ORIGINAL ARTICLE

Lipid status and carotid intima-media thickness in patients with end-stage renal disease

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ABSTRACT

Aim To assess morphological characteristics of carotid blood vessels in uremic patients before to the initiation of the dialysis treatment, and corelate data with various dialysis therapy modules.

Methods The study included 30 patients with end-stage renal disease (ERDS) prior to commencing dialysis, 30 patients treated with haemodialysis and 30 patients treated with continuous ambulatory peritoneal dialysis. The control group consisted of 15 subjects with normal kidney function (eGFR>60ml/min). Carotid intima-media thickness (CIMT), as well as lipid status values (cholesterol, triglycerides, low-density lipoprotein (LDL), high-density lipoprotein (HDL), apolipoprotein A, apolipoprotein B) were evaluated.

Results The significant difference in CIMT was detected between the control and haemodialysis groups ($p<0.001$), and between the control and the peritoneal dialysis group ($p=0.004$). In patients in the predialysis group, CIMT was influenced by cholesterol ($p=0.013$), HDL ($p=0.044$), LDL ($p=0.001$) and ApoB ($p=0.042$) values. A significant difference in CIMT was proved between the haemodialysis and predialysis group of patients ($p<0.001$). The only variable from the patient's lipometabolic profile significantly associated with the change in IMT in uremic patients was HDL. A significant difference was found in the average value for systolic blood pressure ($p<0.001$) and diastolic blood pressure ($p=0.018$) in patients before starting the dialysis treatment compared to patients treated with other dialysis methods.

Conclusion Patients on haemodialysis treatment had a significantly greater CIMT, which is in relation with a higher cardiovascular risk.

Key words: atherosclerosis, kidney failure, prognosis

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INTRODUCTION

Chronic kidney disease (CKD) is defined as a reduction in renal function (glomerular filtration rate <60ml/min/1.73m²) or the existence of evidence of renal damage (such as proteinuria) that lasts for at least 3 months (1,2). When kidney function is reduced that it threatens the patient's life, it is necessary to treat the patient with a system that supports kidney function. There are several ways of treating such patients: peritoneal dialysis (PD), haemodialysis (HD), hemofiltration (HF), hemodiafiltration (HDF), and hemoadsorption (HA) (1,3). Starting dialysis depends on the combination of symptoms, comorbidities and laboratory parameters (3). Atherosclerotic disease manifests through vascular calcification, which can be observed in two arterial structures, in the intima and in the media (4,5,6). Arterial intimal calcification (AIC) is generally associated with atherosclerotic lesions, plaque formation and the development of occlusive lesions. Calcification of vessels and atherosclerotic plaques increases the cardiovascular risk (7). Arterial medial calcification (AMC) occurs in the muscular layer of arteries and causes a decrease in the elasticity of the arterial wall, more often than occlusive lesions (7). AMC is more associated with uraemia (8). Both AIC and AMC can occur in dialysis patients (9). Patients with end-stage renal disease (ESRD) should be understood as those with a high risk of cardiovascular disease, and the effect on the lipid status is essential (10,11). There were no similar investigations on the territory of Bosnia and Herzegovina. The results are very important from the perspective of the management of atherosclerotic disease and dyslipidaemia (12).

The aim of this article was to evaluate the morphological characteristics of carotid blood vessels in uremic patients and correlate values with different modules of dialysis therapy.

METHODS

Patients and study design

The study was conducted at the Clinic for Nephrology and the Clinic for Hemodialysis of the Clinical Centre of the University of Sarajevo during the period January 2020 to January 2022.

The research was retrospective-prospective and provided the assessment of carotid artery remodelling in relation to the lipid profile of uremic patients who did not undergo dialysis treatment, as well as those treated with haemodialysis and peritoneal dialysis in comparison with healthy population.

