UC 577,1; 61 ISSN 0354-3447

Jugoslov Med Biohem 24: 99–106, 2005 *Originalni naučni rad*
Original paper

OXIDIZED LDL AND OTHER LIPIDS AS RISK FACTORS FOR CARDIOVASCULAR DISEASE IN THE PATIENTS WITH METABOLIC SYNDROM

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Summary: We estimated relationship between lipids, LDL oxidation, antioxidant activity and CRP in diabetic patients with metabolic syndrom (MS), with and without coronary heart disease (CHD), in non diabetic patients with and without CHD and in obese patients, with and without diabetes. We didn't find significant difference in lipids among diabetic MS patients. Patients from all subgroups have simillar level of oxLDL, but significant higher comparing with healthy control. MS diabetic patients have oxLDL in positive corelation with TC, LDL-C, non HDL-C and apo B 100, as vell as with molar ratio LDL-C/HDL-C and TG/HDL-C ($p<0.001$). Among non diabetics, CHD patients had higher Lp(a), but oxLDL was simillar in both subgroups. In the nondiabetics we found correlation between oxLDL, TC, LDL-C and TG ($p<0.01$) and apo B 100 ($p<0.001$), but not with TG/HDL-C molar ratio. Obese patients from both groups had simillar lipids profile but, oxLDL was higher, not significant, in non diabetics. We didn't find significant differences in antioxidative activity and CRP in both diabetics MS subgroups, and both obese subgroups. Nondiabetics with CHD have lower E-SOD and E-GPX activity and higher level of CRP than non CHD patients ($p<0.05$). MS diabetics and non diabetics didn't have significant correlation between levels of oxLDL and CRP, but CHD patients (with or without diabetes) had ($p<0. 01$).

Key words: diabetes mellitus type 2, coronary heart disease, lipids, oxLDL, antioxidative status

Introduction

The metabolic syndrome (MS) comprises a constellation of factors associated with adverse outcomes that are relevant to diabetologists and cardiologists. Although recognized several decades ago, the multifactorial nature of the MS was fully appreciated by Raveen GM (1). Development of diabetes coexisting with obesity, hypertension, and disturbance between fibrinolytic and coagulation activity have genetic predisposition. Environmental nutritional factors, excess of weight and sedentary life stile also appears to be important in the development of this clustering of cardiovascular risk factors as a metabolic syndrome. Lipids disorders and diabetes mellitus play very important role for atherogenesis. On the onsets of diabetes type 2 many patients already have cardiovascular complications.

The prevalence of the metabolic syndrome vary according to the definition. There are tree definitions for MS: WHO criteria (2), European Group for The Study of Insulin Resistance Criteria (3) and USA ATP III Criteria (4). The criteria for the definitions are different. According with National Health and Nutritional Survey datas, in the USA prevalence of the MS was 23.7% (5). Applying WHO criteria in Finland and Sweden about 10% of persons with normal glucose tolerance, 40% of persons with Impaired glucose tolerance (IGT) and 70% of patients with type 2 diabetes had MS (6).

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Many different genetic factors play important role in development of metabolic syndrom, as a »thrifty gene«, and Peroxisome Proliferator Activated Receptors (PPARs). The α and γ subfamily of these nuclear receptors are important for lipid and glucose metabolism as well as for inflammation. PPARα in the liver and in the muscles play important role in synthesis, transportation and oxidation of fatty acids (7). PPAR controls glucose metabolism, insulin sensitivity, adipogenesis, and cellular differentiation, fatty acids (FA) up taking and triglyceride (Tg) storage (8). Activated PPARs regulated the transcription of several genes, including those that can increase expression of apo A-I and Apo-II which results increase of HDL and gene for synthesis lipoprotein lipases (LPL). Previous studies, Framingham study (9) and PROCAM (10) have shown that increase of total cholesterol (TC) and LDL cholesterol, and decrease of HDL cholesterol are important for atherosclerosis. However, important predictive impacts for forthcoming cardiovascular events have elevated concentration of small dense LDL particles, and molar ratio of TC/HDL (11, 12). Susceptibility to oxidation of small dense LDL makes them very atherogenetic. In diabetes these particles are glycated and even more susceptible to oxidative modification. The most important step in atherogenesis is oxidative transformation of LDL particles and their clearance trough scavenger's receptors of endothelial cells and macrophages (13, 14). The levels of reactive radicals are increased if here is high level of oxidative substrate (lipids, glucose), as it is in diabetes, or low level of antioxidants. Oxidized LDL activate HMG Co A reductase and suppress LDL receptor gene, resulting in further increasing of already high LDL level. Macrophages uptake oxidized LDL and become lipid-laden foam cells (15).

