



Evaluation of lipid profiles in patients with metabolic syndrome according to cardiovascular risk calculated on the basis of the SCORE chart

Ocena parametrów lipidowych u pacjentów z rozpoznaniem zespołem metabolicznym w zależności od ryzyka sercowo-naczyniowego obliczonego według skali SCORE

Marcin Gierach^{1, 2}, Joanna Gierach², Roman Junik¹

¹Department of Endocrinology and Diabetology, Collegium Medicum in Bydgoszcz, Nicolaus Copernicus University, Toruń, Poland

²Internal Ward, Hospital in Wąbrzeźno, Poland

Abstract

Introduction: Metabolic syndrome predicts the development of CVD. Lipid abnormalities probably have an important influence on the increase of cardiovascular death risk. The SCORE chart includes only total cholesterol level, which may be inadequate. The aim of our study was to evaluate the lipid profile in patients with metabolic syndrome according to the cardiovascular risk calculated on the basis of the SCORE chart.

Material and methods: The study participants comprised 974 patients with metabolic syndrome. The 10-year death risk of cardiovascular disease was calculated on the basis of SCORE chart in all patients. The study group was divided in three subgroups depending on the risk level calculated by SCORE scale.

Results: There was a significantly higher level of LDL-C fraction in the subgroup of very high CV risk in comparison to the group of medium and high CV risk. The level of non-HDL-C was also significantly higher in the group with SCORE ≥ 10 compared to the remaining subgroups of medium and high CV risk.

Conclusions: Increased CV risk in this group of patients may be associated not only with higher TC level, but also the other lipid fractions. The assessment of the CV risk on the basis of the SCORE chart, which includes only TC level, may be inadequate. A modification of the SCORE chart for the European population should be considered (inclusion of LDL-C level, or in selected cases non-HDL-C level instead of TC level). (*Endokrynol Pol* 2016; 67 (3): 265–270)

Key words: metabolic syndrome; cardiovascular risk; SCORE chart

Streszczenie

Wstęp: Zespół metaboliczny poprzedza rozwój choroby sercowo-naczyniowej. Ważny wpływ na zwiększenie ryzyka sercowo-naczyniowego mają prawdopodobnie zaburzenia gospodarki lipidowej. W skali SCORE ujęto jedynie poziom cholesterolu całkowitego, który może być nieadekwatny.

Celem naszej pracy była ocena profile lipidowego u pacjentów z zespołem metabolicznym w zależności od obliczonego na podstawie skali SCORE ryzyka sercowo-naczyniowego.

Materiał i metody: Do badania włączono 974 pacjentów z zespołem metabolicznym. Dziesięcioletnie ryzyko nagłego zgonu z przyczyn sercowo-naczyniowych obliczono na podstawie skali SCORE *chart* u wszystkich pacjentów. Badaną grupę podzielono na 3 podgrupy w zależności od poziomu ryzyka obliczonego za pomocą skali SCORE.

Wyniki: Stwierdzono istotnie wyższe stężenie cholesterolu frakcji LDL w podgrupie pacjentów z bardzo wysokim ryzykiem sercowo-naczyniowym w porównaniu z podgrupą wysokiego i średniego ryzyka sercowo-naczyniowego. Stężenie cholesterolu frakcji non-HDL był również istotnie wyższy w podgrupie bardzo wysokiego ryzyka sercowo-naczyniowego.

Wnioski: Zwiększone ryzyko sercowo-naczyniowe w grupie pacjentów z zespołem metabolicznym może być związane nie tylko z wysokim stężeniem cholesterolu całkowitego, ale również z innymi frakcjami lipidowymi.