Research included 30 patients with ESRD prior to commencing the dialysis treatment, 30 patients treated with haemodialysis and 30 patients treated with continuous ambulatory PD. The control group consisted of 15 subjects with normal kidney function (eGFR>60ml/min). Criteria for the inclusion were patients who gave an informed consent for the inclusion in research, those with ESRD before the start of dialysis treatment, patients treated with HD and continuous ambulatory PD, who had no information on ischemic disease in their medical history at the time of inclusion in the study heart disease, cerebrovascular incident, peripheral arterial disease and generalized atherosclerosis, and patients undergoing dialysis treatment with adequate HD and PD, assessed based on Kt/V. Criteria for exclusion were: patients who did not sign a study informed consent, who were treated with both monitored dialysis modules (HD and CAPD), treated with the dialysis for less than 6 months, with proven changes in peripheral arteries of an idiopathic nature or caused by specific or non-specific inflammatory changes and patients with malignancies and chronic infectious diseases in the last 3 months, patients undergoing dialysis treatment with inadequate HD and PD, assessed on the Kt/V basis.

The Ethical Committee of the Clinical Centre University of Sarajevo approved the study (2019-42-31B, approved October 6, 2019).

Methods

Clinical assessment of vascular changes was made by assessment of carotid intima-media thickness test (CIMT). It was measured by echo tomography using a high-resolution 7.5 MHz probe in B mode (Wall-Track system: W-T, Maastricht, The Netherlands). The measurement was performed by an angiologist at the Clinic for Angiology, who had not been previously aware of the identity and clinical status of the patients. Blood pressure was measured with a mercury sphygmomanometer after 15 minutes of rest, according to the recommendations of the British Hypertension Society (12). The body mass index (BMI) is calculated as the quotient of the patient's

body weight expressed in kg/m². Of the blood laboratory parameters relevant for the assessment of the risk of vascular changes in uremic conditions, lipid status values - cholesterol, triglycerides, low-density lipoprotein (LDL), high-density lipoprotein (HDL), apolipoprotein A, apolipoprotein B, haemoglobin, urea, creatinine, calcium (Ca), phosphorus (P), lipid profile, glycemia and C-reactive protein were monitored. These laboratory tests were performed using standard methods at the Clinic of Clinical Biochemistry and Immunology. The level of intact parathyroid hormone was determined radioisotopically at the Institute for Nuclear Medicine, Clinical Centre of the University of Sarajevo.

Statistical analysis

To present continuous variables, the arithmetic mean for normally distributed values was used, and the median for values whose distribution did not follow a normal curve. To measure the average variability, the standard deviation for normally distributed values was used, and the interquartile range for variables that were not normally distributed. Normality was assessed by analysis of residuals and graphical methods. The collected variables between the four groups defined by the therapy module were tested with the ANOVA F-test if they were continuous variables, while categorical ones were tested with the $\chi 2$ or Fisher's exact test, depending on applicability. For post-hoc comparisons, Dunnett's method was used and compared each of the groups with the control group. To evaluate the CIMT parameter in the four groups defined by the therapy module, the simultaneous 95% confidence interval was used for the difference in the average value. The impact of diabetes on the degree of dyslipidaemia and atherogenesis using a linear model of various indicators of dyslipidaemia and atherogenesis was assessed. To assess the significance of the interaction, the Wald significance test from the linear model was used. All tests were performed at a significance level of 0.05, and all confidence intervals were 95%.

RESULTS

The average age of patients on HD was 54.2±10.81 years, on PD 53.1±15.51 years; in the predialysis group 55.6±13.45 years and in the control group 54.8±8.1 years (p = 0.901) (Table 1).

Post-hoc testing showed that a significant difference in CIMT was found between the control and HD group (p<0.001), and between the control and the group of patients on peritoneal dialysis (p=0.004). There was no statistically significant difference in the measured CIMT values between the control and predialysis groups (Table 2).

