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The level of endogenous testosterone and its correlation with lipid profile in men older than 40 years with acute myocardial infarction

Nivo endogenog testosterona i njegova korelacija sa lipidnim profilom kod muškaraca sa akutnim infarktom miokarda, starijih od 40 godina

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Abstract

Background/Aim. The influence of lipid profile on acute myocardial infarct (AMI) is well known. On the other hand, the role of testosterone (T), as one of the possible predictive factors of AMI in men and its influence on lipid profile in men is still controversial. The aim of the study was to determine levels of T in AMI and six months after AMI in the same group of patients, and to compare with T levels in healthy men. Also we correlated T levels with lipid profile in patients with AMI and 6 months after AMI. **Methods.** The study was designed as prospective study. Patients were divided into III groups: Group I included 35 men, aged 55 ± 3 years, with AMI. Group II included the same 35 patients, analyzed 6 months after AMI. The group III consisted of 20 healthy men aged 57 ± 2.12 years (control group). Blood samples of the group I (AMI) were taken in the first 12 hours from the AMI beginning and also 6 months after AMI (group II). Following analyses were performed: levels of total cholesterol, triglycerides, low density lipoprotein (LDL) cholesterol, high density lipoprotein (HDL) cholesterol, lipoprotein(a) [Lp(a)], apolipoprotein A1 (Apo A1), apolipoprotein B (Apo B) and T. **Results.** Levels of T in patients with AMI (16.86 ± 7.18 nmol/L) as well as 6 months after AMI (18.12 ± 7.96 nmol/L) were statistically significantly lower than those in healthy persons of the same age (27.11 ± 10.48 nmol/L) ($p < 0.001$). In the group I, statistically significant, positive correlation was found between levels of T and HDL cholesterol ($r = 0.403$, $p < 0.05$), and levels of T and Apo A1 ($r = 0.747$, $p < 0.01$). In the group II, statistically significant, positive correlation was also found between levels of T and HDL cholesterol ($r = 0.388$, $p < 0.05$) and T and Apo A1($r = 0.354$, $p < 0.05$). **Conclusion.** This study showed that men, over 40 years of age, with AMI had statistically significantly lower concentrations of endogenous T compared to healthy male population of the same age. Levels of T in the same patients after 6 months from AMI maintained statistically significantly lower values in comparison to those in healthy men.

Key words:
lipids; lipoproteins; myocardial infarction; testosterone.

Apstrakt

Uvod/Cilj. Uticaj lipidnog profila na akutni infarkt miokarda (AIM) dobro je poznat. Nasuprot tome, uloga nivoa endogenog testosterona (T), kao jednog od mogućih prediktivnih faktora AIM i njegovo dejstvo na lipidni profil kod muškaraca sa AIM i dalje su kontroverzni. Cilj studije je bio da se odredi nivo endogenog T u AIM, kao i 6 meseci nakon AIM kod iste grupe ispitanika i da se uporedi sa nivoima T kod zdravih ispitanika. Pored toga, određena je i korelacija nivoa T sa parametrima lipidnog profila u AIM kao i šest meseci nakon AIM. **Metode**. Sprovedena je prospektivna studija u koju su bili uključeni muškarci podeljeni u tri grupe: grupu I činilo je 35 muškaraca, starosne dobi 55 ± 3 godine sa AIM; grupu II sačinjavalo je istih 35 muškaraca koji su analizirani 6 meseci nakon AIM, dok se grupa III sastojala od 20 zdravih ispitanika starosne dobi 57 ± 2,12 godina. Uzorci krvi kod ispitanika grupe I uzimani su u periodu od 12h od nastanka AIM. Kod istih ispitanika krv za analize uzeta je šest mesci nakon preležanog AIM (grupa II). U krvi su određivani nivoi: ukupnog holesterola, triglicerida, lipoproteina male gustine (LDL) holesterola, lipoproteina velike gustine (HDL) holesterola, apolioporoteina A1 (Apo A1), apolipoproteina B (Apo B), lipoproteina(a) [Lp(a)] i nivo endogenog T. **Rezultati.** Nivo T kod ispitanika sa AIM (grupa I) (16,86 ± 7,18 nmol/L), kao i kod istih ispitanika

