LIPOPROTEIN METABOLISM ABNORMALITIES IN PATIENTS WITH CHRONIC RENAL INSUFFICIENCY

Mirjana Jugović[1], Vesna Spasojević-Kalimanovska[2], Slavica Spasić[2], Zorica Nikolić[1]

[1]Clinical Chemistry Laboratory, Dr. L. Lazarević, Health Centre, Šabac
[2]Institute of Medical Biochemistry, University School of Pharmacy, Belgrade

Summary: Patients with chronic renal insufficiency on hemodialysis develop lipoprotein abnormalities that may contribute to increased risk for atherosclerosis. The atherogenic risk for chronic renal insufficiency patients and dialysis treated patients was assessed by measuring total cholesterol, triglycerides, HDL-cholesterol, LDL-cholesterol and calculating the ratio: TC/HDL-C and LDL-C/HDL-C. The examined group consisted of 18 cronic renal insufficiency patients and 60 patients on hemodialysis. The results were compared to a control group of 85 voluntary blood donors. Serum lipid parameters were examined by standard methods. All lipid parameters in hemodialysis patients were statistically different compared to the control group (p<0.05) while chronic renal insufficiency patients showed significant difference only in triglycerides and HDL-cholesterol. Hypertriglyceridemia was present in both examined groups of patients and HDL-cholesterol was lower within both groups. All calculated atherogenic ratios were higher for patients than the control group. Lipid parameters were compared between chronic renal insufficiency and hemodialysis patients but statistical significantly difference was obtained only for HDL-cholesterol (p<0.05). We conclude that increased values of triglycerides and lower HDL-cholesterol in chronic renal insufficiency patients contribute to high incidence of cardiovascular disease. Chronic renal insufficiency patients have impaired reverse cholesterol transport from peripheral cells to lipoproteins, decreased levels of HDL-cholesterol, hypertriglyceridemia prevalence of small, dense LDL and increased levels of potentially atherogenic remnant particles.

Key words: dyslipidemia; hypertriglyceridemia; atherogenic risk, chronic renal insufficiency; hemodialysis.

Introduction

Atherosclerosis and cardiovascular disturbance are common among patients with progressive renal insufficiency and in uremic patients receiving long-term hemodialysis (1, 2). Cardiovascular disease is the most important cause of mortality in end-stage renal disease (3). Early reports suggested that atherosclerosis is accelerated during maintenance hemodialysis for end-stage renal disease. The mechanism for developing atherosclerosis in chronic renal insufficiency and hemodialysis is multifactorial. However, plasma lipid disturbance have been identified as significant risk fac-

tors for cardiovascular disease in this patients. Previous studies have revealed that progressive renal failure is accompanied by abnormalities of lipoprotein transport and that renal insufficiency is predominantly reflected in altered concentrations and composition of individual lipoproteins (4). The main lipid abnormality is an increase in plasma triglyceride and a decrease in HDL-cholesterol concentrations, with smaller change in the levels of cholesterol rich lipoproteins. The principal features of the impaired lipoprotein metabolism include the increase in very-low density lipoprotein (VLDL) and intermediate-density lipoprotein (IDL) fractions, which are mainly due to a defect in the catabo-

Address for correspondence

Vesna Spasojević-Kalimanovska
Institute of Medical Biochemistry
University School of Pharmacy
Vojvode Stepe 450, POB 146,
11000 Belgrade, Yugoslavia
E-mail: vkalima@ pharmacy.bg.ac.yui

lism of triglyceride rich lipoproteins and the decrease in the high-density lipoproten fraction (5, 6). Elevated triglyceride levels can indicate accumulation of triglyceride-rich, potentially atherogenic remnant particles and this finding has been reported in patients treated with chronic hemodialysis. LDL-cholesterol levels are not elevated in end-stage renal disease but changes in size and composition in association with hypertriglyceridemia could potentially result in increased atherogenicity of LDL particles (7). Recent epidemiological studies suggest, however that a predominance of smaller and dense LDL (small, dense LDL) particles is highly atherogenic, and it may be a new risk factor of coronary heart disease. Previous results demonstrate that dyslipidemia of dialysis-treated subjects includes a shift in size distribution of LDL particles toward the potentially atherogenic small LDL particles (8). Small, dense LDL is thought to be more atherogenic due to characteristics which include a lower affinity to LDL receptor, an increased susceptibility to oxidation and an enchanced ability to penetrate to vascular intima and bind to intimal proteoglycans.