The aim of this study was to estimate cardiovascular risk factors (lipids level, oxidative stress level and inflammation reaction) in patients with metabolic syndrome. Lipids level was analyzed by measuring total and HDL cholesterol, and calculating LDL cholesterol, measuring of Lp (a), triglycerides, apo lipoproteins B100, A1, A2 and E. Level of oxidative stress was estimated by measuring oxidized LDL and levels of antioxidative enzymes (E-SOD, E-GPx and Plasma Total Antioxidative Status P-TAS). Inflammation reaction was determined by measuring C-reactive protein (CRP) and fibrinogen.

Material and Methods

Patients aged 45 62 years (n = 197) matched by gender and smoking habit participated in the study. Among this patients we had patients with diagnosed metabolic syndrome (MS), patients with CHD without MS and obese patients with and without diabetes. The MS was diagnosed according with USA ATP III criteria (4). The existence of any three of next listed criteria, constitutes a diagnosis of the MS: waist circumference for men > 102 cm, for women > 88 cm; plasma triglyceride > 1.69 mmol/L; HDL cholesterol for men < 1.04 mmol/L, for women < 1.29; blood pressure > 130/85 mmHg; serum glucose > 6.1 mmol/L. Subjects with TG > 4.5 mmol/L were excluded from the study.

The study was conducted in six groups of patients: group A1: 40 MS patients with type 2 diabetes and coronary heart disease (CHD); group A2: 40 MS patients with type 2 diabetes, without CHD; group B1: 31 patients without diabetes with CHD; group B2: 30 patients without diabetes and CHD; group C1: 30 obese non MS patients without type diabetes; group C2: 26 obese, MS patients with type 2 diabetes.

Blood samples were drawn after 12–14h fast. Serum total cholesterol (TC), HDL cholesterol (HDL-C), and triglycerides (TG) were measured by enzymatic methods. LDL cholesterol (LDL-C) concentrations were calculated using the Friedewald formula. Lipoprotein Lp(a), apoproteins AI, AII, B 100 were measured by nephelometry. Oxidized LDL (ox-LDL) were measured by enzyme linked immunosorbent assay (Mercodia, Sweden) (16). This is solid phase two-site enzyme immunoassay, based on the direct sandwich technique in wich two monoclonal antibodies are directed against separate antigenic determinants on the oxidized apoprotein B molecule. During incubation oxidized LDL in the sample reacts with anti-oxidized LDL antibodies bound to microtitration well. After washing, that removes non-reactive plasma components, a peroxidase conjugated anti-apoprotein B antibody recongniyes the oxidized LDL bound to the solid phase. After a second incubation and a simple washing step that removes unbound enzyme labeled antibody, the bound conjugate is detected by reaction with 3,3',5,5'-tetramethylbenzidine. The reaction is stopped by adding acid to give a colorimetric endpoint that is read spectrophotometrically. The values were expresed in IU/L.

Antioxidant status was estimated by the activities of Cu-Zn Erytrocyte Superoxide Dismutase (EC 1.15.1.1, E-SOD), Se-dependent Erytrocytes Glutathion Peroxidase (EC 1.11.1.9, E-GPX) and Plasma Total Antioxidative Status (P-TAS) using commercial assays developed by Randox Laboratories, based on methodes bz Goldstein, Paglia and Muller (17–19). The p-TAS was determined by the Total Antioxidant Status test after plasma separation. The reagents were used on a Monarch plus automatic analyzer. CRP were measured by nephelometric method.

