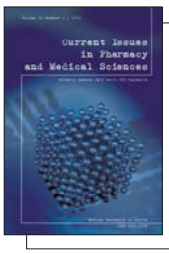

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Lipids and hCRP concentrations in patients with dyslipidemia

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ABSTRACT

Disturbances in lipids, and inflammations plays a key role in the pathogenesis of all stages of atherosclerosis. The aim of present study was to investigate concentration of hCRP, lipids (TC, TG, LDL-C, nonHDL-C, HDL-C) and lipid ratios (TC/HDL-C, LDL-C/HDL-C, TG/HDL-C) in patients with moderate dyslipidemia and compared to the healthy subjects, and relation between hCRP and lipids and lipid ratios. We studied 60 patients with moderately dyslipidemia (male and female) at the age between 21-63 years and the reference group. The reference group constituted of 14 subjects chosen from normolipidemic healthy individuals. The patients with moderate dyslipidemia had significantly increased hCRP, TG, LDL-C levels and significantly increased lipid ratios (TG/HDL, LDL-C/HDL-C). The concentration of hCRP was significantly positively correlated with TG, TC, LDL-C, nonHDL-C concentrations and TG/HDL-C, LDL-C/HDL-C ratios. We concluded that chronic inflammation disease can disturb lipids concentration and can accelerate atherosclerosis. However, these results are required further studies.

Keywords: lipids, hCRP, dyslipidemia

INTRODUCTION

Disturbances in lipids concentration play important role in cardiovascular disease (CVD) [1,6]. The association between CVD and levels of total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C) and high-density lipoprotein cholesterol (HDL-C) has been accepted in diagnostic practice. As a consequence the National Cholesterol Education Program III (NCEP) recommended the analysis of TC in combination with LDL-C and HDL-C as a basis for the screening and treatment of patients with CVD [1,4,6]. Association between elevated total cholesterol, low levels HDL-C, and high levels of triglyceride (TG) and atherogenic cardiovascular disease has been reported [7-11].

Inflammation has been recognized to play an important role in both the initiation and progression of coronary artery disease (CAD). High sensitivity C-reactive protein (hCRP) levels, which are one of the markers of systemic inflammation, are a powerful predictor of future myocardial infarction and cardiac death among apparently healthy individuals [1-3,11,12].

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The aim of present study was to investigate concentration of hCRP, lipids (TC, TG, LDL-C, nonHDL-C, HDL-C) lipid ratios (TC/HDL-C, LDL-C/HDL-C, TG/HDL-C) in patients with moderate dyslipidemia and compared to the healthy subjects, and relation between hCRP and lipids and lipid ratios.

MATERIAL AND METHODS

We studied 60 patients with moderately dyslipidemia (male and female) at the age between 21-63 and the reference group. The reference group constituted of 14 subjects chosen from normolipidemic healthy individuals. The study was conducted in accordance with the guidelines of the Ethics Committee, Medical University of Lublin.

Lipids, lipoproteins, routine laboratory parameters were obtained in serum after 14-hour overnight fasting. Blood was taken from veins into commercial tubes. Serum was immediately separated and stored in aliquots at –80°C. Lipids were determined on Hitachi 902 analyzer. Total cholesterol was determined by the enzymatic colorimetric method. Triglycerides (TG) were determined using standard enzymatic technique. HDL-cholesterol was measured by the enzymatic colorimetric method without precipitation. LDL-cholesterol (LDL-C) was calculated according to the Friedewald formula [5].

Non-HDL-cholesterol (non-HDL-C) was calculated as total cholesterol (TC) minus HDL-C. High-sensitivity C-reactive protein (hCRP) were determined with immunonephelometric methods, Simens Health care Diagnostic Product GmbH on a Dade Behring nephelometer BNII System

Statistical analysis was performed using of the Student-T test and the nonparametric Mann-Whitney U-test for comparisons in patients and the reference group. The relation between concentration of hCRP and lipids levels were examined by Spearman's correlation analysis. The data are expressed as mean ± standard deviation (SD) and median (min-max). Statistical analysis was performed using the STATISTICA program (StatSoft, Krakow, Poland).

RESULTS

Table 1 presents the results of lipids and hCRP concentration in the reference group and patients. The patients with moderate dyslipidemia had significantly increased concentration of TG, LDL-C and hCRP levels and significantly increased lipid ratios (TG/HDL, LDL-C/HDL-C).

The hCRP level was significantly positively correlated with TG, TC, LDL-C, nonHDL-C concentrations and TG/HDL-C, LDL-C/HDL-C ratios (Table 2).