The mechanisms behind the dyslipoproteinemia of uremia are not fully elucidated. The lipoprotein profile itself, including low HDL-C concentrations and accumulation of remnant particles, indicate slow catabolism of triglyceride-rich lipoproteins. Impaired removal of triglyceride-rich lipoproteins is accompanied with disturbances probably occurring at multiple sites along the process. Hypertriglyceridemia and increased lipoprotein remnants in renal insufficiency have been attributed to impaired lipoprotein lipase (LPL) and hepatic triglyceride lipase (HTGL) activity. Lipolysis is the rate limiting step in the catabolism of triglyceride rich lipoproteins. The rate of lipolytic reaction depends on enzyme activity and lipoprotein substrate characteristics. The rate of receptor-dependent lipoprotein uptake is determined by receptor function and the affinity of the lipoproteins for the receptor. Each of these feature can be influenced in chronic renal insufficiency (9).

Several investigators indicate decreased lipoprotein lipase activity in renal failure (10 12). Regarding the mechanism behind the decrease in LPL activity in uremia, three theories prevail, all of them supported by clinical or experimental findings: (1) reduced enzyme synthesis by the parenchyma cell; (2) circulating inhibitor(s) against the enzyme activity and (3) heparin-induced depletion of the functional enzyme pool (in hemodialysis patients).

Lipolysis of triglyceride rich lipoproteins, especially VLDL depends on the aberrant substrate characteristics of these lipoproteins. Metabolism of a lipoprotein particle is generally determined by the apoprotein composition so the disturbed apolipoprotein composition of VLDL in renal insufficiency has to be considered. There is a decrease in the apoCII/apoCIII ratio of VLDL and it is possible that it influences the substrate

quality and the enzyme lipolytic reaction. Apolipoprotein CII and CIII are regulators of the LPL reaction, apo CII being the activator and apo CIII a potential inhibitor (13). In uremia, a number of potential or proven biological modifications might disturb VLDL substrate characteristics. This includes sialinization, carbamylation and glycosylation and some unidentified uremic toxins which might induce structural and functional changes in VLDL (14, 15).

Receptor-mediated uptake of lipoproteins is also disturbed and there is evidence of malfunction of LDL (B-100/E) receptor in CRI patients and a significantly reduced catabolic rate of LDL particle. In addition to a disturbance in LDL-receptor function, there are impairments in the receptor ligand characteristics of uremic LDL. Indeed, carbamylation and glycosylation as well as triglyceride enrichment are known to interfere with the receptor-mediated uptake of LDL. Reduced receptor-mediated uptake of LDL, for whatever reason, explains the usually reported normal LDL cholesterol concentration in spite of decreased production via the lipolytic pathway (16).

Catabolic defect of triglyceride rich lipoproteins (VLDL) in renal chronic renal insufficiency and accumulation of remnant particles is primary attributed to the reduced LPL activity but also to the reduced HL activity. The effect of hepatic lipase activity on the metabolism of remnant particles is controversial and it is more important in the metabolism of HDL particles. Lipoprotein lipase and hepatic lipase deficiencies are causally linked to parathormone excess.

Hemodialysis patients have impaired reverse cholesterol transport from peripheral cells to lipoproteins and there is a progressive decline of apo A containing lipoproteins with reduced HDL-C levels. The reduction in plasma HDL-C concentration in CRI patients is associated with a parallel decline in plasma concentrations of its principal apoprotein constituents, namely apo AI and apo AII.