Atherogenic markers were calculated as high risk values, according with previous recommandations (20): TC / HDL-C > 4.25; LDL-C/HDL -C > 2.90; non HDL -C (TC - HDL -C) > 3.86 and apo B100/apo AI >0.6. Relation of TG/HDL-C was also calculated and used as estimate level of small dense LDL. Cut off 1.33 level of this relation was used as indication of LDL particles size according with previous findings

(21). Ishaemic heart disease was verified according to electrocardiogram (ECG) changes (Minnesota codes 1.1-3, 4.1-4, 5.1-3).

The values are given as means ± SD. The group frequencies were compared by x^2 or Fisher's exact tests. Spearman rank correlations were used to demonstrate relationships between variables. A multiple regression analysis was carried out with CHD, diabetes and obesity as dependent variables and age, sex, and the metabolic syndrome or its components as independent variables. In the multiple regression analysis, cholesterol was used as continuous variables, and the other variables were used as categorical variables. The statistical analyses were performed with an SPSS program for Windows. P value < 0.05 was considered statistically significant.

Results and Disscusion

The study was performed among type 2 diabetic patients with and without CHD, nondiabetics with or without CHD and obese patients with or without diabetes and healthy controls. Diabetes and obesity are independent risk factors for CHD. Ishaemic heart disease was verified according to electrocardiogram ECG changes and according to medical history for myocardial infarction. The group was matched according to gender. The clinical characteristics of subjects are given in *Table I*.

All diabetics were treated with oral medication, the duration of diabetes was 8.5 ± 1.5 yrs. At the moment of study they had poor metabolic control (HbA1c > 7.7%). More than 50% of participants have hypertension, exept in the group of healthy controls. Smoking habits was among 70%.

Lipid profiles were shown in *Table II*. There was no statistical difference in the levels of TC, HDL-C and LDL-C as well as TG between the groups. However, only in the group B1 we found higher level of Lp(a) compare with the levels in group B2 and compare with all other groups ($p < 0.05$). Mean values of TC, LDL-C and non HDL-C were higher than are recomended by NCEP ATP III values in all groups (20). In all groups mean values of TC were above 6.2 mmol/L and mean values of LDL-C was above 4.1 mmol/L and are higher than in this recomendation (*Table II*). In all groups mean values of HDL-C were above 1.0 mmol/L, but there was great variety among the groups, SD was very high. However TC, LDL and non HDL cholesterol were higher among diabetics, esspecialy those with CHD, but there was no statistical significant difference between groups. The lowest values of HD-C was among diabetics with CHD and obese patients but there were no significant difference comparing with other groups. In all groups there was found high level of non HDL-C (*Table II*). All mean values were in range of high atherosclerotic risk (3.86 mmol/L). In all groups there was found

Table I Clinical characteristics of patients

Group	N	Age years	Sex ratio M/F	BMI kg/m^2	Waist circumference (cm)	Hypertension < 130/85 mmHg
A1	40	52.3 ± 5.5	18/22	28.23 ± 4.32	95.76 ± 9.94	24 (60.0%)
A2	40	54.5 ± 3.8	19/21	26.61 ± 4.26	92.48 ± 5.61	21 (50.2%)
B1	31	55.2 ± 4.4	17/14	27.31 ± 3.31	96.12 ± 13.52	19 (63.3%)
B2	30	56.3 ± 5.5	15/15	25.85 ± 3.49	90.91 ± 11.64	12 (38.0%)
C1	30	55.5 ± 3.7	14/16	28.89 ± 2.39	92.50 ± 1.81	16 (53.3%)
C2	26	54.5 ± 2.5	14/12	30.28 ± 3.72	95.58 ± 12.14	22 (84.5%)