None of the monitored lipid profile parameters was significantly associated with IMT thickening in patients treated with HD. In patients treated for PD, there was also no correlation between lipid profile and IMT thickness. A significant correlation was found between IMT and DBP values

Table 1. Demographic characteristics of patients

Variable	Total (n=105)	HD (n=30)	PD (n=30)	ESRD (n=30)	Control group (n=15)	p
			No (%) of patients in the group			
Gender						0.8164
Male	56 (53.3)			17 (56.7)	9 (60)	
Female	49 (46.7)	14 (46.7)	16 (53.3)	13 (43.3)	6 (40)	
Diagnosis						<0.001
Chronic glomerulonephritis	20 (19)	11 (36.7)	3 (10)	6 (20)	0 (0)	
Diabetic nephropathy	34 (32.4)	6 (20)	14 (46.7)	14 (46.7)	0 (0)	
Unknown underlying kidney disease	18 (17.1)	8 (26.7)	4 (13.3)	6 (20)	0 (0)	
Polycystic kidney disease	6 (5.7)	4 (13.3)	2 (6.7)	0 (0)	0 (0)	
Chronic pyelonephritis	12 (11.4)	1 (3.3)	7 (23.3)	4 (13.3)	0 (0)	
Healthy	15 (14.3)	0 (0)	0 (0)	0 (0)	15 (100)	
Diabetes						0.0013
Yes	36 (34.3)	5 (16.7)	15 (50)	15 (50)	1 (6.7)	
No	69 (65.7)	25 (83.3)	15 (50)	15 (50)	14 (93.3)	
Smoking						0.0429
Yes	23 (21.9)	4 (13.3)	5 (16.7)	12 (40)	2 (13.3)	
No	82 (78.1)	26 (86.7)	25 (83.3)	18 (60)	13 (86.7)	
Average age (SD) (years)	54.36 ±12.64	54.2 ±10.81	53.1 ± 15.51	55.6± (13.45)	54.8±8.1	0.901
CIMT (IQR) (mm)	0.805 (0.53- 1.06)	0.741 (0.665- 0.81)	0.855 (0.77- 0.93)	0.986 (0.91- 1.06)	0.637 (0.530- 0.744)	0.031

HD, haemodialysis; PD, peritoneal dialysis; ESRD, end stage renal disease; CIMT, carotid intima-media thickness; IQR, interquartile range;

Table 2. Differences in the CIMT parameter between the groups

Parameter	Difference between CIMT		p
	Value (mm)	95% confidence interval	
HD-control	0.348	(0.193, 0.503)	<0.001
PD - control	0.217	(0.062, 0.372)	0.0037
ESRD - control	0.103	(-0.051, 0.258)	0.250

CIMT, carotid intima-media thickness; HD, haemodialysis; PD, peritoneal dialysis; ESRD, end stage renal disease

for the haemodialysis group, while other parameters had no significant effect on IMT. A significant difference in CIMT was proved between the HD and PD groups of patients (p<0.001). In the ESRD group, the parameters that had a significant impact on CIMT were the CRP (p<0.001) and serum albumin levels (p<0.01) (Table 3).

The highest average blood pressure value was recorded in patients treated with haemodialysis (140.7±13.63 mmHg), while the lowest blood pressure values were recorded in the control group (122±8.62 mmHg). The highest average values of diastolic blood pressure were also recorded in the group treated with haemodialysis (82.3±10.73), while the lowest blood pressure values were recorded in the control group (74±7.37). A significant difference was found in the average value for systolic blood pressure (p<0.001) and diastolic blood pressure (p=0.018) in patients before starting the dialysis treatment compared to patients treated with other dialysis methods (Table 4).

The highest cholesterol values were recorded in the predialysis group, 6.5±1.6 mmol/L, while the lowest ones were recorded in the group of patients treated with one of the dialysis methods, 5.5±1.5 mmol/L. Triglyceride values were also the highest in the predialysis group 2.3±0.88 mmol/L, while the lowest values were recorded in the control group 1.8±0.9 mmol/L (Table 5).