Metabolism of HDL particle is disturbed in several ways. First, an abnormal composition of the HDL particle with predominantly lower apo AI concentrations is the key point of the reverse cholesterol transport. In fact, apo AI and HDL synthetic rates are thought to be reduced in CRI patients. There are experimental results that support this hypothesis since there are uremic »middle moleculesá resulting in a significant decrease in synthesis and secretions of apo AI. There is also evidence of defects in cholesterol esterification due to the reduced LCAT activity in uremia (17, 18).

The lipoprotein abnormalities are present in many patients already at the early stages of renal function impairment to become more accentuated in advancing renal failure (19, 20). From experimental observations and findings of lipid depositions in diseased kidneys it has been suggested that circulating lipopro-

teins may be involved in the development of glomerulosclerosis and tubulointestitial lesions that lead to progressive loss of renal function. After a primary insult the demaged glomerular barrier to the mesangium could allow an influx of macromolecules, such as lipoproteins. Through a series of events analogous to the development of atherosclerosis proliferate and sclerotic processes are triggered, ultimately leading to a partial or complete obliteration of the glomerular capillaries. The glomerular lesions may also lead to a filtration of lipoproteins that may be taken up and induce inflammatory and sclerotic processes in the tubulointestitial tissue (21 23).

The aim of our study was to analyse disturbance in lipoprotein metabolism and the increased risk for atherosclerosis in CRI patients and those with advanced renal failure being on hemodialysis. The atherogenic risk for patients on hemodialysis was assessed by determing: total cholesterol (TC), triglyceride (TG), HDL-C and LDL-C. Also we calculated the atherogenic risk factors that may predict the risk for cardiovascular disease because lipid abnormalities in renal disease are associated with both a progressive decline in renal function and cardiovascular complications.

Materials and Methods

Plasma lipid status was examined in three groups: CRI patients, patients on hemodialysis and a control group. The first examined group consisted of 18 CRI patients (6 females and 12 males). The second one comprised 60 patients (28 females and 32 males) on hemodialysis. Patients were dialysed two to three times weekly for 5 6 hours using bicarbonate (BD) or acetate-based (AD) fluid on cellulose-acetate dialysis membrane. Hemodialysis patients had received replacement therapy from one to ten years. To examine the influence of the type of dialysis on the lipid disturbance we examined 27 patients on acetate and 33 on bicarbonate hemodialysis. Clotting of the extracorporal circuit was avoided by using of an anticoagulant such as low-molecular-weight heparin. The patients received standard heparin (1/3 as loading dose and 2/3 as continuous does) but no other anticoagulant or antiaggregation drugs.

CRI patients had the following ethiology: endemic nephropathy (n=7), glomerulonephritis (n=3), systemic lupus (n=1), chronic pielonephritis (n=2) and 5 were with unknown ethyology. The diagnosis of chronic renal insufficiency is based on the elevated value of creatinine (>178 mmol/L). Patients on hemodialysis were with this ethyology: endemic nephropathy (n=35), glomerulonephritis (n=6), systemic lupus (n=2), reflux nephropatia (n=4), bilateral nephroctomia (n=1) and 12 were with unknown ethyology. Patients were dialysed when symptoms of uremia were detected or the value of serum urea was >35.6 mmol/L or creatinine >763 μmol/L. Patients in predialysis

and dialysis group were consuming protein-restricted diet, which is rich in carbohydrates and fat (commonly prescribed to delay the onset of uremia), none were treated with lipid-lowering drugs or hormone replacement therapy. Individuals with diabetes mellitus were excluded. The obtained results were compared with a control group of 85 (38 females and 47 males) voluntary blood donors. These individuals were matched for age and gender with chronic renal insufficiency and dialysis group.