Table II Basal values of plasma lipids

Lipids	Groups of study subjects					
	A1	A2	B1	B2	C1	C2
Tot. Cholesterol–TC (mmol/L)	6.38 ± 1.04	6.47 ± 1.73	6.80 ± 1.32	6.84 ± 1.67	6.19 ± 1.81	6.22 ± 1.45
HDL Cholesterol–HDL-C (mmol/L)	1.26 ± 0.36	1.35 ± 0.32	1.31 ± 0.41	1.36 ± 0.42	1.26 ± 0.39	1.31 ± 0.32
LDL Cholesterol–LDL-C (mmol/L)	4.27 ± 0.85	4.30 ± 1.74	4.42 ± 1.43	4.56 ± 1.44	4.75 ± 1.47	4.89 ± 1.67
Triglycerides–TG (mmol/L)	2.97 ± 1.99	2.47 ± 2.21	2.52 ± 1.74	3.16 ± 1.09	3.44 ± 2.54	2.76 ± 0.92
Non HDL Cholesterol (mmol/L)	5.25 ± 0.94	5.17 ± 1.76	5.42 ± 1.50	5.20 ± 1.65	5.66 ± 1.64	4.89 ± 1.09
Lp(a) (nmol/mL)	0.32 ± 0.33	0.31 ± 0.33	0.74 ± 0.32	0.31 ± 0.24	0.29 ± 0.27	0.26 ± 0.32
Significance of differences $p < 0.05$ differences between B1 and B2						

high values of TG according to recomendation of NCEP ATP III (2.3 mmol/L). The highest level of TG was found in obese diabetics and diabetics with CHD, but these differences were not significant. These patients groups did not differ significantly in regard to apolipoproteins (*Table III*). Moreover, the highest levels of apo A II was found in healthy control, group B2. Interestingly, the difference observed when patients with diabetes and obesity were compared with other groups according to level of apo B100 was insignificant, but the values were lower. At the same time obese patients had higher levels of this apoprotein. Apo E was the lowest in group A2 and the highest in group C1. According to our results diabetes per se and obesity have proaterogenetic lipid profile.

Previuos studies have shown that oxidized LDL has direct atherogentic effect. We haven't found any statisticly significant difference among diabetics irrespectively to CHD, but it was slightly higher among diabetics with CHD. Also there was no significant difference among healthy and group CHD patients. We found high level of oxLDL in obese nondiabetic patients (*Table IV*). Our results have shown that oxidative modification were occured in all groups on the same level irrespectively to presence of CHD. All measured values were above the reference of healthy control in our study (ox LDL: 78.51±14.50 IU/L).

Radke, Libby and coworkers (22, 23) demostrate that molar ration of TC/HDL-C and CRP have great predictive value for CHD. Ratio of TC/HDL-C above 4.25 mark high atherogenic risk. In all groups we found this relation above 5, and it was the highest ih group of obese patients with diabetes. Diabetics with CHD had significantly higher TC/HDL-C relation then diabetics without CHD, group A1 in compare with group A2 (*Table IV*), ($p<0.05$). Nondiabetics with CHD had this relation higher, but not significantly comparing with nondiabetics without CHD. In groups of obese patients we found significantly higher realation in group with diabetes ($p<0.01$). Relation of LDL-C/HDL-C was above 2.90 in all groups, which imply higher atherogenic risk, but we didnt find significant difference between these groups (*Table IV*). However, this relation was the highest among nondiabetics with CHD and obesity.

Prospective studies have established that small dense LDL particles is the best predictor of future CHD in diabetic patients (24, 25). Their atherogenicity is coming from their susceptibility to oxidative modification. Some studies have shown that molar ratio of TG/HDL-C 1.33 can be use as indicator of LDL particle size (23). We have found this ratio over 1.33 in all our groups, the highest in group of diabetics with CHD and obese diabetics (*Table IV*). There was significant difference of this molar ration between diabetics with and without CHD and between nondiabetics with or without CHD ($p < 0.05$). On the other hand, in group of obese diabetes patients this ratio was higher comparing to group of obese nondiabetics, but this difference was not statistical significant.