Table 3. Carotid intima-media thickness (CIMT) and risk factors

Group (No of patients)		CIMT	CRP	Ca	Albumin	Time of dialysis	SBP	DBP
HD (30)	Pearson Correlation	1	0.337	-0.082	0.337	-0.175	0.331	0.468*
	Sig. (2-tailed)		0.146	0.731	0.146	0.461	0.154	0.037
PD (30)	Pearson Correlation	1	0.399*	0.076	-0.237	-0.298	-0.084	0.034
	Sig. (2-tailed)		0.029	0.239	0.206	0.109	0.657	0.860
ESRD (30)	Pearson Correlation	1	0.001	-0.058	-0.511†		0.170	0.280
	Sig. (2-tailed)		0.997	0.760	0.004		0.370	0.133

*Correlation is significant at the 0.05 level; †Correlation is significant at the 0.01 level; CIMT, carotid intima-media thickness; HD, haemodialysis; PD, peritoneal dialysis; ESRD, end stage renal disease, SBP, systolic blood pressure; DBP, diastolic blood pressure.

Table 4. Clinical and biochemical parameters of the patients

Variable	Mean value (standard deviation)					p
	Total (n=105)	HD (n=30)	PD (n=30)	ESRD (n=30)	Control group (n=15)	
SPB (mmHg)	134.29 (14.13)	140.7 (13.63)	137 (13.68)	131.3 (13.06)	122 (8.62)	<0.001
DBP (mmHg)	79.81 (9.8)	82.3 (10.73)	82 (8.47)	78 (9.97)	74 (7.37)	0.018
CIMT (mm)	0.83 (0.24)	1 (0.21)	0.9 (0.22)	0.7 (0.23)	0.6 (0.13)	<0.001
Cholesterol (mmol/L)	5.85 (1.5)	4.8 (1.31)	6.2 (1.19)	6.5 (1.6)	6 (1.17)	<0.001
Triglycerides (mmol/L)	2.1 (0.92)	2.2 (1.25)	1.9 (0.47)	2.3 (0.88)	1.8 (0.9)	0.266
HDL (mmol/L)	1.14 (0.31)	0.9 (0.23)	1.3 (0.3)	1.1 (0.31)	1.2 (0.25)	<0.001
LDL (mmol/L)	3.61 (1.11)	2.5 (0.67)	3.9 (0.69)	4.4 (0.99)	3.8 (0.9)	<0.001
Lipoprotein (a) (mg/dL)	0.54 (0.37)	0.5 (0.18)	0.4 (0.34)	0.4 (0.24)	1.2 (0.2)	<0.001
apoA (mg/dL)	1.48 (0.46)	1.1 (0.21)	1.9 (0.38)	1.5 (0.44)	1.4 (0.27)	<0.001
apoB (mg/dL)	1.22 (0.36)	1.2 (0.28)	1.2 (0.32)	1.3 (0.43)	1 (0.35)	0.019
apoB_apoA (mg/dL)	0.89 (0.38)	1.1 (0.27)	0.6 (0.14)	1 (0.47)	0.7 (0.3)	<0.001
BMI (kg/m2)	25.63 (3.32)	26.3 (4.41)	25.2 (2.2)	25.4 (3.11)	25.5 (3.2)	0.618
urea (mmol/L)	21.11 (9.02)	23.7 (6.07)	21.1 (5.08)	26.4 (7.89)	5.3 (1.18)	<0.001
creatinine (mmol/L)	708.14 (322.39)	845.3 (231.17)	724.5 (113.97)	867.6 (239.9)	82.2 (10.8)	<0.001
Haemoglobin(g/dL)	113.14 (20.21)	114.5 (25.47)	108.3 (9.23)	102.5 (10.06)	141.3 (12.69)	<0.001
C-reactive protein (mg/L)	7.91 (12.72)	9 (21.77)	5.8 (3.01)	11.5 (8)	2.7 (0.98)	0.113
Calcium (mmol/L)	2.14 (0.38)	1.9 (0.64)	2.2 (0.17)	2.2 (0.15)	2.2 (0.11)	0.011
Phosphor (mmol/L)	1.59 (0.39)	1.8 (0.35)	1.6 (0.21)	1.7 (0.28)	0.9 (0.11)	<0.001
Calcium-Phosphor product (mmol/L)	3.37 (1.01)	3.5 (1.34)	3.5 (0.53)	3.8 (0.64)	2 (0.2)	<0.001
Parathormone (pg/mL)	265.87 (207.06)	391.8 (213.01)	239.7 (175.57)	287.7 (171.15)	22.7 (11.93)	<0.001
Albumin (g/L)	34.78 (5.75)	39.4 (4.62)	30.8 (2.93)	31.4 (4.49)	40.2 (2.73)	<0.001
The average duration of dialysis (years)	2.72 (3.25)	6.2 (3.24)	3.3 (1.64)	0 (0)	0 (0)	<0.001