Blood samples were drawn from all patients after overnight fasting. In the subjects on hemodialysis, blood was collected prior to dialysis and heparinization. Serum lipid parameters were examined by standard biochemical methods. TC and TG concentrations were determined by standard, automated enzymatic assays using commercially available regents (CHOD-PAP, and glycerol-3-phosphate oxidase, Randox) on Technicon RA-X 1000. The interassay CV for TC was 1.6% and 0.9% at 3.2 mmol/L and 7.8 mmol/L, respectively. For TG the CV was 1.9% at 1 mmol/L and 1.8% at 2.2 mmol/L. HDL-C was analysed after precipitation of non-HDL lipoproteins and subsequent determination as cholesterol in the supernatant. The interassay CV for HDL-C was 5.5 % at 2.05 mmol/L and 4% at 0.98 mmol/L. LDL-C was calculated according to Friedewald formula. Atherogenic risk factors were calculated as TC/HDL-C and LDL-C/HDL-C ratio.

Statistical evaluation was performed using nonparametric Mann-Whitney's U-test for unpaired data. The Spearman-Person test was used to evaluate the correlation between lipid parameters in the examined groups. Data were expressed as mean SD.

Results

Blood lipid parameters in the two patients groups compared to the control are given in *Table I*. In the predialysis group, patients with CRI, lipid disturbance is characterised by elevated TG and decreased TC, LDL-C and HDL-C concentrations. When compared to the controls, there were significant differences in the mean serum TG concentrations (2.41 1.09 vs.1.40 0.80 mmol/L, p<0.001). This group of patients had significantly lower mean HDL-C concentrations compared to controls (1.07 0.24 vs.1.37 0.28 mmol/L, p<0.001). Decrease in TC and LDL-C is not significant compared to the control group (p>0.05). Risk factors were higher for patients compared to controls and there is statistically significant difference only for TC/HDL-C ratio (p<0.05).

All lipid parameters in hemodialysis patients were statistically different compared to the control group. In hemodialysis patients lipid disturbance is primary characterised by hypertriglyceridemia and elevated serum TG were found in more than a half of dialysed patients. Mean values for lipid parameters in this group

Table I   Plasma lipid concentrations in CRI patients, dialysis treated patients and healthy controls

	CRI patients	Hemodialysis patients	Healthy controls
Number	18	60	85
Age	49.28 11.26	54.48 11.84	
Sex, male/female	12/6	33/27	47/38
TC, mmol/L	5.25 1.52	4.84 1.23**	5.61 1.50
TG, mmol/L	2.41 1.09***	2.11 1.19***	1.40 0.80
HDL-C, mmol/L	1.07 0.24***	0.94 0.37***	1.37 0.28
LDL-C, mmol/L	3.08 1.21	3.00 1.04*	3.60 1.35
TC/HDL-C	5.13 2.1*	5.78 2.30***	4.27 1.49
LDL-C/HDL-C	3.04 1.66	3.51 1.83**	2.76 1.24

* $P < 0.05$ vs. healthy control; **$P < 0.01$ vs. healthy control; ***$P < 0.001$ vs. healthy control

Table II   Comparison of laboratory data between two groups of CRI patients: on bicarbonate and acetate hemodialysis

	Acetate dialysis	Bicarbonate dialysis	P
N	27	33	
Age	53.37 12.64	55.39 11.25	
Sex, male/female	21/6	12/21	
TC, mmol/L	4.70 1.39**	4.94 1.10*	0.22
TG, mmol/L	1.90 1.08*	2.27 1.26***	0.14
HDL-C, mmol/L	1.00 0.39***	0.90 0.34***	0.22
LDL-C, mmol/L	2.91 1.28*	3.08 0.79	0.13
TC/HDL-C	5.35 2.43*	6.12 2.17***	0.16
LDL-C/HDL-C	3.34 1.80	3.64 1.87***	0.46

* $P < 0.05$ vs. healthy controls; **$P < 0.01$ vs. healthy controls; ***$P < 0.001$ vs. healthy controls.

