami bez otyłości ($n = 44$, średni wiek 67,9 ± 9,2 roku) i z otyłością ($n = 29$, średni wiek 65,8 ± 6,9 roku) w stężeniu insuliny (7,3 ± 2.3 μU/ml *vs*. 12,0 ± 7,3 μU/ml; $p < 0,01$), HOMA-IR (1,8 ± ± 0,7 μU/ml × mmol/l *vs*. 3,0 ± 2,0 μU/ml × mmol/l; $p < 0,01$) i dziennym skurczowym ciśnieniu tętniczym (128,0 ± 10,8 mm Hg *vs*. 134,1 ± 10,1 mm Hg; $p < 0,02$).

Wnioski: 24-godzinny podwójny iloczyn oraz BMI może być parametrem uzupełniającym ocenę insulinooporności u pacjentów bez cukrzycy, z nadciśnieniem tętniczym i chorobą wieńcową potwierdzoną angioplastyką tętnic wieńcowych lub obecnością ≥ 70% zwężenia w co najmniej jednej tętnicy wieńcowej.

Słowa kluczowe: podwójny iloczyn, insulinooporność, choroba wieńcowa

Kardiol Pol 2013; 71, 1: 47–52

Adres do korespondencji:
lek. Marcin Wirtwein, Katedra Farmakologii, Gdański Uniwersytet Medyczny, ul. Dębowa 23, 80–211 Gdańsk, tel/faks: +48 58 349 25 42, e-mail: marwirt@interia.pl; wsob@gumed.edu.pl
Praca wpłynęła: 25.03.2012 r. **Zaakceptowana do druku:** 13.09.2012 r.