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CLINICAL STUDY

Left ventricular remodeling and arterial remodeling in patients with chronic kidney disease stage 1–3

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Abstract

Introduction: Chronic kidney disease (CKD) is an independent factor for cardiovascular system complications, such as arterial hypertension, left ventricular hypertrophy (LVH), heart failure or accelerated atherosclerosis progression. The aim of the paper was to analyze left ventricular and arterial remodeling in patients with CKD stages 1–3 to identify the subclinical marker of cardiovascular system damage which changes first in the course of CKD. *Methods*: The examined group consisted of 90 patients with CKD stage 1–3 and 30 subjects constituting the control group. Left ventricular mass index (LVMI), left ventricular relative wall thickness (RWT) and ejection fraction (EF) were determined by echocardiographic examination. Pulse wave velocity (PWV) between the carotid and femoral arteries as well as common carotid artery intima–media thickness (IMT) was measured. 24-h ambulatory blood pressure monitoring was performed in all subjects. *Results*: No differences were found between blood pressure values in the examined groups of patients with CKD1, CKD2 and CKD3. Concentric remodeling was found in 20.0%, concentric hypertrophy in 22.2% and eccentric hypertrophy in 18.9% of patients. LVMI values in patients with CKD2 and 3 were higher than in the control group. IMT values in patients with CKD3 were higher than in patients with CKD2. PWV in patients with stage 3 CKD was significantly higher than in the control group ($p < 0.05$). *Conclusions*: In the course of CKD, the left ventricle undergoes remodeling earlier than large arterial vessels. Echocardiographic assessment of LVH in early stages of CKD may identify patients at increased cardiovascular risk.

Keywords

Chronic kidney disease, left ventricular myocardial hypertrophy, pulse wave velocity, vascular stiffness

History

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Introduction

Chronic kidney disease (CKD) affects approximately 11% of adults. It constitutes an independent risk factor for cardiovascular diseases (CVD).[1] Cardiovascular morbidity and mortality rates are higher in patients with early stages of CKD and increase along with the level of glomerular filtration rate (eGFR) impairment.[2]

Cardiovascular lesions in patients with CKD are associated with traditional risk factors, such as male gender, tobacco smoking, arterial hypertension (AH), lipid disorders and diabetes, as well as factors peculiar to kidney disease: anemia, malnutrition, oxidative stress, chronic inflammatory state, hyperhomocysteinemia or uremic toxemia.[3]

A decrease in glomerular filtration rate (GFR) is associated with left ventricular remodeling and an increase prevalence of left ventricular hypertrophy (LVH).[4,5] At the same time, there occurs an increase in pulse wave velocity (PWV), which constitutes a marker of arterial stiffness.[5,6] Increased arterial stiffness is consequence of altered structural and mechanical properties of arterial wall.[7] It constitutes one of the pathogenetic factors of LVH due to the associated increase in left ventricular afterload.[8]

One of the indicators of structural remodeling of the arterial wall is intima–media thickness (IMT), i.e., thickness of the tunica intima and tunica media layers of blood vessels measured ultrasonographically.[9] A higher IMT value is considered as a factor associated with the subclinical manifestation of atherosclerosis.

The aim of the paper was to evaluate left ventricular remodeling and arterial vascular remodeling in patients with CKD stage 1–3 and to identify the subclinical marker of cardiovascular system damage which changes first in the course of CKD.

Patients and methods

The study was performed in the period from September 2012 to November 2014, after having been granted an approval

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from the Bioethics Committee at the Nicolaus Copernicus University Collegium Medicum in Bydgoszcz. Informed consent was obtained from all patients included in the study.

The study consisted of three phases:
I – included an evaluation of the cardiovascular system in patients with CKD1–3 and the control group,
II – consisted of 6-month supplementation of Gold Omega 3, 2×1000 mg in patients with CKD1–3 and the control group,
III – included an evaluation of the cardiovascular system in patients with CKD and the control group after supplementation of omega-3.

This paper contains data of patients with CKD stage 1–3 and control group on the cardiovascular system before starting omega-3 acids supplementation. This phase of the study lasted from September 2012 to December of 2013.

Recruitment of research subjects was based on a review of medical history of patients treated at the Nephrology Outpatient Clinic of the University Hospital No. 1 in Bydgoszcz.

The study group consisted of 90 patients with CKD stage 1–3 without diabetes who from September 2012 until July 2013 if they were to visit a doctor in the Clinic of Nephrology. Forty patients refused to participate in the study. Preliminary verification of patients in the study was based on serum creatinine in the blood made on a visit to the Department of Laboratory Diagnostics of the University Hospital No. 1 in Bydgoszcz or in another laboratory to which the patient is reported. On this basis, GFR was calculated and initially 30 patients were classified as group 1 patients with CKD, 30 patients in group 2 CKD and 30 patients in stage 3 CKD. Finally, on the basis of the calculated eGFR after using the formula CKD-EPI[10] and patients were separated into three groups: group I – patients with CKD1 ($n = 30$; 33.3%), group II – patients with CKD2 ($n = 33$; 36.7%), group III – patients with CKD3 ($n = 27$; 30.0%). Immunosuppressive therapy was a criterion for exclusion of the patient from the study.