Table III   Correlation coefficients between lipid parameters in the examined group and the statistical significance

	CRI patients	Hemodialysis patients	Healthy controls
TC vs. HDL-C	0.14	0.03	0.08
TC vs. LDL-C	0.90*	0.92*	0.96*
TC vs. TG	0.69*	0.52*	0.62*
HDL-C vs. LDL-C	0.02	0.15	0.19
HDL-C vs. TG	0.19	0.48*	0.42
LDL-C vs. TG	0.49*	0.47*	0.52*

* $P < 0.05$

indicate a decrease in TC and LDL-C concentrations and compared to healthy controls these decrese is statistically significant (p<0.05). When compared to the matched control group, hemodialysis patients had significantly reduced mean HDL-C (0.94 0.37 vs. 1.37 0.28 mmol/L, p<0.001) and significantly higher TG concentrations (2.11 1.19 vs.1.40 0.80 mmol/L, p<0.001). All calculated ratios were higher for patients than the control group and the increase for both risk factors, were statistically significant (p<0.01).

In comparison to patients with CRI, patients on long-term hemodialysis had a more severe lipid abnormalities with significantly lower HDL-C level (0.94 0.37 vs.1.07 0.24 mmol/L, p<0.05). All other mean values of lipid parameters and risk ratios were lower in the dialysis group but these differences were not significant.

Influence of acetate dialysis on acid-base balance and metabolic pathways are well known, so we also tested the hypothesis of a different influence of the dialysis fluid on lipid metabolism. Patients on dialysis

treatment were separated in two groups: acetate dialysis (N=27) and bicarbonate dialysis (N=33). Subjects on both types of hemodialysis also had higher TG and lower HDL-C than the control group *(Table II)*. The obtained lipid parameters were also compared between patients on acetate dialysis and those on bicarbonate dialysis but there was no statistical difference. Patients on bicarbonate dialysis had higher mean values of TG compared to acetate dialysis (2.27   1.26 vs. 1.90   1.08 mmol/L) and lower HDL-C (0.90   0.34 vs. 1.00   0.39 mmol/L) but there was no statistically significant difference (p = 0.14 and p = 0.22).

In *Table III* results of Spearman-Pearson correlation method for all lipid parameters are presented. It should be noted that TG are positively correlated to TC and LDL-C with a statistically significant coeficient of correlation (p<0.05) but not to HDL-C. A negative correlation was found between HDL-C and most of the other lipid parameters. TG were found to be negatively correlated to HDL-C for all examined groups, but only dialysis treated patients had a statistically significant correlation (r=   0.48, p<0.05).

Discussion

Patients with chronic renal insufficiency and those on chronic hemodialysis treatment are at elevated atherogenic risk and dyslipidemia appears to be one of the major risk factors. Our study indicates abnormal lipoprotein profile in CRF patients and patients on hemodialysis. Serum triglycerides are elevated in both groups whereas cholesterol (total and LDL) levels are whithin the values expected for healthy subjects. Obtained results demonstrate an increase of TG concentrations that is statistically significant compared to the control group and these results are in agreement with other authors (2, 4, 5). Mean values of serum TG are higher in CRI patients compared to the values for dialysed patients but not statistically significant, although other authors (19) indicated higher values within dialysed patients (1.5   0.7 vs 2.0   1.0 mmol/L, p<0.01). Treatment of renal insufficiency with a protein-restricted diet, which is rich in carbohydrates and fat, can exacerbate the hypertriglyceridemia, so these could be a reason for dissagreement of our results with other authors (4). Hypertriglyceridemia obtained in the examined group is the most significant lipid disturbance. It suggests the disturbance in the catabolism of triglyceride-rich lipoproteins, accumulation of atherogenic remnants particles and also takes part in the modification of the composition and structure of LDL particles.