CIMT, carotid intima-media thickness; HD, haemodialysis; PD, peritoneal dialysis; ESRD, end stage renal disease; SBP, systolic blood pressure; DBP, diastolic blood pressure; HDL, high-density lipoprotein; LDL, low-density lipoprotein; BMI, body mass index; apoA, apolipoprotein A; apoB, apolipoprotein B

Table 5. Lipid parameters of uremic patients

Variable	Total (n=105)	HD and PD (n=60)	Control group (n=15)	ESRD (n=30)	p
Cholesterol (mmol/L)	5.85 (1.5)	5.5 (1.43)	6 (1.17)	6.5 (1.6)	0.015
Triglycerides (mmol/L)	2.1 (0.92)	2.1 (0.94)	1.8 (0.9)	2.3 (0.88)	0.267
HDL (mmol/L)	1.14 (0.31)	1.1 (0.33)	1.2 (0.25)	1.1 (0.31)	0.692
LDL (mmol/L)	3.61 (1.11)	3.2 (0.98)	3.8 (0.9)	4.4 (0.99)	<0.001
Lipoprotein (a) (mg/dL)	0.54 (0.37)	0.4 (0.28)	1.2 (0.2)	0.4 (0.24)	<0.001
apoA (mg/dL)	1.48 (0.46)	1.5 (0.51)	1.4 (0.27)	1.5 (0.44)	0.646
apoB (mg/dL)	1.22 (0.36)	1.2 (0.3)	1 (0.35)	1.3 (0.43)	0.007
apoB_apoA (mg/dL)	0.89 (0.38)	0.9 (0.32)	0.7 (0.3)	1 (0.47)	0.05

HD, haemodialysis; PD, peritoneal dialysis; ESRD, end stage renal disease, HDL, high-density lipoprotein, LDL, low-density lipoprotein, apoA, apolipoprotein A; apoB, apolipoprotein B

DISCCUSSION

In previous research, it was established that vascular alterations on carotid arteries can be an early sign of atherosclerosis and a predictor of generalized vascular changes in the dialysis population (14). Patients with CKD have a different lipid profile than those patients with dyslipidemia in the general population, which depends on the degree of kidney disease, and the lipid status. The present study showed the morphological characteristics of the carotid artery in uremic patients before the initiation of the dialysis treatment, and correlated data with various dialysis therapy modules, primarily lipid profile as one of the risk factors of accelerated atherosclerosis.

Compared to the general population, dialysis patients have a 10-fold higher risk of cardiovascular disease (14,15). However, many studies have shown that changes in the structure of arteries and vascular calcification are determined by conventional cardiovascular risk factors rather than by the degree of renal dysfunction or electrolyte imbalance (16).

CIMT is a non-invasive marker that can indicate the presence of generalized atherosclerosis (17). Cross-sectional studies with cohorts without CKD have shown the association with CIMT, the existence of cardiovascular risk and the presence of cardiovascular disease (17). Benedetto et al. were the first to demonstrate that CIMT is an independent predictor of mortality in patients on HD or PD. They demonstrated that a 0.1 mm increase in IMT predicts a 24% higher risk of cardiovascular mortality (18). In our study, the highest CIMT was noted in HD patients, as well as a statistically significant difference in CIMT values was recorded between the PD and predialysis groups.