In all our groups level of oxidative modification was similar taking account the levels of oxLDL and atherogentic LDL-C/HDL-C ratio. Other atherogenic ratio Tg/HDL-C was clearly combined with diabetes

Table III Plasma Apolipoproteins

Apoproteins	Groups of study subjects					
	A1	A2	B1	B2	C1	C2
Apo A I (g/L)	1.77 ± 0.35	1.75 ± 0.38	1.69 ± 0.46	1.82 ± 0.38	1.64 ± 0.41	1.63 ± 0.39
Apo A II (g/L)	343.60 ± 68.80	343.30 ± 58.70	341.50 ± 91.60	363.90 ± 80.80	358.60 ± 94.50	338.20 ± 60.80
Apo B 100 (g/L)	1.37 ± 0.36	1.39 ± 0.41	1.37 ± 0.34	1.41 ± 0.34	1.52 ± 0.42	1.42 ± 0.32
Apo E (g/L)	80.21 ± 30.13	78.10 ± 34.20	95.30 ± 39.70	93.40 ± 57.30	99.50 ± 69.70	97.70 ± 43.20

Table IV LDL (oxLDL) and atherogenic indexes

Index	Groups of patients					
	A1	A2	B1	B2	C1	C2
Ox LDL(IU/L)	119.3 ± 45.8	106.8 ± 67.8	114.4 ± 38.4	113.0 ± 71.5	133.6 ± 78.2	106.6 ± 44.3
TC/HDL-C	5.57 ± 1.83	5.02 ± 1.55*	5.61 ± 2.64	5.48 ± 1.6	5.06 ± 1.66	6.07 ± 2.34♦♦
LDL-C/HDL-C	3.28 ± 1.83	3.19 ± 1.61	3.87 ± 1.31	3.06 ± 1.72	3.92 ± 1.42	3.00 ± 1.66
Tg/HDL-C	2.23 ± 1.54	1.51 ± 0.87*	2.29 ± 1.16	1.75 ± 1.33 ♣	2.50 ± 1.93	2.72 ± 1.09

Significance of differences:
* $p < 0.05$ between A1 and A2
♣ $p < 0.05$ between B1 and B2
♦♦ $p < 0.01$ between C1 and C2

Table V Oxidized LDL (Ox-LDL) and antioxidant status

Parameters	Groups of patients					
	A1	A2	B1	B2	C1	C2
Ox-LDL(IU/L)	119.3 ± 45.8	106.8 ± 67.8	114.4 ± 38.4	113.0 ± 71.5	133.6 ± 78.2	106.6 ± 44.3
E-SOD (U/g Hb)	988.7 ± 54.4	969.9 ± 72.2	877.5 ± 99.2	1007.1 ± 95.9*	945.0 ± 99.8	985.4 ± 60.7
E-GPX (U/g Hb)	29.92 ± 3.63	27.52 ± 5.10	27.72 ± 5.34	28.24 ± 5.23*	28.41 ± 5.23	29.00 ± 4.64
P-TAS (nmol/L)	1.29 ± 0.19	1.32 ± 0.23	1.18 ± 0.26	1.15 ± 0.30	1.22 ± 0.28	1.33 ± 0.22

Significance of differences:
* $p < 0.05$ between B1 and B2

Table VI Oxidized LDL (Ox-LDL) and inflammation

Parameters	Groups of patients					
	A1	A2	B1	B2	C1	C2
Ox-LDL (IU/L)	119.3 ± 45.8	106.8 ± 67.8	114.4 ± 38.4	113.0 ± 71.5	133.6 ± 78.2	106.6 ± 44.3
CRP (mg/L)	7.03 ± 6.38	6.70 ± 5.93	11.58 ± 15.25	6.19 ± 7.23♣	7.59 ± 5.84	6.50 ± 1.71
Fibrinogen (g/L)	4.61 ± 1.39	3.68 ± 0.73*	4.21 ± 1.31	3.49 ± 0.78♣♣	3.84 ± 1.21	4.14 ± 1.24