There is also strong evidence for significant HDL-C decrease in both groups of patients. Patients on long-term hemodialysis showed more severe lipid abnormality with lower serum HDL-C (0.94   50.37 mmol/L). Our results are in agreement with other studies of dyslipidemia in chronic renal disease and

hemodialysis patients (19, 24). Lower values for HDL-C in the dialysis treated patients was statistically significantly different compared to CRI patients (p<0.05). Decrease in HDL-C, accompanied with the lower apolipoprotein AI indicates impaired reverse cholesterol transport from peripheral cells to liver and lower anti-atherogenic potencial of HDL lipoproteins. Hypertriglyceridemia and increased lipoprotein remnants in renal insufficiency have been attributed to lower lipoprotein lipase and hepatic lipase activity. Lipoprotein lipase activity correlates positively with HDL-C and inversely with TG level (4, 9). Our lower values for HDL-C are in agreement with lower activity of these enzymes. We found a significant inverse correlation between HDL-C and TG *(Table III)*. In this respect it may be important that in the present study, whilst there was a statistically significant correlation between TG concentration and HDL-C in the dialysis patients as expected, no such relationship was found in the predialysis group. Additional studies are clearly needed to define the precise mechanisms that lead to lower HDL-C in patients on dialysis.

Lipoprotein metabolism is influenced by many determinants as the dialysis treatment itself, the use of heparin as anticoagulant, the membrane material and underlying uraemia. Chronic administration of heparin induces a number of changes in the plasma lipid composition, and especially prominent is a significant rise in plasma TG levels. A possible explanation for heparin potentially contribution to lipid metabolism dysfunction is the interaction of heparin with lipolytic enzymes. Heparin increases the displacement of lipoprotein lipase from its binding sites on endothelial cells and interferes in the metabolism od triglyceride-rich lipoproteins, such as VLDL. Previous studies indicated an improvement in lipid profile of hemodialysis patients following a reduction of heparin dosage or by using low-molecular-weight heparin (25, 26). Our patients were treated with low-molecular-weight heparin and these must also be concerned in comparison with other results as a factor that improves lipid profile.

The influence of different types of dialysis fluid on lipid dysfunction could be of clinical importance. Examined patients were on acetate and bicarbonate dialysis and the obtained results for blood lipid parameters suggests the lipid abnormalities in both types of dialysis but there was no significant differences between these two groups of patients *(Table II)*. Influence of acetate dialysis on acid-base balance in dialysis patients is well known and there are indications that dialysis fluid could have effects on lipid status of patients on chronic hemodialysis. Our data suggest that lipid abnormalities during hemodialysis are not influenced, amongst other factors, by the type of dialysis fluid.

Values for TC and LDL-C indicate lower concentrations in both groups of patients and the obtained results for patients on long-term dialysis are statistically different from the control group. Although TC and

LDL-C are lower, both atherogenic risk factors TC/HDL-C and LDL-C/HDL-C are elevated in both groups of patients suggesting high risk of cardiovascular diseases development. We and others have reported that in patients on dialysis the TC/HDL-C ratio is abnormally high (5.78 2.30) and this ratio is higher compared to CRI patients but not statistically significant (27).

Obtained lipoprotein profile for both patients groups, characterised with marked elevation of TG and lower HDL-C is concerned to be very atherogenic Although elevated LDL-C is a clasical atherogenic risk factor for cardiovascular disease, new evidences suggest that even normal LDL-C can be a very atherogenic particle (8). Indipendent of the ethyology, hypertriglyceridemia leads to prominent compositional and configurational changes of LDL-particles. Alteration in composition, size and configuration of LDL from diabetic and hemodialysis patients, impaired LDL receptor mediated degradation and these modified LDL particles are metabolised via nonsaturable scavanger receptors.

In studies of populations without renal failure, small LDL particles have been associated with hypertriglyceridemia and a low HDL-C, abnormalities characteristic of the uremic dyslipidemia and frequently linked with insulin resistance and syndrome X. Hypertriglyceridemia usually reflects triglyceride-enrichment of VLDL, the natural precursor of LDL. Larger triglyceride enriched VLDL particles form a ready substrate for cholesterol ester transfer protein (CETP), which facilitates trafficking of triglyceride from VLDL to LDL in exchange for cholesterol. Thus, triglyceride content of the precursor VLDL is an important determinant of the size of the LDL (7). The resultant triglyceride-rich LDL particles are smaller and denser with alteration in

the lipid-protein ratio and these leads to configuration changes of apo B which impair binding to the receptor. Small, dense LDL particles have a lower binding affinity for the apoprotein B/E receptor, resulting in longer residence times in plasma, prolonged exposure to oxidant injury and enchanted uptake by non-receptor dependent mechanisms. In addition, small, dense LDL is known to show a greater susceptibility to oxidation (28, 29).