In a study with ESRD patients who were treated with different dialysis modules, it was shown that haemodialysis is an independent predictor of the intimal thickening of the carotid arteries (19). On the other hand, in a study (20) that compared healthy individuals with dialysis patients treated with HD or PD, it was proven that the dialysis population had significantly greater structural changes in the carotid arteries compared to the control group. Similar results were obtained in our study in terms of a greater atherogenic process in the dialysis population, but also in patients who are in the non-dialysis ESRD stage compared to the control group with normal kidney function.

Mutluay et al. showed that CIMT was significantly higher in HD and PD patients compared to the control group (20) with no significant difference between CIMT in the PD and HD patients. However, when the sample was stratified according to the duration of dialysis treatment, it was concluded that peritoneal dialysis treatment could have a stronger atherogenic effect after five years of the dialysis treatment compared to the HD treatment

Also, Shoji et al. (21) concluded that CIMT values were higher in predialysis and the HD patients compared to the control group, while there was no significant difference in values between groups with CKD. When it comes to the morphological characteristics of the carotid blood vessels in the observed groups, it was found that the lipid profile did not affect the CIMT in either the HD or the PD group, while in the patients in the predialysis group, a statistically significant relationship was recorded between the IMT thickness and lipid profile. Studies have confirmed that CIMT is an independent predictor of cardiovascular mortality in chronic dialysis patients (22-24).

Lipid disturbances have a significant effect on the outcomes of dialysis treatment. Studies have shown that a decrease in HDL is a significant feature of patients with CKD (25,26). The metabolism of HDL is significantly suppressed in patients undergoing haemodialysis compared to those treated with peritoneal dialysis (25,26). The authors explained the increased values of LDL by the

occurrence of a glucotoxic effect that occurs due to the use of solutions based on glucose, which exposes patients treated with peritoneal dialysis to a greater risk of developing atherosclerosis (27,28). Also, one of the mechanisms that could explain elevated LDL values is hypoalbuminemia, which occurs in patients with PD and is a consequence of protein loss via effluent, and the lipid disorder, in this case, can be interpreted by the same mechanism as in patients with nephrotic syndrome (27). The obtained results show that patients on PD have an increased risk of atherosclerotic changes due to elevated values of total and LDL cholesterol and hypertriglyceridemia. Plasma concentrations of HDL and apolipoprotein A, which have a protective role, were normal in most subjects.

This cross-sectional study had a limitation in the interpretation of the results, primarily due to the small sample of patients. Also, the impact of dyslipidemia on the development of atherosclerosis in uremic patients should be observed over a while, and additional diagnostic methods should be researched to confirm or rule out the existence of changes in large blood vessels and heart valves. Nevertheless, this finding may highlight the importance of following up on kidney function among those with atherosclerosis, particularly when they develop heart failure in the clinical course.

The results of this study suggest the association of significant morphological changes on CCA, which are considered surrogate-generalized atherosclerosis in non-dialysis ESRD and dialysis patients which in turn gives CIMT the importance of a predictor of the development of accelerated atherosclerosis in those patients. However, the treatment with dialysis, with the regulation of vasoactive molecules and other vascular risk factors, importantly impedes vascular remodelling. Intensive monitoring of hemodynamic and non-hemodynamic vascular risk factors and vascular parameters in CKD patients is necessary for the prevention and even regression of undesired changes in the vascular system.

In conclusion, patients on the HD treatment had a significantly a greater thickness of the CIMT suggesting greater tendency to develop cardiovascular incidents compared both to the group of PD patients and the healthy population. The choice of a dialysis method could have an impact on slowing down vascular remodelling in uremic patients.

