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Serum CETP level in post-renal transplant patient (Tx)

Stężenie CETP w surowicy krwi pacjentów po transplantacji nerki

Hyperlipidemia is a common disorder after kidney transplantation and it is associated with increased morbidity and mortality in kidney transplant patients. It is known that kidney transplant patients have a very high risk for developing cardiovascular disease (CVD) and the risk for CVD even is even 50-60-fold higher than in general population. Those patients may die with the functioning graft thus CVDs are an important cause of graft loss, particularly after the first year posttransplantation [7, 15, 19]. Dyslipidemia occurs in at least 60% [13] of patients after renal transplantation. It is characterized by higher values of total cholesterol (TC), triglycerides (TG), low density cholesterol (LDL-C), very low density cholesterol (VLDL) and apolipoprotein B. Other changes include variable effects on HDL-C, apo(a) and the accumulation of atherogenic remnants and triglyceride-rich lipoproteins [10]. The reasons for those disturbances are not yet completely understood. Some reports suggest that this is mainly due to the use of a variety of different immunosuppressive therapies. The use of immunosupressants (cyclosporine A, tacrolimus) and steroids come with serious side effects, such as dyslipoproteinemia, hypertension, hyperglycemia and posttransplant diabetes mellitus and peptic ulcers [7, 10, 13–15, 19]. On the other hand, lipoprotein and lipid disorders are also caused by deregulation of lipoprotein transfer proteins and metabolic enzymes. Cholesterol ester transfer protein (CETP) is principally associated with HDL in plasma and plays a key role of a modulator not only of the intravascular metabolism of HDL and apoA-I but also triglyceride (TG)-rich proteins and low-density lipoprotein, mediates the transfer of cholesteryl esters from HDL to proatherogenic apoB-lipoproteins, with heterotransfer of TG mainly from VLDL to HDL [2, 3, 6, 21]. Several polymorphisms have been described at the CETP locus which seems to be an important genetic factor determining plasma lipid levels. CETP activity is elevated in the dyslipidemias of metabolic disease involving insulin resistance and moderate to marked hypertriglicerydemia and is intimately associated with premature atherosclerosis and high cardiovascular risk [2,3].

Lipid management in kidney transplant patients is important to improve the outcome after successful transplantation and to minimize the occurrence of CVD. Although hyperlipidemia could be treated by modifying immunosuppressant protocol, the use of statins and proper low-fat diet, elevated lipid concentrations still remains a concern. Evidence exists that the consequence of CETP activity may depend on metabolic setting, in particularly on triglyceride levels [11, 12, 19].

The aim of this study was to investigate serum lipid and lipoprotein profiles and lipid and lipoprotein ratios, CETP levels and BMI index in post-renal transplant patients with TG>150mg/dl and TG<150mg/dl.