Significance of differences:
* $p < 0.05$ between A1 and A2
♣ $p < 0.05$ between B1 and B2
♣♣ $p < 0.01$ between B1 and B2

Table VII Correlations between lipids and oxidized LDL

Correlations	Groups of patients							
	A1 + A2		B1 + B2		A1 + B1		A2+ B2	
	r	p	r	p	r	p	r	p
OxLDL-TC	0.698	***	0.332	**	0.273		0.592	***
OxLDL-LDL -C	0.722	***	0.272	*	0.252		0.602	***
OxLDL-Non HDL-C	0.793	***	0.322		0.328		0.539	***
OxLDL-TG	0.298	*	0.420		0.324	*	0.410	**
OxLDL-LDL/HDL -C	0.686	***	0.011		0.164		0.261	*
OxLDL-Tg/HDL-C	0.494	***	0.174		0.373	*	0.201	
OxLDL-Apo B 100	0.723	***	0.403	***	0.330	*	0.675	**
OxLDL-Fibrinogen	0.318	*	0.196		0.260		0.123	
OxLDL-CRP	0.099		0.139		0.438	**	0.070	

Significance of correlations
* $p < 0.05$
** $p < 0.01$
*** $p < 0.001$

especialy in group with CHD and obesity. We have analyzed antioxidative activity E-SOD i E-GPX, as well as total serum antioxidative status in all groups and the results are shown in *Table V*. We have found significantly higher enzyme activity in group of nondiabetics without CHD ($p<0.05$), in contrary to all other groups. Also, there was no significant difference in TAS among all groups. According to this findings we can conclude that in diabetic and obese patients there is lower activity of both enzymes and TAS, despite presence of CHD. As marker of inflammation process we analyze in our study CRP and fibrinogen (*Table VI*). Mean valeus of CRP was above reference and was higher than 5 mg/L in all studied groups. The highest values was found in group B1, nondiabetics with CHD and was significantly higer when compared with healthy control ($p<0.05$), in spite of high SD (23, 24) which makes our date consistent with other similar studies. Fibrinogen was significantly higher in group of diabetics with veryfied CHD comparing to nondiabetics without CHD ($p<0.05$), moreover in group of nondiabetics with CHD this difference was even higher in comparing with nondiabetics without CHD. At the same time in obese patients gro-