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šest meseci nakon AIM (grupa II) (18,12 ± 7,96 nmol/L) bio je statistički značajno niži u odnosu na zdravu kontrolnu grupu iste starosti (27,11 ± 10,48 nmol/L) ($p < 0,001$). U grupi I dobijena je statistčki značajna, pozitivna korelacija između nivoa T i HDL holesterola ($r = 0.403$, $p < 0,05$) i visoko statistički značajna, pozitivna korelacija između nivoa T i Apo A1($r = 0,747$, $p < 0,01$). U grupi II, takođe je nađena statistički značajna pozitivna korelacija između nivoa T i HDL holeserola ($r = 0,388$, $p < 0,05$), kao i nivoa T i Apo A1 ($r = 0,354$, $p < 0,05$). **Zaključak.** Ova studija je pokazala da su nivoi T kod mušaraca starijih od 40 godina sa AIM visoko stistički značajno niži u odnosu na nivoe T kod zdravih muškaraca iste životne dobi. Nivoi T kod ispitanika sa AIM zadržavaju statistički značajno niže vrednosti i šest meseci nakon AIM u poređenju sa zdravom kontrolnom grupom.

Ključne reči:
lipidi; lipoproteini; infarkt miokarda; testosteron.

Introduction

Androgens as well as estrogens show influence on many risk factors related to cardiovascular diseases (CVD) [1]. The basic risk factors for CVD are: hypercholesterolemia, low lovel of high density lipoprotein (HDL) cholesterol, high level of low density lipoprotein (LDL) cholesterol, hypertension and cigarette consumption. Epidemiological studies have shown that each of these factors is of high importance depending on the degree of exposure. The common feature of these factors is their ability to damage the arterial endothelium. Hypertension produces an increased mechanical stress on blood vessels. Cigarette smoking causes transient but intensified release of free radicals into the arterial system, and oxidized cholesterol can act as endothelial toxin [2]. Besides conventional risk factors including: diabetes mellitus, positive family history and age, the additional factors such as abdominal obesity, alcohol consumption and physical inactivity could be added; those risk factors represented the focus of research in many studies, e.g. INTERHART, a global risk factors study for acute myocardial infarction (AMI) [3]. Major studies concerning risk factors for CVD (INTERHART, AMORIS, MONICA/CORA), focused special attention to the role of apolipoproteins as informative indicators for CVD and AMI, primarily apolipoprotein B (Apo B) and apolipoprotein A1 (Apo A1) [4].

The influence of androgens on lipid status and interpretation of results obtained is extremely controversial. The fact that androgens usually reduce levels of HDL cholesterol has been used throughout history to characterize these steroids as harmful to blood vessel health [5]. But, along with these findings, it has been noticed that reduction of lipoprotein(a) [Lp(a)] level and plasma triglycerides could lead to a reduction of CVD risk [1].

Although risk factors for CVD do not appear to be isolated, and cholesterol and triglycerides metabolism is highly interconnected, the fact that triglycerides concentrations vary day by day in an individual to a much greater extent compared to cholesterol concentrations, cholesterol level was marked as stronger predictor for CVD [6].

In puberty boys, the increase in testosterone concentrations was followed by a decrease in HDL cholesterol concentrations, probably as a result of hepatic lipase induction, a sex hormone sensitive enzyme of the lipoprotein metabolism. This decline in HDL cholesterol levels represents the basic difference and a higher risk for early development of CVD in men compared to women. Unlike the puberty period in men, in the later years there is a positive correlation between concentrations of testosterone and HDL cholesterol due to the influence of testosterone on the hepatic synthesis of Apo A1 [7].