MATERIAL AND METHODS

The studies were performed in 42 the post-renal transplant patients (Tx) (women, n=20 and men, n=22) at the age between 21–60. Tx patients were without diabetes, liver disease, active inflammatory disease, malignancy, obesity, glucose intolerance but 25 patients had hypertension. The post-renal transplant patients received cyclosporine A + prednisone (n=28), tacrolimus + prednisone (n=11) and sirolimus + prednisone (n=3). Hypertensive patients with hypertriglyceridemia treated with athorvastatine or simvastatin used anti-hypertensive medications. Forty five healthy patients were the reference group (22 women and 23 men, aged 22 to 60 years). The healthy subjects were chosen from among apparently normolipidemic individuals who were symptom free and had no evidence of previous cardiac, hypertensive or renal disease. They were without diabetes, liver disease, active inflammatory disease, malignancy, obesity or glucose intolerance. Tx patients were divided into 2 groups: triglyceride (TG)>150mg/dl and TG<150 mg/dl using TG concentration of 150mg/dl as a cut-point. Recommendation for diabetic patients and with renal dyslipidemia suggests that plasma triglycerides should be reduced to a level of 150 mg/dl (<1.7 mmol/l) [9, 10], so for that reason the TG cut-point was 150mg/dl. Venous blood was drawn after a 14-hour overnight fasting, and plasma was obtained by centrifugation at 3000 rpm at 4^{o} C immediately after blood collection. Samples were either used for measurements immediately or stored frozen at -80^{o}C. Routine laboratory parameters (the level of urea, uric acid, creatinine, total protein, albumin) were determined using Au 400 analyser (Olimpus), and hemoglobin using ADVIA analyser, Bayer. Lipids and lipoproteins were determined on Hitachi 902 analyser. The total cholesterol (TC) was estimated by the enzymatic-colorimetric method, BIOMAXIMA, HDL-C by the direct method with immunoinhibition (AB-WAKO – BIOMAXIMA). HDL cholesterol, which is not bound with enzymes (cholesterol esterase and cholesterol oxidase) and chromogens producing a coloured complex were determined. Triglycerides (TG) were determined using the standard enzymatic technique (BIOMAXIMA). LDL-cholesterol (LDL-C) was calculated according to the Friedewald formula [8]. Non-HDL-C was calculated as total cholesterol minus HDL-C. Apo AI, apoB were measured by Roche kit using the turbidimetric methods. The Wako CETP test is an in vitro assay for the quantitative determination of cholesteryl ester transfer protein (CETP) concentration in human serum. A sample is treated with the pretreatment solution, which contains detergent. CETP in the sample is released from lipoproteins. The released CETP reacts with the monoclonal antibody (CETP-4) coated on the solid phase, and with the HRP-labelled monoclonal antibody (CM5, a-27) in a double-step reaction. The activity of the enzyme bound to the sample is determined by using hydrogen peroxide and the chromogen (*o*-phenylenediamine). The CETP concentrations of specimens are obtained in µg/mL by using the calibration curve.

S t a t i s t i c a l a n a l y s i s was performed using one-way analysis of the ANOVA variance and multiple comparisons for assessment of the mean ± standard deviation (SD) in post-renal transplant patients and compared to the reference group. The data were expressed as means standard deviation and values of $p<0.05$ were considered significant. Statistical analysis was performed using the STATISTICA program (StatSoft, Krakow, Poland).

RESULTS

The selected clinical and routine laboratory parameters are presented in Table 1. Tx patients with TG>150mg/dl had worse clinical and laboratory parameters than Tx patients with TG<150mg/dl and both groups were worse in comparison to the reference group. According to the results, we divided the examined post-transplant patients into two groups. The criterion of the division was the concentration

of TG. Tx patients were divided into two groups with TG>150mg/dl and TG<150 mg/dl using TG concentration of 150mg/dl as a cut-point. Table 2 presents the lipid and lipoprotein parameters and CETP concentration in both groups Tx patients and the reference group. Tx patients with TG>150 mg/dl had: TG, TC, nonHDL-C, and lipid ratios (TC/HDL-C, TG/HDL-C) significantly higher but lipoprotein ratios (apoAI/apoB, HDL-C/apoAI) were significantly lower in comparison to patients with TG<150mg/dl. However, lipids (TG, TC, LDL-C, nonHDL-C, HDL-C) and lipid ratios (TC/HDL-C, LDL-C/HDL-C, TG/HDL-C) and lipoproteins (apoAI, apoB) and lipoprotein ratios (apoAI/apoB, HDL-C/apoAI) in both groups were significantly disturbed as compared to healthy subjects. Moreover, both studied groups Tx patients had no change in CETP levels and were non-statistically different as compared to the reference group.

Table 1. Clinical and routine laboratory parameters in post-renal transplant patients (Tx) with TG>150 mg/dl and TG<150mg/dl and reference group

	TG>150mg/dl Tx patients n=30	TG<150mg/dl Tx patients n=12	Reference group n=45
Age years	45.60 ± 11.04	43.84 ± 11.20	45.89 ± 14.50
BMI kg/m^2	26.07 ± 4.50	24.01 ± 3.51	23.01 ± 1.91
Urea mg/dl	55.88 ± 26.11*	48.30 ± 20.11*	18.80 ± 12.60
Creatinine mg/dl	1.72 ± 0.7*	1.53 ± 0.74*	0.80 ± 0.2
Total protein g/dl	7.13 ± 0.58	6.94 ± 0.55	7.25 ± 0.30
Albumin g/dl	4.20 ± 0.27	4.22 ± 0.39	4.5 ± 0.40
Hemoglobin mg/dl	13.70 ± 1.72	14.10 ± 1,50	14.70 ± 1.60
GFR	58.71 ± 17.48	65.80 ± 19.70	117.50 ± 12.30