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TRANSPARENCY DECLARATION

Competing interests: none to declare.

REFERENCES

1. Vaidya SR, Aeddula NR. Chronic Renal Failure. 2022 Aug 10. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2022.
2. Šečić D, Turohan A, Begić E, Rebić D, Pepić E, Begić Z, Iglica A, Begić N, Metović A, Mušanović J. Serum creatinine versus corrected Cockcroft-Gault equation according to Poggio reference values in patients with arterial hypertension. Int J Appl Basic Med Res 2022; 12:9-13.
3. Thiery A, Séverac F, Hannedouche T, Couchoud C, Do VH, Tiple A, Béchade C, Sauleau EA, Krummel T; REIN registry. Survival advantage of planned haemodialysis over peritoneal dialysis: a cohort study. Nephrol Dial Transplant 2018; 33:1411-9.
4. Neto RM, Ramos JGL, Medjedovic E, Begic E. Increased of the carotid intima media thickness in preeclampsia. J Perinat Med 2020; 48:787-91.
5. Begic E, Obradovic S, Jankovic S, Romanovic R, Djenic N, Dzudovic B, Jovic Z, Malovic D, Subota V, Stavric M, Ljuca F, Kusljugic Z. Increased C-reactive protein is associated with major adverse cardiovascular events after STEMI. Erciyes Med J 2020; 42:276-80.
6. Obradovic S, Begic E, Jankovic S, Romanovic R, Djenic N, Dzudovic B, Jovic Z, Malovic D, Subota V, Stavric M, Ljuca F, Kusljugic Z. Association of PC and AT levels in the early phase of STEMI treated with pPCI with LV systolic function and 6-month MACE. Acta Clin Belg 2020; 1-7.
7. Zwakenberg SR, de Jong PA, Hendriks EJ, Westerink J, Spiering W, de Borst GJ, Cramer MJ, Bartstra JW, Doesburg T, Rutters F, van der Heijden AA, Schalkwijk C, Schurgers LJ, van der Schouw YT, Beulens JWJ; SMART Study Group. Intimal and medial calcification in relation to cardiovascular risk factors. PLoS One 2020; 15:e0235228.
8. Sutliff RL, Walp ER, El-Ali AM, Elkhatib S, Lomashvili KA, O'Neill WC. Effect of medial calcification on vascular function in uremia. Am J Physiol Renal Physiol 2011; 301:F78-83.
9. London GM, Guérin AP, Marchais SJ, Métivier F, Pannier B, Adda H. Arterial media calcification in end-stage renal disease: impact on all-cause and cardiovascular mortality. Nephrol Dial Transplant 2003; 18:1731-40.