The effect of androgen on levels of LDL cholesterol in plasma, which represents a classic metabolic risk factor in men, is difficult to be interpreted and analyzed. In some men who abused anabolic–androgenic steroids (AAS), extremely high values of LDL cholesterol were found, indicating an elevated risk for CVD. In contrast, an increase in LDL cholesteril did not occur in patients who used androgens for the purpose of contraception or substitution therapy, whereas in one of studies, decline in LDL cholesterol levels was observed in patients who abused AA [6].

Although LDL cholesterol is known as the primary lipid risk factor for CVD, there are several limiting factors for using only it as a main risk factor. Recent data suggest that apolipoproteins are important indicators and predictors for CVD primarily Apo A1, which represents anti-atherogenic high density lipoprotein. Several studies, including two major AMORIS [7] and INTERHART [8], as well as MONICA/KORA STUDY [9], showed strong direct relationship between high levels of the Apo B/Apo A1 ratio and the increased risk of fatal AMI. Apo B is found in very low density lipoproteins (VLDL), medium density lipoproteins (IDL), as well as in large boyant LDL and sd-LDL, with one molecule of Apo B in each of these atherogenic particles. Therefore, the total number of Apo B reflects the total number of atherogenic particles. Apo B also plays role in the "capture" of these lipoproteins in the walls of blood vessels. Apo B synthesized in the liver also stabilizes and allows the transport of cholesterol and triglycerides in VLDL, IDL to large boyant LDL and plasma sd-LDL. Apo B serves as a ligand for Apo B and Apo E receptors and thus facilitates cholesterol intake in peripheral tissues and liver. Apo A1 is the main protein of HDL particles and is the major initiator of reverse cholesterol transport. The balance between Apo B and Apo A1, as well as the Apo B/ Apo A1 ratio increase the risk of CVD, the higher ratio the higher is risk.

In addition to the standard lipid profile parameters as well as the above mentioned apolipoproteins, Lp(a) which originates from LDL modification, may also be one of the predictors of CVD and AMI. Due to its structural similarity to plasminogen, Lp(a) impairs plasma synthesis and the fibrinolysis process [10]. Lp(a) also plays a role in macrophage binding through high affinity receptors, which leads to the

formation of foam cells and discharge of cholesterol into atheromatous plaques [11]. The correlation between Lp(a) and risk for CVD and AMI was first suggested in some cross-sectional and prospective studies, while in some studies contradictory results were obtained. In a prospective PROCAM study which included 788 men aged 35–65, with follow-up period of 10 years, the risk of acute coronary events was 2.7 times higher in patients with Lp(a) levels > 20 mg/dL [12].

The aim of the study was to determine levels of testosterone in men older than 40 years in AMI and six months after AMI, and to compare with testosterone levels in healthy men of the same age. Another aim was to examine correlation of testosterone levels with lipid profile prameters in men over 40 years of age in AMI and six months after AMI.

Methods

The study was designed as prospective clinical study. Clinical examination and recruitment of participants were conducted at the Clinic for Endocrinology, Diabetes and Metabolic Diseases of the Clinical Center of Serbia and the Clinic for Cardiology of the Emergency Center (Coronary Unit) in Belgrade. Laboratory analyses were performed in the Center for Medical Biochemistry of the Clinical Center of Serbia in Belgrade.

Patients were divided into three groups: group I included 35 men aged 40–80 years with AMI; group II included the same 35 men who were analyzed six months after the AMI; group III (control group) consists of 20 healthy men aged 40–80 years.

All groups were homogeneous concerning body mass index (BMI) and age. Total ischemic time in the group I was shorter than 12 hours. All participants were taken blood samples early in the morning for the following analyses of lipid profile: total cholesterol, triglycerides, low LDL cholesteril, HDL cholesterol, Lp(a), Apo A1 and Apo B. Hormone analysis included levels of testosterone.

Hormone and lipid parameters were determined immediately in AMI event (in patients of the group I) as well as six months after discharge from the hospital (the group II).