P<0.05* – vs. reference group

Table 2. Lipids, lipoproteins and lipid and lipoprotein ratios in post-renal transplant patients (Tx) with TG>150 mg/dl and TG<150mg/dl and reference group

	TG>150mg/dl Tx patients n=30	TG<150mg/dl Tx patients n=12	Reference group n=45
TG mg/dl	207.55 ± 41.51*	127.11 ± 26.05*†	89.68 ± 25.71
TC mg/dl	203.81 ± 27.21*	182.20 ± 20.30†	179.50 ± 27.03
LDL-C g/dl	122.47 ± 23.42*	115.05 ± 20.18	97. 92 ± 22.42
HDL-C mg/dl	39.47 ± 8.65*	41.45 ± 11.7*	57.81 ± 8.31
nonHDL-C mg/dl	164.57 ± 30.29*	140.84 ± 20.15*†	113.89 ± 23.89
CETP µg/ml	1.495 ± 0.56	1.504 ± 0.364	1.657 ± 0.370
ApoAI mg/dl	149.15 ± 18.97*	150.57 ± 20.23	163.1 ± 10.55
ApoB mg/dl	92.09 ± 17.63*	84.57 ± 15.19	72.90 ± 15.63
ApoAI/apoB	1.60 ± 0.37*	1.89 ± 0.40*†	2.24 ± 0.38
HDL-C/apoAI	0.24 ± 0.04*	0.27 ± 0.04*†	0.35 ± 0.04
TC/HDL-C	5.33 ± 0.98*	4.68 ± 1.04*†	2.96 ± 0.56
LDL-C/HDL-C	3.24 ± 0.93*	2.87 ± 0.90*	1.64 ± 0.47
TG/HDL-C	5.34 ± 2.01*	3.53 ± 1.31*†	1.43 ± 0.50

P<0.05* – vs. reference group; † – vs. TG>150mg/dl

DISCUSSION

Dyslipidemia is an important complication in renal transplant patients. Increased serum lipid levels are recognized as a potential risk for cardiovascular disease in transplant patients. Many studies have shown that dyslipidemia is a major factor in cardiovascular and hypertensive diseases [7,13,19]. Cholesterol ester transfer protein (CETP) plays a key role in lipoprotein metabolism, transferring cholesteryl esters from HDL to other lipoproteins. The reason for increased lipid levels after transplantation is not yet completely understood. Some reports suggest that prescription of cyclosporine, beta blockers, or steroids as well as other factors, such as gender, age, and kidney dysfunction, may be potential causes [11, 21]. Our study showed that Tx patients who were treated with prednisone, cyclosporine A, tacrolimus and statins had lipid and lipoprotein abnormalities including increased TG, TC, LDL-C, nonHDL-C, apoB and lipid ratios, and decreased HDL-C, apoAI and lipoprotein ratios. A variety of immunosuppressive therapies in our Tx patients seem to be one of the main factors that influence posttransplant lipidemic profiles. However, these patients had no change in CETP levels as compared with TG>150mg/dl and TG<150mg/dl and reference group. Recently, Pahl et al. [16] in their study revealed a marked reduction in plasma ApoAI and LCAT but no change in either PLTP or CETP concentration or activity in stable haemodialysis-dependent ESRD patients. These findings confirm the role of ApoAI and LCAT deficiencies and exclude the significant participation of PLTP and CETP in the pathogenesis of dyslipidaemia in this population.

CETP gene polymorphism has a relationship to lipid profile. Zeybek et al. [21] showed that patients carrying CETP Taq1BB1 B1 allele and B1B1 genotype were associated with serum lipid profiles in renal transplant patients but they do not suggest that these genotypes are associated with increased in serum lipid profiles. CETP expression in Tg mice delays plasma clearance and liver uptake of TG-rich lipoproteins by two mechanisms: (i) transferring TG to HDLs and increasing CE content of the remnant particles and (ii) by diminishing LPL expression. These findings show that the level of CETP expression can influence the responsiveness to dietary fat and may lead to fat intolerance. [17]. Three CETP genotypes that are associated with moderate inhibition of CETP activity (and, therefore, modestly higher HDL-C levels) show weakly inverse associations with a coronary risk. The combined per-allele odds ratios (Ors) for coronary disease were compatible with the expected reductions in the risk for equivalent increases in HDL-C concentration in available prospective studies [18]. A major effect of the CETP A373P polymorphism on HDL-C, LpAI, and Lp -I: A-II and a minor effect of the APOE2 polymorphism on apoAI and LpAI were found. These effects should contribute to the risk of cardiovascular disease. This should be further evaluated in prospective studies [6].