Table 1. Lipid parameters and hCRP in the reference group and patients with moderate dyslipidemia

	Reference group n=14	Patients n=60	P
TG mg/dl	67 ± 22	113 ± 51	0.05
TC mg/dl	173 ± 17	198 ± 38	NS
LDL-C mg/dl	85 ± 18	116 ± 35	0.05
HDL-C mg/dl	62 ± 8	59 ± 10	NS
nHDL-C mg/dl	110 ± 20	139 ± 40	NS
TC/HDL-C	2.77 ± 0.4	3.55 ± 0.8	NS
LDL-C/HDL-C	1.77 ± 0.3	2.10 ± 0.75	0.05
TG/HDL-C	1.08 ± 0.4	2.05 ± 0.8	0.05
hCRP mg/L	0.03 (0.016-0.203)	0.29 (0.016-5.81)	0.001

Value expressed as mean ± SD and median (min-max)

Table 2. Correlaton hCRP and lipids and lipids ratios in patients with moderate dyslipidemia

	R	P
TG	0.266	0.05
TC	0.466	0.001
LDL-C	0.443	0.001
nHDL-C	0.437	0.001
TG/HDL-C	0.326	0.01
LDL-C/HDL-C	0.339	0.01

DISCUSSION

It is now accepted that inflammation plays a key role in the pathogenesis of all stages of atherosclerosis. Our results indicate that patients with moderate dyslipidemia had significantly increased concentrations of hCRP, TG, LDL-C and TG/HDL-C, LDL-C/HDL-C ratios. The concentration of hCRP was correlated with TG, TC, LDL-C and non-HDL-C levels and with TG/HDL-C, and LDL-C/HDL-C ratios. Association between hCRP and lipids indicated that slightly increased hCRP can increase, total cholesterol, TG, LDL-cholesterol and non-HDL cholesterol in patients with moderate dyslipidemia.

Studies suggest that hCRP predicting cardiovascular events. The measuring hCRP in the general adult population in the United States have found the median values of approximately 0.8mg/L. The recent standardization of the hCRP assay allows acceptable precision down to and below 0.3 mg/L. It is within these lower, previously normal ranges, that hCRP levels seem to have predictive abilities for coronary heart disease events [1,3]. High-sensitivity C-reactive protein may provide prognostic value in a wide variety of clinical scenarios, from asymptomatic subjects to patients hospitalized for acute coronary syndromes. Authors suggest that increased hCRP is associated with a significant risk of incident cardiovascular events after correcting for traditional risk factors [1,3,12]. Studies have also shown that the relative impact of elevated hCRP on the prediction of cardiovascular events is as large, individually, as that of LDL cholesterol, HDL cholesterol, blood pressure, and smoking (37,38). The median LDL-C at the initiation of the trial was 108mg/dl. A recently released analysis also demonstrated that patients achieving a low CRP and low LDL after 1 year of therapy had lower events rates than patients with high CRP and low LDL [5,6]. The high hCRP levels have been reported to be associated with an increased risk of further coronary events in patients with CAD [1,3,12].

Atherogenic dyslipidemia is low levels of high-density lipoprotein cholesterol (HDL-C) as well as elevated triglycerides (TG) concentrations, while LDL-C level may only be marginally elevated in this setting [6,7,8,10]. A single apolipoprotein B100 (apoB) molecule is present in all major atherogenic particles of liver origin (very-low and intermediate-density lipoproteins (VLDL and IDL), and LDL). Therefore, measurement of apoB provides direct information on the number of atherogenic particles (LDL and non-HDL), irrespective of their size. These atherogenic particles, collectively known as non-HDL lipoproteins, are associated with atherogenic dyslipidemia [7-11]. Ratios were introduced to increase epidemiological prediction of cholesterol, and include the ratio of total or LDL-C to HDL-C, and that of apoAI to apoB. It is now established that both apoB measurement and non-HDL-C are better predictor of CVD that LDL-C (even when the latter is directly measured instead of derived from Friedewald's formula), evidence which should eventually lead to some revision of the current risk paradigm [7-11].

Recently, it was shown, that triglyceride-rich lipoprotein (TRL) lypolysis products provide a pro-inflammatory stimulus that can alter endothelial barrier function. TRL lipolysis releases neutral and oxidized FFAs that induce

endothelial cell inflammation. Therefore, the oxidative metabolism of FFA in endothelial cells can produce inflammatory responses, TRL lipolysis can also release mediators of oxidative stress that may influence endothelial cell function in vivo by stimulating intracellular reactive oxygen species (ROS) production [7,13].

CONCLUSION

The results suggest that chronic inflammation disease can disturb lipids concentration and can accelerate atherosclerosis. However, these results require further studies.

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