Ocena ryzyka sercowo-naczyniowego na podstawie skali SCORE, która zawiera jedynie stężenie cholesterolu całkowitego, może być nieodpowiednia. Trzeba rozważyć modyfikacje skali SCORE w populacji Europejczyków (włączenie do oceny cholesterolu frakcji LDL, non-HDL, zamiast cholesterolu całkowitego). (*Endokrynol Pol* 2016; 67 (3): 265–270)

Słowa kluczowe: zespół metaboliczny; ryzyko sercowo-naczyniowe; skala SCORE



Introduction

Cardiovascular diseases (CVD) are a significant cause of death in Europe and worldwide [1–4]. In Poland CVD caused 46.0% of all deaths in 2010 [5]. In the European Union, to estimate cardiovascular disease risk and to apply primary cardiovascular (CV) prevention, the SCORE chart is used. The SCORE chart evaluates the 10-year death risk of CVD and is based on the following risk factors: gender, age, systolic blood pressure [SBP], total cholesterol [TC], and smoking.

Metabolic syndrome (MetS) comprises a constellation of CV risk factors that include abdominal obesity, insulin resistance, glucose intolerance, elevated blood pressure or antihypertensive drug treatment, low levels of high-density lipoprotein (HDL-C) cholesterol, and elevated triglyceride (TG) levels [6–10]. Abundant evidence shows that MetS predicts the development of CVD [11–13].

Lipid abnormalities probably have an important influence on the increase in CV death risk. Also, in patients with MetS, lipid disorders have an unquestionable impact on the increase of the risk of CV death.

It is worth mentioning that according to the SCORE chart, to assess CV risk only the total cholesterol level is taken into consideration. But sometimes TC may be misleading, for example in patients with MetS, who often have low HDL-C levels. Calculation of low-density lipoprotein cholesterol (LDL-C) is also very important because it is still widely used and it is the first lipid target of therapy (IA class) according to the guidelines for the management of dyslipidaemias of the European Atherosclerosis Society and the European Society of Cardiology (ESC/EAS) [14, 15]. In patients with multiple CV risk factors LDL-C is suggested as being essential to effectively manage the overall risk [16, 17]. Thus, for an adequate risk analysis, at least HDL-C and LDL-C should be analysed [14].

Therefore, the aim of our study was to evaluate the lipid profile in patients with MetS according to the CV risk calculated on the basis of the SCORE chart.

Material and methods

The described study was conducted in a two-centre (University Hospital No. 1 in Bydgoszcz, District Hospital in Wąbrzeźno, Poland) screening study of 36 months duration (2011–2014 years). The study participants comprised 974 patients, average aged 58.3 years (\pm SD 11.9; min — 42; max — 71), with MetS diagnosed according to the 2005 IDF (International Diabetes Federation) criteria (meeting at least three criteria). The criteria are presented in Table I.

Table I. IDF criteria of metabolic syndrome

Tabela I. Kryteria rozpoznania zespołu metabolicznego w IDF

Abdominal obesity [cm]	F \geq 80 or M \geq 94
Arterial hypertension (HT) [mm Hg]	\geq 130/85 or treated for arterial hypertension
Triglycerides (TG) [mg/dL]	\geq 150 [1.7 mmol/L] or treated for dyslipidaemia
HDL-C [mg/dL]	< 50 [1.3 mmol/L] in women and < 40 [1.0 mmol/L] in men
Fasting glycaemia [mg/dL]	\geq 100 [5.6 mmol/L] or treated for diabetes

Anthropometric measurements (height, weight, and waist circumference — WC) were taken in all subjects. Body mass index (BMI) was calculated according to the formula: body weight (in kilograms) divided by the square of body height (in metres). WC was measured at the point midway between the last rib and the iliac crest. Demographic factors (age, gender, obesity) were determined. Blood pressure was measured by automatic blood pressure monitor. The measurements were performed with the participant in a seated position, on the right and left upper arm, after at least 10 minutes of rest and 5-minute intervals. Fasting total plasma cholesterol, triglycerides, and high-density lipoprotein cholesterol were determined in all patients. Low-density lipoprotein cholesterol was calculated using the Friedewald formula. The non-high-density lipoprotein cholesterol (non-HDL-C) values were obtained finding the difference between TC and HDL-C. In patients with waist circumference > 80 cm in females and > 94 cm in males or with fasting glucose level > 100 mg/dL the oral glucose tolerance test (OGTT) was performed. Glomerular Filtration Rate (GFR) [mL/min/1.73 m²] was measured by means of MDRD formula.