Although the reduction in plasma neutral lipid transfer activity, as a result of either CETP gene mutation or CETP inhibition, is known to produce a major yet unrivaled increase in HDL-C, its impact on atherosclerosis prevention still remains a matter of debate. In patients with acute myocardial infarction (MI), high cholesteryl esters transfer (CET) rates are characterized by the presence of the high CETP mass/high non-HDL-C/low HDL2b triad. The association of high CET rates with young age at first MI lends support to a significant contribution of CETP to the accelerated progression of disease among asymptomatic patients. Indeed, CETP has been found to increase the risk for future CHD, but only individuals with an accumulation of triglyceride-rich $VLDL_1$ acceptors [1,20]. In contrast, when clearance of VLDL and LDL is optimal, CETP might no longer act as a proatherogenic factor, and may provide an alternative pathway for HDL cholesteryl esters to be removed from the booldstream. When apoB-containing lipoprotein clearance was accelerated by treatment with statins, the B1B1 Taq1B genotype with high CETP levels was not accompanied by an

elevation in CHD risk [1,20]. Although CETP concentration is a major determinant of CETP activity *in vivo*, its activity is also dependent on lipoproteins. In an attempt to assess the combined effects of CETP concentration and the level and composition of lipoproteins in a comprehensive way, the rate of CET (cholesteryl ester transfer) from a tracer dose of HDL toward endogenous lipoprotein particles was measured in total serum. MI patients were heterogeneous for both CETP activity and lipoprotein profiles. Compared with the lower tertiles, the high CET rate group combined elevated CETP and high triglyceride levels. The CET is assumed to closely play the role of triglyceride-rich lipoprotein and CETP in driving cholesterol esters out of HDL, resulting in shrinkage of the particles [1,4,20]. The study with APOE*3-Leiden mice showed that the proatherogenic role of CETP is mainly the consequence of its VLDL-C increasing effect, suggesting that CETP inhibition could lead to a reduction in atherosclerosis development mainly by lowering VLVL-C and LDL-C levels. As a consequence, CETP inhibition may not be a good alternative or additive for current LDL-C lowering therapies, e.g. with statins, CETP inhibition may even interfere with the beneficial effect of CETP on stabilization of atherosclerotic lesions by increasing collagen [5]. Thus, favorable effects of CETP inhibition on plasma VLDL-C and LDL-C and HDL-C levels, may not outweigh the possible detrimental effects on plaque stability. Therefore, the strategy CETP inhibition in the general dyslipidemic population should be pursued with care. The increase in VLDL-C is the most important factor contributing to the proatherogenic effect of CETP in E3L mice. Furthermore, CETP may positively influence atherosclerotic lesion stability by enhancing the collagen content of atherosclerotic lesions [5]. Taken together, our findings suggest that dyslipidemia, dyslipoproteinemia in Tx patients may be an explanation for the treatment of prednisone, cyclosporine, tacrolimus and statins but not the concentration of CETP mass [19].

CONCLUSIONS

It is cocluded that Tx patients with hipertriglyceridemia received statins and immunosuppressive therapy tend to exclude dysregulation of CETP in the pathogenesis of dyslipidemia but future studies are required.