10. Lokesh S, Kadavanu TM, Green SR, Dutta TK, Hemachandar R, Ramachandrappa AK, Tiwari SR, Govindasamy E. A Comparative study of lipid profile and cardiovascular risk biomarkers among chronic haemodialysis patients and healthy individuals. J Clin Diagn Res 2016; 10:OC15-9.
11. Miseljic S, Aziri B, Begic E, Rebic D, Džubur A, Mišeljic N, Mekic M, Resic H, Begic N, Zukic F. Hemodialysis parameters and pulse wave velocity. Int J App Basic Med Res 2022; 1-7.
12. Funamizu T, Iwata H, Chikata Y, Doi S, Endo H, Wada H, Naito R, Ogita M, Kato Y, Okai I, Dohi T, Kasai T, Isoda K, Okazaki S, Miyauchi K, Minamino T. A prognostic merit of statins in patients with chronic hemodialysis after percutaneous coronary intervention-a 10-year follow-up study. J Clin Med 2022; 11:390.
13. Sheppard JP, Lacy P, Lewis PS, Martin U; Blood Pressure Measurement Working Party of the British and Irish Hypertension Society. Measurement of blood pressure in the leg-a statement on behalf of the British and Irish Hypertension Society. J Hum Hypertens 2020; 34:418-9.
14. Junyent M, Martínez M, Borràs M, Coll B, Valdivielso JM, Vidal T, Sarró F, Roig J, Craver L, Fernández E. Predicting cardiovascular disease morbidity and mortality in chronic kidney disease in Spain. The rationale and design of NEFRONA: a prospective, multicenter, observational cohort study. BMC Nephrol 2010; 11:14.
15. Major RW, Cheng MRI, Grant RA, Shantikumar S, Xu G, Oozeerally I, Brunskill NJ, Gray LJ. Cardiovascular disease risk factors in chronic kidney disease: a systematic review and meta-analysis. PLoS One 2018; 13:e0192895.
16. Hinderliter A, Padilla RL. Association of carotid intima-media thickness with cardiovascular risk factors and patient outcomes in advanced chronic kidney disease: the RRI-CKD study. Clin Nephrol 2015; 84:10-20
17. Emma P, Mary RE, Elena K, Alun HD, Edwina AB. Association between carotid artery intima-media thickness and cardiovascular risk factors in CKD. Am J Kidney Dis 2005; 46:856-62.
18. Benedetto FA, Mallamaci F, Tripepi G, Zoccali C. Prognostic value of ultrasonographic measurement of carotid intima media thickness in dialysis patients. J Am Soc Nephrol 2001; 12:245864.
19. Vaziri ND. Oxidative stress in uremia: nature, mechanisms, and potential consequences. Semin Nephrol 2004; 24:469-73.
20. Mutluay R, Degertekin CK, Poyraz F, Yılmaz MI, Yücel C, Turfan M, Tavil Y, Derici Ü, Arınsoy T, Sindel S. Dialysis type may predict carotid intima media thickness and plaque presence in end-stage renal disease patients. Adv Ther 2012; 29:370-82.
21. Shoji T, Emoto M, Tabata T, Kimoto E, Shinohara K, Maekawa K, Kawagishi T, Tahara H, Ishimura E, Nishizawa Y. Advanced atherosclerosis in predialysis patients with chronic renal failure. Kidney Int 2002; 61:2187-92.
22. Nishizawa Y, Shoji T, Maekawa K, Nagasue K, Okuno S, Kim M, Emoto M, Ishimura E, Nakatani T, Miki T, Inaba M: Intima-media thickness of carotid artery predicts cardiovascular mortality in hemodialysis patients. Am J Kidney Dis 2003; 41:S76 –9.
23. Ekart R, Hojs R, Hojs-Fabjan T, Balon BP. Predictive value of carotid intima media thickness in hemodialysis patients. Artif Organs 2005; 29:615-9.
24. Małyszko J, Łebkowska U, Małyszko JS, Myśliwiec M. Structural changes in carotid arteries and impairment of fibrinolysis in hemodialyzed and peritoneally dialyzed patients. Med Sci Monit 2009; 15:CR644-9.
25. Ferro CJ, Mark PB, Kanbay M, Sarafidis P, Heine GH, Rossignol P, Massy ZA, Mallamaci F, Valdivielso JM, Malyszko J, Verhaar MC, Ekart R, Vanholder R, London G, Ortiz A, Zoccali C. Lipid management in patients with chronic kidney disease. Nat Rev Nephrol 2018; 14:727-49.
26. Holzer M, Schilcher G, Curcic S, et al. Dialysis Modalities and HDL Composition and Function. J Am Soc Nephro 2015; 26:2267-76.
27. Kanbay M, Bavbek N, Delibasi T, Koca C, Kaya A, Altay M, Akcay A, Duranay M, Yigitoglu R. Effect of peritoneal dialysis solution type on serum lipid levels in end-stage renal disease. Ren Fail 2007; 29:309-13.
28. Zvizdić F, Begić E, Dilić M, Sokolović Š, Lepara O. Effect of atorvastatin on systolic and diastolic function in patients with heart failure with reduced ejection fraction (HFrEF). Med Glas (Zenica) 2021; 18:357-61.