All patients were informed concerning the methodology of the study and all of them voluntarily filled out the informed consent. The study was approved by the Ethics Committee of the Clinical Center of Serbia.

Biochemical analyses were performed by chromatography methods, and tetsosteron levels by radioimmunoassay (RIA).

Results were reported as mean ± standard deviation and presented in tables. Differences between groups were assessed by Student's *t* test. Correlations between parameters were analyzed with Spearman's correlation test. Differences were considered statistically significant at $p < 0.05$. SPSS 20.0 software was used for the statistical analyses.

Results

The group of patients with AMI and the control group were homogenous in BMI and age, and no statistically significant differences were find between them for BMI (28.40 ± 2.84 kg/m^2 vs. 26.45 ± 2.01 kg/m^2, respectively) and age (55 ± 3 years vs. 57 ± 2.12 years, respectively).

Testosterone levels in patients of the group I (16.86 ± 7.18 nmol/L) were statistically significantly lower than those in the control group (27.11 ± 10.48 nmol/L) ($p < 0.001$). Also, highly statistically significant difference was obtained by comparing testosterone levels in patients 6 months after AMI (the group II) (18.12 ± 7.96 nmol/L) with those in the control group (27.11 ± 10.48 nmol/L) ($p < 0.001$). No statistically significant difference was found between testosterone levels in patients with AIM and 6 months after AMI (Table 1).

Statistically significantly higher levels of cholesterol, LDL cholesterol and Apo B were obtained in AMI patients (the group 1) compared to those 6 months after AMI in the group 2 ($p < 0.05$) (Table 2). All these three values were slightly increased in the group I, comparing with referent range for cholesterol (3.1–5.1 mmol/L), LDL cholesterol (1.55–3.4 mmol/L) and Apo B (0.66–1.33 g/L).

Correlations of levels of testosterone and parameters of the lipid profile in patient with AMI and six months after AMI are given in Table 3.

In the group I, statistically significant, positive correlation was found between levels of testosterone and HDL cholesterol ($p < 0.05$), as well as testosterone and Apo B ($p < 0.01$).

In the group II, statistically significant positive correlation was also found between levels of testosterone and HDL cholesterol ($p < 0.05$) as well as testosterone and Apo A1 ($p < 0.05$).

Table 1
Testosterone levels in patients with AMI (the group I), 6 months after AMI (the group II) and in the control group (the group III)

Groups of patients	Testosterone levels (nmol/L)			
	min	max	mean	SD
I (n = 35)	1.52	48.62	16.86	7.18
II (n = 35)	6.88	46.12	18.12	7.96
III (n = 20)	15.80	49.04	27.11	10.48

AMI – acute myocardial infarction; SD – standard deviation.

Table 2

Lipid parameters in patients with AMI (the group I) and 6 months after AMI (the group II)

Parameters	Group I	Group II	
	mean ± SD	mean ± SD	*p*
Total cholesterol, mmol/L	5.75 ± 1.33	4.70 ± 1.29	< 0.05
HDL cholesterol, mmol/L	1.13 ± 0.31	1.05 ± 0.29	> 0.05
LDL cholesterol, mmol/L	3.70 ± 1.11	2.79 ± 1.23	< 0.05
Triglycerides (mmol/L)	1.89 ±1.52	1.98 ± 1.13	> 0.05
Apolipoprotein A1 (Apo A1), g/L	2.39 ± 0.69	2.02 ± 0.46	> 0.05
Apolipoprotein B (Apo B), g/L	1.62 ± 0.73	1.11 ± 0.41	< 0.05
Apo B/Apo A1	0.66 ± 0.23	0.59 ± 0.23	> 0.05
Lipoprotein(a), g/L	0.47 ± 0.21	0.38 ± 0.26	> 0.05

AMI – acute myocardial infarction; HDL – high density lipoprotein; LDL – low density lipoprotein; SD – standard deviation.