The 10-year death risk of CVD was calculated on the basis of SCORE chart in all patients. The study group was divided in three subgroups depending on the risk level calculated by SCORE scale: patients with medium (1–4%), high (5–9%), and very high CV risk (\geq 10%). Additionally, patients with diabetes mellitus type 1 and 2 without CV risk factor or target organ damage, or moderate chronic kidney disease (GFR 30–59 mL/min/1.73m²) or markedly elevated single CV risk factor (e.g. familial dyslipidaemia or severe hypertension) were included ad hoc into the group of high CV risk. Whereas, to the group of very high CV risk (\geq 10%), additionally, patients with DM type 1 or type 2 with one or more CV risk factor/s and/or target organ damage, documented CVD (including coronary disease, peripheral artery disease or stroke), or severe

Table II. The results of serum profile in the study group divided into three subgroups according to CV risk (SCORE chart) (mean value \pm standard deviation [minimum–maximum])**Tabela II.** Wyniki zaburzeń lipidowych grupy badanej podzielonej na 3 podgrupy według stwierdzonego ryzyka sercowo-naczyniowego (skala SCORE) (wartość średnia \pm odchylenie standardowe [minimum–maksimum])

Parameter	Subgroup 1 medium CV risk (SCORE 1–4%) (N = 376)	Subgroup 2 high CV risk (SCORE 5–9%) (N = 369)	Subgroup 3 very high CV risk (SCORE \geq 10%) (N = 229)
BMI [kg/m ²]	30.7 \pm 5.77 (18.36–45.77)	30.5 \pm 5.6 (18.7–42.8)	30.8 \pm 4.3 (18.6–44.6)
LDL-C [mg/dL]	107.7 \pm 40.3 (51.0–257)	110.7 \pm 39.3 (49.0–231.0)	129.4 \pm 42.4 (49.0–266.0)
HDL-C [mg/dL]	41.5 \pm 10.8 (29.0–96.0)	43.8 \pm 13.3 (27.0–103.0)	42.5 \pm 11.7 (32.0–78.0)
TG [mg/dL]	149.7 \pm 100.5 (59.0–542.0)	134.7 \pm 69.0 (62.0–476.0)	167.8 \pm 96.4 (68.0–489.0)
Non-HDL-C [mg/dL]	135.9 \pm 46.1 (48.0–192.0)	137.7 \pm 43.1 (45.0–254.0)	161.7 \pm 47.6 (48.0–258.0)

chronic kidney disease (GFR < 30 mL/min/1.73 m²) were included.

Exclusion criteria were as follows: (1) a history of heart surgery or other cardiovascular interventions, (2) congenital defects of the heart, (3) cardiac rhythm disorders, (4) pregnancy, (5) electrolyte disorders, (6) inflammation, (7) anaemia, (8) prostate disease, (9) Cushing's syndrome, (10) thyroid disorders.

The results of the study were analysed statistically. Quantitative data were presented as arithmetic means with standard deviations. In order to verify the conformity of variable distribution with normal distribution, the Shapiro–Wilk test was used. For the comparison of the subgroups the Kruskal–Wallis test was used. Additionally, a nonparametric version of the Student's t-test for independent variables was used. All hypotheses were verified at $p = 0.05$. Values of $p < 0.05$ were considered as statistically significant. All statistical analyses were performed using Statistica 10.0 software (Statsoft Poland, Bydgoszcz).

The study protocol was approved by the Bioethics Committee of Ludwig Rydygier Collegium Medicum in Bydgoszcz, Nicolaus Copernicus University in Toruń. All subjects gave their informed consent for participation in the study.

Results

The study group consisted of 974 patients [574 females (58.9%) and 400 males (41.1%)] of mean age 58.3 ± 11.9 years (min 42; max 71 years). The mean value of BMI was 30.7 ± 5.5 kg/m², mean WC was 107.9 ± 13.1 cm, and mean SBP was 144.7 ± 20.1 mm Hg. Mean values of lipid fractions were as follows: LDL-C — 113.9 ± 41.2 mg/dL; TG — 148.0 ± 89.0 mg/dL, HDL-C — 42.6 ± 11.9 mg/dL and non-HDL-C — 142.5 ± 46.4 mg/dL.