The control group consisted of 30 individuals without AH, obesity, diabetes, CKD and with normal creatinine concentration in blood serum.

The causes of CKD in the studied patient population were as follows: chronic glomerulonephritis confirmed by renal biopsy ($n = 16$; 17.7%), hypertensive nephropathy ($n = 3$; 3.3%), polycystic kidney disease ($n = 29$; 32.2%), gouty nephropathy ($n = 5$; 5.5%), nephrolithiasis ($n = 23$; 25.5%), status after nephrectomy for pyonephrosis, trauma or tumor ($n = 3$; 3.3%). In 11 (12.2%) cases, the cause of CKD remained unknown.

Each patient underwent a clinical assessment which included the measurement of arterial blood pressure (BP) and the calculation of body mass index (BMI). Systolic (SBP) and diastolic blood pressures (DBP) were measured using the A&D Medical UA-631 automatic blood pressure monitor. Subsequently, mean arterial pressure (MAP) was calculated from the formula $MAP = DBP + 1/3(SBP - DBP)$ (mmHg) and pulse pressure (PP) was calculated from the formula $PP = SBP - DBP$ (mmHg).

Each of the studied patients was subjected to 24-h ambulatory blood pressure monitoring (ABPM) using the A&D TM2430 device at home. Cuff size was adapted to a patient's arm circumference. BP measurement was taken every 30 min. The patients were instructed on the operation principles of the apparatus. In the evaluation of BP results obtained by ABPM, mean values of SBP, DBP, MAP and PP for the entire period of 24 h were analyzed. Mean values of MAP and PP were calculated in the same manner as a one-time measurement with the automatic device.

The duration of AH and the administered antihypertensive treatment were determined on the basis of medical history and documentation analysis.

All subjects underwent outpatient an echocardiographic examination including measurements of interventricular septum thickness in end-diastole (IVSd), left ventricular internal dimension in end-diastole (LVIDd) and posterior wall in end-diastole (PWd), all taken in accordance with guidelines set out by the American Society of Echocardiography.[11] Left ventricular mass was calculated using the formula developed by Devereux and colleagues: $LVM = 0.8[1.04(IVSd + LVIDd + PWd)^3 - LVIDd^3] + 0.6$ (g), after which the left ventricular mass index was calculated (LVMI = LVM/BSA – where BSA stands for body surface area).[12] Left ventricular end-diastolic volume (EDV) and end-systolic volume (ESV) were assessed using area–length method. Ejection fraction (EF) was calculated subsequently. Left ventricular relative wall thickness was also calculated ($RWT = 2 * PWd)/LVIDd$.

Impairment of left ventricular systolic function was identified when $EF < 55\%$. The criterion for LVH identification was $LVMI > 95$ g/m^2 for women and $LVMI > 115$ g/m^2 for men.[13] On the basis of LVMI and RWT, four types of left ventricular geometry were determined:
- normal structure of the left ventricle: without LVH and RWT ≤ 0.42,
- concentric remodeling: without LVH and RWT > 0.42,
- concentric hypertrophy: LVH and RWT > 0.42,
- eccentric hypertrophy: LVH and RWT ≤ 0.42.

Aortic PWV was measured between the carotid artery and the femoral artery using the Complior device (Artech Medical, Pantin, France).

Testing was performed under fasting conditions, in a quiet room, after a 10-min rest, in the supine position on an outpatient basis. One sensor was placed at a palpable pulse site on the right carotid artery, while the second sensor was placed at a palpable pulse site on the right femoral artery. Time (t) between the occurrence of pulse wave in the carotid and femoral arteries was measured automatically in 10 subsequent cycles and averaged. Pulse wave distance (d) was accepted as the distance between sensor attachment sites on the carotid and femoral arteries multiplied by coefficient 0.8 in accordance with current guidelines.[14] PWV was calculated using the equation $PWV = d/t$ and expressed in [m/s]. PWV was measured twice in each subject, one measurement taken directly after another, and mean values were calculated.

Each subject underwent an ultrasonographic measurement of common carotid artery IMT. The patients were examined in the supine position, after a 5-min rest. IMT was measured 10–30 mm below the carotid bifurcation in three points free from atherosclerotic plaque both on the left- and right-side. Arithmetic mean was then calculated from the obtained results.