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SUMMARY

Cholesterol ester transfer protein (CETP) is principally associated with HDL in plasma and it plays the role of a key modulator not only of the intravascular metabolism of HDL and apoA-I but also triglyceride(TG)-rich lipoproteins and low-density lipoprotein, it mediates the transfer of cholesteryl esters from HDL to proatherogenic apoB-lipoproteins, with heterotransfer of TG mainly from VLDL to HDL. The studies were performed in 42 post-renal transplant patients (Tx) (women, n=20 and men, n=22) at the age between 21–60 years. The post-renal transplant patients received cyclosporine A + prednisone (n=28), tacrolimus + prednisone (n=11) and sirolimus + prednisone (n=3). Hypertensive patients with hypercholesterolemia and hypertriglyceridemia treated with fibrate and athorvastatin or simvastatin used anti-hypertensive medications. Forty five healthy patients were the reference group (22 women and 23 men, aged 22 to 60 years). Tx patients were divided into 2 groups: triglyceride (TG)>150mg/dl and TG<150 mg/dl using TG concentration of 150mg/dl as a cut-point. The aim of this study was to investigate serum lipid and lipoprotein profiles and lipid and lipoprotein ratios, CETP levels and BMI index in post-renal transplant patients with TG>150mg/dl and TG<150mg/dl. Tx patients with TG>150 mg/dl had TG, TC, nonHDL-C, and lipid ratios (TC/HDL-C, TG/HDL-C) significantly higher but lipoprotein ratios (apoAI/apoB, HDL-C/apoAI) were significantly lower as a comparison to patients with TG<150mg/dl. However, lipids (TG, TC, LDL-C, nonHDL-C, HDL-C) and lipid ratios (TC/HDL-C, LDL-C/HDL-C, TG/HDL-C) and lipoproteins (apoAI, apoB) and lipoprotein ratios (apoAI/apoB, HDL-C/apoAI) in both groups were significantly disturbed as compared to healthy subjects. Moreover, both studied groups of Tx patients had no change in CETP levels and were non-statistically different as compared to the reference group. We conclude that Tx patients with hypertriglyceridemia received statins and immunosuppressive therapy tend to exclude dysregulation of CETP in the pathogenesis of dyslipidemia but future studies are required.

STRESZCZENIE

Białko transportujące estry cholesterolu połączone z cząstką HDL jest modulatorem nie tylko wewnątrznaczyniowego metabolizmu HDL i apoAI, lecz także lipoprotein bogatych w TG i LDL, pośredniczy w transferze estrów cholesterolu z HDL do promiażdżycowych apoB-lipoprotein, i w odwrotnym kierunku – TG głównie z VLDL do HDL. Badania były wykonane u 42 pacjentów po transplantacji nerki (Tx) (kobiety, n=20 i mężczyźni n=22) w wieku 21–60 lat. Badani pacjenci otrzymywali cyklosporynę A + prednison (n=28), tacrolimus + prednison (n=11) i sirolimus + prednison (n=3). Pacjenci z nadciśnieniem, hipercholesterolemią i hipertriglyceridemią leczeni byli fibratami i atorwastatyną albo simwastatyną oraz lekami obniżającymi ciśnienie krwi. Czterdzieści pięć zdrowych osób stanowiło grupę referencyjną (22 kobiety i 23 mężczyzn) w wieku 22–60 lat. Pacjenci po transplantacji nerki byli podzieleni na dwie grupy: TG>150mg/dl i TG<150mg/dl, gdzie stężenie TG 150 mg/dl było kryterium podziału. Celem badań było oznaczenie profilu lipidów, lipoprotein, wskaźników lipidowych i lipoproteinowych, stężenia CETP i BMI u pacjentów po transplantacji nerki z TG>150mg/dl i TG<150mg/dl. Tx pacjenci z TG>150mg/dl mieli istotnie podwyższone stężenia TG, TC, nonHDL-C i wartości wskaźników lipidowych (TC/HDL-C, TG/HDL-C), natomiast znamiennie obniżone wartości wskaźników lipoproteinowych (apoAI/apoB, HDL-C/apoAI) w porównaniu z grupą pacjentów z TG<150mg/dl. Jednakże oznaczane stężenia lipidów (TG, TC, LDL-C nonHDL-C, HDL-C) i wartości wskaźników lipidowych (TC/HDL-C, LDL-C/HDL-C, TG/HDL-C) oraz stężenia lipoprotein (apoAI, apoB) i wskaźników lipoproteinowych

(apoAI/apoB, HDL-C/apoAI) w obu grupach były istotnie zaburzone w porównaniu ze zdrowymi osobami. Natomiast stężenia CETP w obu badanych grupach były podobne i nie różniły się istotnie statystycznie w stosunku do grupy referencyjnej. Na podstawie badań stwierdzono, że pacjenci po transplantacji nerki z hipertriglyceridemią leczeni statynami i lekami immunosupresyjnymi wykazują tendencje wykluczające udział CETP w patogenezie dyslipidemii, jednak dalsze badania powinny być prowadzone.