CRI patients are known from experience to have an increased risk of myocardial infarction and ischemia when compared to age and sex-matched controls (30). Lipid abnormalities probably represent one of the several potentially correctable cardiovascular risk factors associated with renal disease. Most studies in general population have emphasized the risks associated with hypecholesterolemia, and it has been more difficult to argue the case for correction of hypertriglyceridemia, the predominant lipid abnormality associated with renal insufficiency. A change in LDL subfraction profiles associated with the accumulation of denser particles is associated with an increased risk of ischaemic heart disease in non-renal populations and should be added to the list of potentially atherogenic disturbances of lipoprotein metabolism associated with uremia (31, 32).

In conclusion, our results indicate that a prominent characteristics of lipid abnormalities in CRI patients are marked hypertriglyceridemia and low HDL-C. Obtained lipoprotein profile is associated with atherogenesis and higher incidence of atherosclerotic cardiovascular complications.

POREMEĆAJ METABOLIZMA LIPOPROTEINA U HRONIČNOJ RENALNOJ INSUFICIJENCIJI

Mirjana Jugović[1], Vesna Spasojević-Kalimanovska[2], Slavica Spasić[2], Zorica Nikolić[1]

[1]*Kliničko-biohemijska laboratorija, Zdravstveni centar Dr. L. Lazarević – Šabac*
[2]*Institut za medicinsku biohemiju, Farmaceutski fakultet, Beograd*

Kratak sadržaj: U pacijenata sa hroničnom renalnom insuficijencijom javlja se poremećaj u metabolizmu lipoproteina koji nosi povećani rizik od ateroskleroze. U ovom radu ispitivan je aterogeni rizik kod bolesnika sa HRI i bolesnika na hemodijalizi određivanjem ukupnog holesterola, triglicerida, HDL-holesterola i LDL-holesterola i izračunavanjem odnosa uH/HDL-H i LDL-H/HDL-H. Poremećaji lipidnog statusa ispitivani su u grupi od 18 pacijenata sa hroničnom renalnom insuficijencijom, grupi od 60 pacijenata na hemodijalizi i poređenjem sa kontrolnom grupom od 85 dobrovoljnih davalaca krvi. Lipidni parametri u serumu određivani su standardnim biohemijskim metodama. Svi lipidni parametri u ispitivanoj grupi pacijenata na hemodijalizi u poređenju sa kontrolnom grupom statistički su se značajno razlikovali (p<0,05), dok su u grupi pacijenata sa hroničnom renalnom insuficijencijom postojale značajne razlike u trigliceridima i HDL-holesterolu. Pacijenti sa hroničnom renalnom insuficijencijom i na hemodijalizi imali su vrlo izraženu hipertrigliceridemiju i niži HDL-holesterol u odnosu na kontrolnu grupu. Svi izračunati aterogeni faktori rizika bili su viši kod pacijenata u odnosu na kontrolnu grupu. Poređenje

lipidnih parametara kod pacijenata sa hroničnom renalnom insuficijencijom u odnosu na pacijente na hemodijalizi ukazalo je na značajnu razliku samo za HDL-holesterol ($p<0,05$). Dobijene visoke vrednosti triglicerida i niske vrednosti HDL-holesterola kod ispitivanih bubrežnih pacijenata ukazuju na povećani rizik od kardiovaskularnih oboljenja. Rezultati se slažu sa drugim istraživanjima koja ukazuju na poremećaj u reverznom transportu holesterola, hipertrigliceridemiju i veću zastupljenost malih gustih LDL čestica, kao i na povišene nivoe potencijalno aterogenih »remnantá čestica.

Ključne reči: dislipidemija; hipertrigliceridemija; aterogeni rizik, hronična renalna insuficijencija; hemodijaliza.

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