Each patient underwent a fasting blood draw for the purposes of laboratory testing which included the determination of: hemoglobin concentration, creatinine concentration in serum, uric acid, urea, C-reactive protein (CRP) concentration and lipid profile.

Statistical analysis

The obtained results were analyzed statistically using the STATISTICA (Bydgoszcz, Poland) software from StatSoft Inc. The ANOVA non-parametric test was used to compare results in more than two groups. The Tukey's test was used *post-hoc* for a detailed identification of statistically different groups. Data were expressed as mean value ± standard deviation (SD). The distribution of variables was analyzed using the Shapiro–Wilk test. For variables of non-normal distribution, both the range and median have been presented. The statistical significance level was accepted as $p < 0.05$.

Results

Characteristics of the studied patient groups with CKD1–3 and the control group are presented in Table 1. In CKD1, higher IVSd was found alongside no differences in the remaining echocardiography parameters compared to control and normal BP values. In CKD2, a thickening of IVSd and PWd and an increase in LVMI compared to the control were discovered. In CKD3, a thickening of IVSd and PWd and an increase in LVMI, RWT and IMT were found. Table 2 presents the multivariate analysis of IVSd and LVMI in patients with CKD stage 1–3. Due to the analysis, the small size of individual groups was carried out without splitting stages of CKD. In multivariate analysis, IVSd is significantly correlated with BMI ($p = 0.0004$), number of antihypertensive drugs ($p = 0.008$), eGFR CKD-EPI ($p = 0.005$), serum hemoglobin levels ($p = 0.008$). LVMI is significantly correlated with number of antihypertensive drugs ($p = 0.0002$), hemoglobin concentration ($p = 0.009$), ABPM SBP ($p = 0.037$).

Arterial stiffness, as measured by PWV in patients with CKD3 was significantly higher than in the control group ($p < 0.05$).

In the present study, AH was diagnosed in 56.7% of patients with CKD1, 84.9% with CKD2 and 96.3% with CKD3.

Duration of hypertension and antihypertensive therapies and statin therapy in groups of patients with CKD1–3 is shown in Table 3.

AH duration was significantly statistically longer in patients with CKD3 compared to patients with CKD1 and 2 ($p < 0.001$, $p < 0.05$). The number of antihypertensive medications used increased along with advancing stages of CKD and presented as follows: in CKD1 the median was 1.0 (range: 0–4), in CKD2 the median was 1.0 (range: 0–5) and in CKD3 the median was 2.0 (range: 0–5).

No statistically significant differences were identified between the studied groups of patients with CKD in terms of the value of BP measured both in a physician's office and by ABPM.

Left ventricular concentric hypertrophy was found in 20 subjects (22.2%) and left ventricular eccentric hypertrophy in 17 subjects (18.9%). Additionally, concentric remodeling was present in 18 subjects (20.0%).

Table 1. Characteristics of the studied groups of patients with CKD1–CKD3 and the control group.