On the basis of the CV risk calculated using the SCORE scale, three subgroups were extracted. The group of medium CV risk (SCORE 1–4%) consisted

of 376 patients (38.6%). To the group of high CV risk (SCORE 5–9%) 369 patients (37.9%) were included. The group of very high CV risk (SCORE \geq 10%) consisted of 229 patients (23.5%). The results of serum lipid profile of the extracted subgroups are presented in Table II.

There was a significantly higher level of LDL-C fraction in the subgroup of very high CV risk in comparison to the group of medium and high CV risk (129.4 ± 42.4 vs. 107.7 ± 40.3 , $p = 0.0001$ and 110.7 ± 39.3 , $p = 0.0001$, respectively). The level of non-HDL-C was also significantly higher in the group with SCORE \geq 10 compared to the remaining subgroups of medium and high CV risk (respectively, 161.7 ± 47.6 vs. 135.9 ± 46.1 and 137.7 ± 43.1 , $p = 0.0001$). TG level was higher in the subgroup of very high CV risk compared to the subgroup of high CV risk (167.8 ± 96.4 vs. 134.7 ± 69.0 , $p = 0.0001$). There were no statistically significant differences in HDL-C levels between the subgroup of very high CV risk and the other subgroups. The comparison between particular subgroups is shown in Table III.

Discussion

About 80% of CVD is associated with such behavioural risk factors as: unhealthy diet, physical inactivity, tobacco use, and harmful use of alcohol. The effects of unhealthy diet may show up in individuals as raised blood lipids, overweight, and obesity. These factors can be easily measured in primary care facilities [2].

In many countries, including Poland, the most widespread modifiable risk factors are lipid disorders. They occur in more than 60% of Polish adults — about 18 million people > 18 years old [5, 18]. According to the WOBASZ study the frequency of elevated TC is at the level of 67% in males and 64% in females, respectively; the elevated LDL-C level is 60% in men and 55% in women; hypertriglyceridaemia is 32% in males and 20% in females, and decreased HDL-C level is 15% in men and 17% in women [18]. In the POLSENIOR

Table III. Comparison of lipid profile between subgroups extracted according to SCORE scale**Tabela III.** Porównanie profilu lipidowego w poszczególnych podgrupach w zależności od skali SCORE

Parameter	(I) SCORE	(J) SCORE	The difference of averages (I-J)
BMI [kg/m ²]	1-4%	5-9%	.293
		≥ 10%	-.034
	5-9%	1-4%	-.293
		≥ 10%	-.327
	≥ 10%	1-4%	.034
		5-9%	.327
LDL-C [mg/dL]	1-4%	5-9%	-3.03
		≥ 10%	-21.73*
	5-9%	1-4%	3.03
		≥ 10%	-18.69*
	≥ 10%	1-4%	21.73*
		5-9%	18.69*
HDL-C [mg/dL]	1-4%	5-9%	-2.36*
		≥ 10%	-1.04
	5-9%	1-4%	2.36*
		≥ 10%	1.31
	≥ 10%	1-4%	1.04
		5-9%	-1.31
TG [mg/dL]	1-4%	5-9%	15.01
		≥ 10%	-18.05
	5-9%	1-4%	-15.01
		≥ 10%	-33.06*
	≥ 10%	1-4%	18.05
		5-9%	33.06*
nonHDL-C [mg/dL]	1-4%	5-9%	-1.83
		≥ 10%	-25.82*
	5-9%	1-4%	1.8319
		≥ 10%	-23.99*
	≥ 10%	1-4%	25.82*
		5-9%	23.99*

study hypercholesterolaemia was noticed in 62% of patients above ≥ 65 years of age (56% in males and 66% in females) [19].

In patients with MetS abdominal obesity is frequently accompanied by lipid metabolism disorders, insulin resistance, and elevated blood pressure. In our study it occurred in 98% of patients. Abundant evidence shows that MetS predicts the development of CVD [11-13, 20]. Takahashi showed that individuals with MetS have a four-fold greater probability of high CAD risk score [3]. Also, the results of other studies indicate

greater probability for an increase risk of CVD in people with MetS [21].