Table 3

Correlation of levels of testosterone with levels of lipid parameters in patients with AMI (group I) and 6 months after AMI (group II)

Lipid parameters	Group I		Group II	
	r	*p*	r	*p*
Total cholesterol	0.318	> 0.05	0.184	> 0.05
HDL cholesterol	0.403	< 0.05	0.388	< 0.05
LDL cholesterol	0.268	> 0.05	0.255	> 0.05
Triglycerides	-0.052	> 0.05	-0.065	> 0.05
Apolipoprotein A1 (Apo A1)	0.747	< 0.01	0.354	< 0.05
Apolipoprotein B (Apo B)	0.298	> 0.05	0.133	> 0.05
Apo B /Apo A1	-0.118	> 0.05	-0.079	> 0.05
Lipoprotein(a)	0.281	> 0.05	0.328	> 0.05

AMI – acute myocardial infarction; HDL – high density lipoprotein; LDL – low density lipoprotein; r – coefficient of correlation.

Discussion

Men, unlike women, do not experience a sudden decrease in concentration and production of sex hormones in middle age, but a gradual decline of endogenous testosterone has been present since 30 years of every man's life. Research of the role of testosterone in maintenance of male health, as a new field of endocrinology, occurred in 1998 at the first world congress "Aging Male". During the past 20 years, decrease in testosterone levels has gone from "Andropause" to "late onset hypogonadism – LOH" and, ultimately, the involutive hypoandrogenism as the most acceptable definition of changes in concentrations of endogenous testosterone in the aging process in men. The problem with the name and nomenclature is only part of the controversy associated with testosterone and its role in the development of various pathological processes in men.

Many large-scale studies with a large number of participants tried and partly managed to give an answer on the role of sex hormones and their impact on the cardiovascular system in women, but in men this mostly was not the case. For this reason, and especially because of the diametrically different results obtained in animal models and in some smaller studies, over the past 10 years, increasing attention has been paid to the role of sex hormones in the prevention, treatment and occurrence of CVD and AMI in men [13].

Testosterone in AMI

In the last few years, several studies, trials, and case studies reported an increased risk of developing AMI in men who received testosterone [14, 15]. Thus, in a study of Layton et al. [15], 2,898 patients with coronary events demonstrated an increased risk of AMI, cardiovascular insult and unstable angina pectoris immediately after testosterone injection [15]. What has always provoked controversy concerning levels of testosterone is the question what are the appropriate, "normal" values of testosterone levels depending on the age. Avoiding supraphysiological doses and maintaining a physiological balance potentially unwanted effects of testosterone are omitted. For this reason, in recent years one of the largest studies, a retrospective cohort study of Li et al. [16] compared occurance of AMI in 200,000 participants receiving testosterone therapy with that in 200,000 hypogonadic patients who did not receive testosterone therapy over a one-year period, and no association between testosterone therapy and AMI was found. In favor of the positive effect of substitution therapy with testosterone in hypogonadal males, a large cohort study of Cheetham et al. [17], conducted on 8,808 individuals, reported smaller risk of developing AMI in the follow-up period of 3.4 years.

What differentiated our study from recent trials was that we monitored levels of testosterone in patients with AMI and

six months after AMI, as well as in age and BMI comparable group of healthy men. The obtained results showed highly significantly lower levels of testosterone not only in the ischemic period of 12 hours from the onset of AMI but six months after the acute phase as well, compared to the healthy control group.

Lipids as risk factors for CVD and AMI

Lipid status with all its components (cholesterol, HDL cholesterol, LDL cholesterol, Lp(a), triglycerides, Apo A1, Apo B) was completely processed and statistically analyzed in order to determine its correlations with concentrations of endogenous testosterone in patient with AMI and 6 months later.

Statistically significantly higher values were demonstrated for LDL cholesterol and cholesterol in subjects with AMI compared to values found after six months in the same subjects.