ups there was no significant difference respectively to the presence of diabetes in these groups. These results imply importance of these two markers as a predictors for CHD. In diabetic patients we have found statisticaly significant correlation between levels of ox LDL and TC, LDL-C and non HDL-C, as well as with apo B and LDL-C/HDL -C ratio, ($p < 0.001$). There was also correlation between level of oxLDL and level of TG ($p < 0.05$). We didn't find expected negative correlation between oxLDL and HDL-C. At the same time there was strong correlation between ox LDL and TG/HDL-C ratio ($p < 0.001$). Our results are consistent with published data (25, 26). Previous studies have observed that B type of LDL particles is mostly present in diabetics, even when the level of LDL cholesterol is within the normal range. We didn't find strong correlation between oxLDL and CRP, but in contrast, there was significant correlation between ox LDL and fibrinogen ($p < 0.05$). However, among non diabetics we found significant but not strongly, correlation between ox LDL and TC, LDL-C, non HDL-C and TG, as well as with apo B100. In-group of non-diabetics we didn't find correlation between ox LDL and TG/HDL-C ratio, which imply that in these group of patients is less level of small dense LDL particles. In these groups we didn't find any correlation with inflammatory markers. In group of patients with verified CHD, regardless to diabetes, we found only correlation between oxLDL with TG, apo B 100 and TG/HDL-C ratio ($p < 0.05$), and very strong correlation with CRP ($p < 0.01$). These findings imply predictive values of CRP as a CHD marker. Contrary to previous results we didn't find correlation between oxLDL and TC, LDL, non HDL cholesterol and LDL/HDL ratio in group CHD which is result of statin treatment as a prevention of progression CHD. In groups of patinets without CHD, regardles to diabetes there was strong correlation between ox LDL and other atherogenic risk markers In groups without CHD rhere was no correaltion between oxLDL and inflamatory markers, which confirm previous date that fibrinogen and CRP are independent risk markers for CHD. Inerestingly, we didn't find expected negative correlation between level of oxidative modification of LDL and HDL-C, apo AI and AII. This finding suggest that other factors influence degree of oxidation, especialy poor metabolic control in diabetics, or mostly high levels of small dense LDL particles. Oxidation of LDL particles was higher in all our groups in comparing with healthy control, despite unexpected high levels of HDL cholesterol in all groups. There were no significant correlation between ox-LDL and apo E in all our groups. We haven't find significant difference between oxLDL and antioxidative enzyme nor with TAS in all our groups. We can emphasize that risk groups for developing CHD, such as diabetic or obese patients with or without diabetes do not differ in lipid profile comparing to the patients with verified CHD, irrespectively to the presence of diabetes. However, level of LDL oxidation in diabetics correlates with LDL/HDL cholesterol ratio as well as with TG/HDL-C ratio, whose imply presents of small dense LDL particles. Similar correlations are found among non diabetics, but at the significantly lower level. Oxidized LDL and CRP correlate in patients with CHD irrespectively to presence of diabetes, but do not correlate among non diabetics and obese patients with or without diabetes. This findings confirm significant correlation of oxidative modification of lipids in patients with CHD.

Acknowledgements. The authors thank Mercodia Company from Sweden, for supporting this project.

OKSIDOVANI LDL I RIZIKO FAKTORI ZA KARDIOVASKULARNE BOLESTI U PACIJENATA SA METABOLIČKIM SINDROMOM

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Kratak sadržaj: Ispitan je odnos lipida, oksidacije LDL, antioksidativne aktivnosti i CRP u bolesnika sa metaboličkim sindromom (MS) i dijabetesom, sa i bez ishemijske bolesti srca (IBS), u nedijabetičara sa i bez IBS i u gojaznih bolesnika sa i bez dijabetesa. Kod MS dijabetičara sa i bez IBS nije nađena značajna razlika u lipidima. Pacijenti u svim podgrupama su imali sličan nivo oxLDL, ali značajno viši u poređenju sa zdravom kontrolnom. MS dijabetični bolesnici su imali pozitivnu korelaciju ox-LDL sa ukupnim, LDL i non-HDL holesterolom, sa apo B 100 kao i sa molarnim odnosima LDL/HDL holesterol i TG/HDL holesteroln ($p < 0,001$). Među nedijabetičarima, IBS bolesnici su imali viši Lp(a), dok je oxLDL je bio sličan u obe podgrupe. U nedijabetičara je nađena korelacija ox LDL sa ukupnim holesterolom, LDL holesterolom, trigliceridima ($p<0,01$), kao i sa apo B 100 ($p < 0,001$). Obe podgrupe gojaznih imaju sličan nivo lipida, ali je oxLDL bio viši u gojaznih nedijabetičara. Nije nađena značajna razlika u antioksidativnom statusu i CRP u obe podgrupe MS dijabetičara i u gojaznih. Nedijabetičari sa IBS imali su manju aktivnost E-SOD i E-GPX a viši CRP nego oni bez IBS. Kod MS dijabetičara ali ni kod nedijabetičara nema korelacije između oxLDL i CRP, ali je ona značajna u IBS bolesnika sa ili bez dijabetesa ($p<0,01$).

Ključne reči: Diabetes mellitus tip 2, koronarna bolest srca, lipidi, oxLDL, antioksidativni status

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Received: August 8, 2004

Accepted: November 14, 2004