Lipid abnormalities probably have an important influence on the increase of CV risk. In patients with MetS, lipid metabolism disorders also have an unquestionable impact on the increase of the risk of CV death.

In the European Union, the SCORE chart is used to estimate CVD risk. The SCORE chart evaluates the 10-year death risk of cardiovascular disease and is based on the following risk factors: gender, age, SBP, TC, and smoking.

According to the most important recommendations concerning lipid disorder therapy, published in 2001 and actualised in 2004 (NCEP-ATP III, National Cholesterol Educational Program — Adult Treatment Panel), to stratify treatment, patients should be divided into groups of different risk level according to the SCORE scale. Adequate therapeutic goals were designated for each group.

It is accordant with guidelines of scientific societies. According to the ESC/EAS guidelines, TC is recommended to estimate total CV risk by means of the SCORE system [14]. Also, the National Lipid Association (NLA) suggest that for the European population the SCORE charts should be still recommended. Taking into account TC level, the assessment of CV risk in patients, especially those with MetS, can be very difficult. Thus, for an adequate risk analysis, at least HDL-C and LDL-C should be analysed [14].

In our study it was shown that there was a significantly higher level of LDL-C fraction in the subgroup of very high CV risk in comparison to the group of medium and high CV risk (129.4 ± 42.4 vs. 107.7 ± 40.3 , $p = 0.0001$ and 110.7 ± 39.3 , $p = 0.0001$, respectively). LDL-C, which is calculated using Friedewald's formula, has some limitations. Especially in the case of high TG values (> 500 mg/dL) or increased levels of glycaemia the LDL-C level can be underestimated. Also, in patients with MetS with insulin resistance LDL-C can underestimate the LDL particle (LDL-P) number. Despite its limitations, the calculated LDL-C is still widely used and it is the first lipid target of therapy according to the ESC/EAS guidelines for the management of dyslipidaemias (IA class) [14, 15].

Banach et al. [16, 17] also suggest that LDL-C in patients with multiple CV risk factors is essential to effectively manage the overall risk. On the other hand, the reviewed published evidence showing a weak and potentially misleading association between LDL-C and coronary risk in patients with chronic kidney disease (CKD) [22]. Furthermore, in the Framingham Offspring Study [23] (a Project of the National Heart, Lung, and Blood Institute and Boston University) Cromwell WC et al. determined that in managing patients at risk

for CVD to LDL-C target levels, it is unclear whether LDL-C provides the optimum measure of residual risk and adequacy of LDL-lowering treatment [23]. They suggest that LDL-P was a more sensitive indicator of low CVD risk than either LDL-C or non-HDL-C, implying a potential clinical role for LDL-P as a goal of LDL management.

Another lipid fraction evaluated in our study was TG. A statistically significant higher level of TG was observed in the group of very high CV risk in comparison to the group of high CV risk (167.8 ± 96.4 vs. 134.7 ± 69.0 , $p = 0.0001$). Yamamoto et al. suggest that hypertriglyceridaemia may become an independent risk factor for atherosclerosis in addition to cholesterol [24]. The results of some other studies, published recently, also suggest that non-fasting TG may carry information regarding remnant lipoproteins associated with increased CV risk [2, 25, 26]. It is still debated how this should be used in clinical practice.

The UKPDS identified HDL-C as the second most important coronary risk factor, after LDL-C, in patients with T2DM [27, 28]. In many patients with MetS atherogenic dyslipidaemia (AD) (i.e. increased level of TG and decreased level of HDL-C) is found. Post hoc analyses of prospective trials in stable CHD patients revealed that elevated plasma levels of TG and low plasma concentrations of HDL-C are associated with this high risk [29]. Plana et al. [30] also determined that AD is an important risk factor for CVD. They examined 1137 patients and found that AD prevalence was about 27% (34.1% in diabetics). They ascertained that when LDL-C levels are controlled, AD is more prevalent in patients at highest CV risk and with DM [30]. Chapman et al. [31] noticed that in many countries AD is on the rise, due to the increasing prevalence of MetS, and is more prevalent in individuals at high risk of CVD. Interestingly, Hermans et al. [32] suggested that $\log(\text{TG}/\text{HDL-C})$ allows for grading of AD and estimating non-LDL-related vascular risk in DM females. In the Copenhagen City Heart Study, increased risk for myocardial infarction (MI), ischaemic stroke, and mortality was evident at markedly elevated TG (> 450 mg/dL), although these data were not adjusted for non-HDL-C [33].