HDL cholesterol with its anti-atherogenic effects marks one of frequent controversies associated with levels of endogenous testosterone and its influence on HDL cholesterol level [18]. The evident decline in HDL cholesterol in puberty is associated with testosterone jumping (negative correlation) due to the induction of sex hormone sensitive enzyme of lipoprotein metabolism, hepatic lipase, is one of the main causes of the early onset of CVD in men compared to women [7]. Contrary to this, in many studies a positive correlation between levels of endogenous testosterone and HDL cholesterol has been demonstrated in older man [19], which we also confirmed in our study. In our study statistically significant positive correlation was observed between levels of testosterone and HDL cholesterol in AMI patients (the group I), as well in the group II, six months after AMI. This positive correlation can be explained by hepatic effect of testosterone on the production of Apo A1. The Massachusetts male aging study (MMAS) showed a positive and highly statistically significant correlation of HDL cholesterol levels and levels of endogenous testosterone in males over 40 years of age with or without CVD, thus definitely confirming the fact that there is difference of endogenous testosterone effect on HDL cholesterol and risk factors in older men compared to men immediately after puberty [20]. Similar results were obtained in the San Antonio Hearth Study, where a positive correlation between levels of endogenous testosterone and HDL cholesterol in 178 men with normal glycemic values was demonstrated. It was concluded that the less atherogenic lipid profile (lower triglyceride values and higher HDL cholesterol values) was present in men with a higher concentration of endogenous testosterone vs. women in whom the increased concentration of androgens was in a strong correlation with high levels of triglycerides and low HDL values [21].

There were no statistical significant correlation between testosterone levels with the levels of triglycerides. A negative correlation was obtained, which, although not statistically significant, corresponded in many ways to the results of large studies. Tromso Study also dealt with the effect of endogenous testosterone on levels of triglycerides during the day in 1,274 men who did not have a verified CVD and who participated in the population study. Analyzing triglyceride levels taken during the day, their linear increase has been demonstrated in subjects with endogenous testosterone levels below 50th percentile. On the contrary, in men with values of endogenous testosterone above 50th percentile there were no statistically significant changes in triglyceride levels during the day. Also highly statistically negative correlation was found between levels of triglycerides and endogenous testosterone and it was highly statistically positive related to HDL cholesterol. Men with poor lipid profile (HDL cholesterol < 0.9 mmol/L and triglicerides > 1.8 mmol/L) had significantly lower testosterone levels compared to men with normal lipid profile [22]. The conclusion of this large study was that low level of endogenous testosterone correlates with the linear rise in triglycerides during the day, and that it is independently associated to a poor lipid profile indicating that low levels of testosterone affect the poor triglyceride metabolism.

LDL cholesterol represents one of the risk factors for the development of CVD and, unlike HDL cholesterol, shows positive correlation with that risk [23]. In addition, the role of Lp(a) as an important risk factor for the development of CVD has been highlighted over the past years. Due to the structural similarity with plasminogen, as well as its binding properties with high affinity macrophages and the formation of foam cells, Lp(a) directly affects the development of CVD [24]. In our study, we did not find statistically significant correlation between levels of testosterone and levels of LDL cholesterol and Lp(a) in patients with AMI as well as six month later in the same patients.

In our study we found statistically significant, positive correlation between testosterone levels and levels of Apo A1 in both AMI groups (the group I and the group II). The role of Apo A1, Apo B, as well as their ratio (quotient) in development of CVD and AMI is known from major studies such as AMORIS [7], INTERHART [8] and MONICA/CORA [9].

The AMORIS study showed that high levels of Apo B highly correlated with an increased risk of developing CVD and AMI, while the level of Apo A1 had a protective role in both men and women. In that prospective study, more than 175,000 men and women of the Swedish population were monitored during 98 months, nearly 2,000 of them died due to AMI. Apo B was labeled as a stronger marker for CVD than LDL cholesterol, and especially for subjects with normal/lower LDL cholesterol values [7]. A single variable representing the strongest indicator for the occurrence of fatal myocardial infarction was the Apo B/Apo A1 ratio. This ratio was an indicator of the risk of fatal myocardial infarction, independently of lipid phenotype, especially when other lipid levels were normal or low [25]. This ratio was a stronger risk factor for CVD compared to all other ratios: triglicerides/HDL cholesterol, LDL cholesterol/HDLcholesterol or non-HDL cholesterol/HDL cholesterol [26].