Unfortunately, in our study we did not observe statistically significant differences between HDL-C levels in the subgroup of very high CV risk in comparison to the subgroup of moderate and high CV risk (42.5 ± 11.7 vs. 41.5 ± 10.8 , $p = 0.62$ and 43.8 ± 13.3 , $p = 0.51$).

Wiklund et al. suggested that selected individuals at high CV risk HDL-C, Lp(a), and ratios such as LDL-C/HDL-C or apoB/apoA1 are not recommended as treatment targets [34].

It is worth noting that in our study that non-HDL-C levels were significantly different between the group of

medium and high risk and the group of very high risk (135.9 ± 46.1 vs. 161.7 ± 47.6 , $p = 0.0001$, 137.7 ± 43.1 vs. 161.7 ± 47.6 , $p = 0.0001$, respectively). There was no significant difference between the group of medium and high CV risk. In epidemiological studies non-HDL-C is used as an estimation of the total number of atherogenic particles in plasma. Interestingly, non-HDL-C can provide a better risk estimation compared with LDL-C [35], particularly in MetS. The National Cholesterol Education Program Adult Treatment Panel III (NCEP ATP III) identified non-HDL-C even as the second lipid target of therapy after LDL-C [36]. It is also a very important target of therapy for CHD prevention [36]. It is easily calculated from TC minus HDL-C. Brunzellet et al. suggested that non-HDL-C is a better measure than LDL-C for identifying patients at high risk, who had multiple CV risk factors [37]. The American Diabetes Association (ADA) and the American College of Cardiology (ACC) Foundation recommended non-HDL-C goals of <100 mg/dL for all CHD patients and diabetic patients with any other CV risk factor, and a goal of <130 mg/dL for all CV metabolic risk patients with two major CV risk factors. Also, the NLA endorsed the importance of non-HDL-C [38]. Some authors [39,40] suggest that it is important to determine whether adding information on apoB, apoA1, Lp(a), or lipoprotein-associated phospholipase A2 to TC, LDL-C, and HDL-C improves CV risk prediction.

Conclusions

In patients with MetS of very high CV risk according to the SCORE chart, significantly higher levels of LDL-C, TG, and non-HDL-C were observed in comparison to the group of high and medium CV risk. Increased CV risk in this group of patients may be associated not only with higher TC levels, but also the other lipid fractions.

The results of our study and analysis of the literature concerning this topic suggest that assessment of the CV risk on the basis of the SCORE chart, which includes only the TC level, may be inadequate. Perhaps it is worth considering modification of the SCORE chart for the European population, to include LDL-C level or, in selected cases, non-HDL-C level, instead of TC level. Furthermore, in order to assess the CV risk more accurately in the groups of very high and high CV risk, TG and HDL-C level should be taken into consideration additionally.

References

1. Reiner Z. Managing the residual cardiovascular disease risk associated with HDL-cholesterol and triglycerides in statin-treated patients: a clinical update. *Nutr Metab Cardiovasc Dis* 2013; 23: 799–807.
2. Global status report on noncommunicable diseases 2010. Geneva, World Health Organization, 2011.

3. Takahashi MM, Oliveira EP, Rochitti de Carvalho AL. Metabolic syndrome and dietary components are associated with coronary artery disease risk score in free-living adults: a cross-sectional study. *Diabetology & Metabolic Syndrome* 2011; 3: 1–7.
4. Hu FB. Diet and lifestyle influences on risk of coronary heart disease. *Curr Atheroscler Rep* 2009; 11: 257–263.
5. Zdrojewski T, Rutkowski M, Bandosz P. Prevalence and control of cardiovascular risk factors in Poland. Assumptions and objectives of the NATPOL 2011 Survey. *Kardiol Pol* 2013; 71: 381–392.
6. Ferrario CM, Joyner JN, Colby C et al. The COSEHC Global Vascular Risk Management quality improvement program: first follow-up report. *Vasc Health Risk Manag* 2013; 9: 391–400.
7. Grundy SM. Metabolic syndrome: a multiplex cardiovascular risk factor. *J Clin Endocrinol Metab* 2007; 92: 399–404.
8. Grundy SM, Brewer HB, Cleeman Jr. Definition of metabolic syndrome: Report of the National Heart, Lung, and Blood Institute/American Heart Association conference on scientific issues related to definition. *Circulation* 2004; 109: 433–438.
9. Gierach M, Gierach J, Junik R. Insulin resistance and thyroid disorders. *Endocrinol Pol* 2014; 65: 70–76.
10. Zdrojowy-Wełna A, Tupikowska M, Kołackov K et al. The role of fat mass and obesity-associated gene (FTO) in obesity — an overview. *Endocrinol Pol* 2014; 65: 224–231.
11. De Simone G, Devereux RB, Chinali M. Strong Heart Study Investigators. Prognostic impact of metabolic syndrome by different definitions in a population with high prevalence of obesity and diabetes: the Strong Heart Study. *Diabetes Care* 2007; 30: 1851–1856.
12. Dekker JM, Girman C, Rhodes T. Metabolic syndrome and 10-year cardiovascular disease risk in the Hoorn Study. *Circulation* 2005; 112: 666–673.
13. Gami AS, Witt BJ, Howard DE. Metabolic syndrome and risk of incident cardiovascular events and death: a systematic review and meta-analysis of longitudinal studies. *J Am Coll Cardiol* 2007; 49: 403–414.
14. ESC/EAS Guidelines for management of dyslipidaemias The Task Force for the management of dyslipidaemias of the European Society of Cardiology (ESC) and the European Atherosclerosis Society (EAS). *Atherosclerosis* 217S 2011; S1–S44.
15. Catapano AL, Reiner Z, De Backer G. European Society of Cardiology (ESC); European Atherosclerosis Society (EAS). ESC/EAS Guidelines for the management of dyslipidaemias. The Task Force for the management of dyslipidaemias of the European Society of Cardiology (ESC) and the European Atherosclerosis Society (EAS). *Atherosclerosis* 2011; 217: 3–46.
16. Banach M, Serban C, Aronow WS. Lipid, blood pressure and kidney update 2013. *Int Urol Nephrol* 2014; 46: 947–961.
17. Banach M, Rysz J. Current problems in hypertension and nephrology. *Expert Opin Pharmacother* 2010; 11: 2575–2578.
18. Broda G, Rywik S. Multicenter national Polish population health status tests — WOBASZ project with defined problems and treatment goals. *Kardiol Pol* 2005; 63 (6 suppl. 4): S601–S604.
19. Bledowski P, Mossakowska M, Chudek J. Medical, psychological and socioeconomic aspects of aging in Poland. Assumptions and objectives of the PolSenior Project. *Exp Gerontol* 2011; 46: 1003–1009.
20. Wannamethee SG, Shaper AG, Lennon L et al. Metabolic syndrome vs Framingham Risk Score for prediction of coronary heart disease, stroke, and type 2 diabetes mellitus. *Arch Intern Med* 2005; 165: 2644–2650.
21. Carr MC, Brunzell JD. Abdominal obesity and dyslipidemia in the metabolic syndrome: importance of type 2 diabetes and familial combined hyperlipidemia in coronary artery disease risk. *J Clin Endocrinol Metab* 2004; 89: 2601–2607.