The impressive INTRHARTH study, based on 30,000 patients from 52 countries all over the world, also showed that the Apo B/Apo A1 ratio was the strongest risk factor among the other conventional risk factors for AMI [8].

Several other studies have confirmed that the Apo B/Apo A1 ratio is in a strong correlation with increased ca-

rotid artery intima-media thickness and that this ratio progressively increases in patients with metabolic syndrome. This ratio was in a positive correlation with the CVD risk, described as the first or myocardial reinfarction [27].

The MONICA/Cora Study included 1,414 men and 1,436 women aged 35–64 years without a previous history of myocardial infarction. The period of follow-up was in average 13 years, during which 114 men and 31 women had a coronary event, of which 71 were fatal and 74 were not. The strongest correlation was demonstrated for high Apo B levels as well as Apo B/Apo A1 ratio and risk for myocardial infarction [4]. The results of that are completely coherent with those obtained in the INTERHART study, based on 15,000 AMI patients compared to 15,000 healthy controls. Both studies have shown that Apo B/Apo A1 ratio is the most important among all risks factors besides: smoking, hypertension, abdominal obesity, diabetes, alcohol, psycho-social stress, vitamin intake, and physical inactivity. The results were independent concerning gender, age and ethnicity. The Apo B/Apo A1 ratio remained the strongest risk factor after the multivariate analyses were performe [8].

In the last few years, numerous studies estimated the role as well as the significance of the Apo B/Apo A1 ratio concerning CVD and AMI incidence. The study published in 2015 explored the predictive value of Apo B/Apo A1 ratio and non-HDL cholesterol values and their effects on CVD incidence [28]. The study was conducted on 826 patients, of whom 532 had CVD, 165 of them unipolar, 175 bipolar disorders, and 192 multipolar CVD vs. 294 healthy subjects. After a follow-up period of 3 years, it has been confirmed that there is a positive correlation among high values of the Apo B/Apo A1 ratio and non-HDL cholesterol with the most serious, multiply forms of coronary heart disorders and the increased risk of developing adverse events such as: angina pectoris, AMI, heart insufficiency, stroke and death caused by CVD.

Statistically significantly higher Apo B values we found in the patient with AMI compared to the same subjects six months after myocardial infarction.

Negative, but not statistically significant correlation of endogenous testosterone levels and the Apo B/Apo A1 ratio we found in both AMI groups (groups I and II).

Analyses of studies conducted so far as well as the results of our study suggest that natural endogenous testosterone has a positive or neutral effect on the development of CVD and AMI. The antiatherogenic mechanism of testosterone is unknown, in general, but several solutions have been offered so far. Some data emphasize the modulating effect of endogenous testosterone on the risk factors for CVD: diabetes [29], insulin resistance [30], obesity [31], hypercholesterolemia [32, 33], and hypertriglyceridemia [32]. It has been assumed that increase of triglycerides levels is modified by changes in hepatic triglyceride lipase [34]. On the contrary, endogenous testosterone can have a direct effect on HDL cholesterol by increasing the hepatic production of Apo A1 as the main protein component of nascent high density lipoprotein [28].

Conclusion

This study showed that men over 40 years of age with AMI have highly statistically significantly lower concentrations of endogenous testosterone compared to healthy male population of the same age. Statistically significantly lower concentrations of testosterone are maintained even six months after AIM. In our study, highly statistically significant, positive correlation was found between levels of endogenous testosterone and levels of HDL cholesterol and Apo A1 in men with AMI as well in the same patients six months after AMI. Long-term, well designed prospective clinical trials are required to verify potential testosterone role, its interaction with parameters of the lipid profile and possible predictive value in men with AMI.

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