22. Tonelli M, Wanner C. for the Kidney Disease: Improving Global Outcomes Lipid Guideline Development Work Group Members (2014). Lipid management in chronic kidney disease: synopsis of the kidney disease: improving global outcomes 2013 clinical practice guideline. *Ann Intern Med*. 2014; 160: 182. doi: 10.7326/M13-2453.
23. Cromwell WC, Otvos JD, Keyes MJ. LDL particle number and risk of future cardiovascular disease in the Framingham Offspring Study — Implications for LDL management. *Clin Lipidology* 2007; 1: 583–592.
24. Yamamoto A, Yamamura T, Kawaguchi A et al. Triglyceride and glucose intolerance as a risk factor for coronary heart disease. *Cardiology* 1991; 78: 185–193.
25. Bansal S, Buring JE, Rifai N. Fasting compared with non-fasting triglycerides and risk of cardiovascular events in women. *JAMA* 2007; 298: 309–316.
26. Nordestgaard BG, Benn M, Schnohr P. Non-fasting triglycerides and risk of myocardial infarction, ischemic heart disease, and death in men and women. *JAMA* 2007; 298: 299–308.
27. Turner RC, Millns H, Neil HA. Risk factor for coronary artery disease in non-insulin dependent diabetes mellitus: United Kingdom Prospective Diabetes Study (UKPDS:23). *BMJ* 1998; 316: 823–828.
28. Sadikot S, Hermans MP. Here we go again... The metabolic syndrome revisited! *Diab Metab Syndr* 2010; 4: 111–120. doi: 10.1016/j.dsx.2010.05.011
29. Miller M, Cannon CP, Murphy SA. Impact of triglyceride levels beyond low-density lipoprotein cholesterol after acute coronary syndrome in the PROVE IT-TIMI 22 trial. *J Am Coll Cardiol* 2008; 51: 724–730.
30. Plana N, Ibarretxe D, Cabre A et al. Prevalence of atherogenic dyslipidemia in primary care patients at moderate-very high risk of cardiovascular disease. *Cardiovascular risk perception. Clin Investig Arterioscler* 2014; 26: 274–284. doi: 10.1016/j.arteri.2014.04.002.
31. Chapman MJ, Ginsberg HN, Amarenco P. Triglyceride-rich lipoproteins and high-density lipoprotein cholesterol in patients at high risk of cardiovascular disease: evidence and guidance for management. *Eur Heart J* 2011; 32: 1345–1361; doi: 10.1093/eurheartj/ehr112.
32. Hermans MP, Ahn SA, Rousseau MF. The atherogenic dyslipidemia ratio [log(TG)/HDL-C] is associated with residual vascular risk, beta-cell function loss and microangiopathy in type 2 diabetes females. *Lipids Health Dis* 2012; 11: 132.
33. Nordestgaard BG, Benn M, Schnohr P. Nonfasting triglycerides and risk of myocardial infarction, ischemic heart disease, and death in men and women. *JAMA* 2007; 298: 299–308.
34. Wiklund O, Pirazzi C, Romeo S. Monitoring of lipids, enzymes, and kinase in patient on lipid-lowering drug therapy. *Curr Cardiol Rep* 2013; 15: 397–399.
35. Cui Y, Blumenthal RS, Flaws JA. Non-high-density lipoprotein cholesterol level as a predictor of cardiovascular disease mortality. *Arch Intern Med* 2001; 161: 1413–1419.
36. Robinson JG, Wang S, Smith BJ et al. Meta-analysis of the relationship between non-high-density lipoprotein cholesterol reduction and coronary heart disease risk. *J Am Coll Cardiol* 2009; 53: 316–322.
37. Brunzell JD, Davidsson M, Furberg CD. Lipoprotein management in patients with cardiometabolic risk: consensus conference report from the American Diabetes Association and the American College of Cardiology Foundation. *J Am Coll Cardiol* 2008; 51: 1512–1524.
38. Blaha M, Blumenthal R, Brinton E et al. National Lipid Association Task force on Non-HDL cholesterol. The importance of non-HDL cholesterol reporting in lipid management. *J Clin Lipidol* 2008; 2: 267–273.
39. Di Angelantonio E, Gao P, Pennells L et al. Lipid-related markers and cardiovascular disease prediction. *JAMA* 2012; 307: 2499–2506.
40. Lai HM, Aronow WS, Mercado AD et al. The impact of statin therapy on long-term cardiovascular outcomes in an outpatient cardiology practice. *Arch Med Sci* 2012; 8: 53–56.