Parameter	Control (C) ($n = 30$)	CKD1 ($n = 30$)	CKD2 ($n = 33$)	CKD3 ($n = 27$)	ANOVA p
Gender women/men	19/11	14/16	16/17	11/16	
Age (years)	54 ± 11	50 ± 11	57 ± 11[1]	63 ± 7[2,3]	<0.05
BMI (kg/m^2)	24.8 ± 3.1	26.5 ± 4.0	27.4 ± 4.07[3]	27.8 ± 4.0[3]	<0.05
Total cholesterol (mmol/L)	4.99 ± 1.55	5.35 ± 1·29	4.92 ± 1·07	5.02 ± 1·37	NS
HDL fraction cholesterol (mmol/L)	1.41 ± 0.41	1.26 ± 0·37	1·33 ± 0·35	1.19 ± 0·36	NS
LDL fraction cholesterol (mmol/L)	2.92 ± 0.96	3.12 ± 0·99	2.71 ± 0·86	2.63 ± 0·99	NS
Triglycerides (mmol/L)	1.13 ± 0.46	1.57 ± 0·85	1.42 ± 0·60	1.71 ± 0·71[4]	<0.05
Creatinine (μmol/L)	67.18 ± 17.68	66.30 ± 15·03	87·52 ± 14·14[2,4]	129·95 ± 25·64[4,5,6]	<0.0001
Uric acid (μmol/L)	253.4 ± 79.7	274.2 ± 61.3	348.5 ± 92·2[4,5]	397.9 ± 98·7[2,4]	<0.0001
eGFR CKD-EPI (mL/min/1.73 m^2)	100.92 ± 23.57	101.42 ± 9.45	74.47 ± 8.98[2,4]	46.36 ± 8.07[2,4,7]	<0.001
Hemoglobin (mmol/L)	8.63 ± 0.80	9·07 ± 0.93	8·71 ± 1.27	8·50 ± 0.91	NS
CRP (mg/L)	2.57 ± 5.12	1.36 ± 2.34	2.80 ± 3.58[5]	3.38 ± 5.76	<0.05
SBP ABPM (mmHg)	123 ± 10	124 ± 11	125 ± 13	131 ± 16	NS
DBP ABPM (mmHg)	74 ± 5	75 ± 8	75 ± 8	77 ± 9	NS
MAP ABPM (mmHg)	91 ± 6	91 ± 9	90 ± 10	95 ± 11	NS
PP ABPM (mmHg)	49 ± 7	48 ± 6	50 ± 8	54 ± 12	NS
SBP (mmHg)	132 ± 15	136 ± 14	135 ± 14	141 ± 16	NS
DBP (mmHg)	80 ± 9	86 ± 9	83 ± 10	84 ± 6	NS
MAP (mmHg)	98 ± 9	103 ± 10	100 ± 9	103 ± 8	NS
PP (mmHg)	52 ± 13	50 ± 9	51 ± 13	58 ± 15	NS
IVSd (cm)	0.88 ± 0.16	1.04 ± 0.21[3]	1.05 ± 0.20[4]	1.17 ± 0.21[4]	<0.001
LVIDd (cm)	4.81 ± 0.49	5.03 ± 0.50	4.94 ± 0.44	4.95 ± 0.56	NS
PWd (cm)	0.88 ± 0.15	0.98 ± 0.17	1.01 ± 0.17[3]	1.10 ± 0.15[1,4]	<0.001
LVIDs (cm)	3.22 ± 0.41	3.32 ± 0.41	3.18 ± 0.43	3.42 ± 0.54	NS
EF (%)	65 ± 6	65 ± 5	64 ± 8	62 ± 10	NS
RWT (cm)	0.37 ± 0.08	0.39 ± 0.07	0.41 ± 0.08	0.45 ± 0.06[1,8]	<0.05
LVM (g)	150.9 ± 39.3	194.42 ± 59.71[3]	193.32 ± 52.14[3]	222.95 ± 71.34[4]	<0.001
LVMI (g/m^2)	82.6 ± 17.8	99.2 ± 25.1[3]	98.59 ± 20.66[3]	113.14 ± 30.55[4]	<0.001
IMT (mm)	0·63 ± 0.13	0·59 ± 0.13	0.67 ± 0.13	0.72 ± 0.13[5]	<0.05
PWV (m/s)	8.58 ± 1.63	8.66 ± 2.00	9.18 ± 2.09	10.03 ± 2.39[3]	<0.05

In the group of patients with CKD1–3, the following statistically significant correlations of IMT were found: negative with eGFR CKD-EPI ($r=-0.21$; $p<0.05$), EF ($r=-0.37$; $p<0.001$), and positive with age ($r=0.67$; $p<0.001$), RWT ($r=0.33$; $p<0.05$), LVM ($r=0.38$; $p<0.001$), LVMI ($r=0.43$; $p<0.001$), CRP ($r=0.43$; $p<0.001$), SBP ABPM ($r=0.37$; $p<0.001$). In the above-mentioned group, a statistically significant positive correlation was also found between PWV and DBP ABPM ($r=0.24$; $p<0.05$). Statistically significant positive correlations of LVMI were found with creatinine concentration ($r=0.38$; $p<0.001$), BMI ($r=0.25$; $p<0.05$), RWT ($r=0.35$; $p<0.05$), SBP ABPM ($r=0.37$; $p<0.001$), DBP ABPM ($r=0.23$; $p<0.05$). In the same group, a statistically significant negative correlation was found between LVMI and EF ($r=-0.23$; $p<0.05$). In patients with CKD1, a statistically significant positive correlation was found between LVMI and number of antihypertensive drugs ($r=0.43$; $p=0.04$). In patients with CKD2, a statistically significant positive correlation was found between LVMI and BMI ($r=0.35$; $p=0.05$), LVMI and AH duration ($r=0.53$; $p=0.002$). In patients with CKD3, a statistically significant negative correlation was found between LVM and heart rate ($r=-0.42$; $p=0.028$), and positive correlations of LVMI and hemoglobin concentration ($r=0.43$; $p=0.04$), SBP ABPM, MAP ABPM and PP ABPM ($r=0.39$; $p=0.012$, $r=0.40$; $p=0.04$, $r=0.43$; $p=0.024$, respectively) and number of antihypertensive drugs ($p=0.038$, $r=0.40$). For comparison, the control group demonstrated the influence of age ($r=0.43$; $p=0.17$), SBP ABPM, DBP ABPM, MAP ABPM ($r=0.48$; $p=0.007$, $r=0.62$; $p=0.0000$, $r=0.51$; $p=0.004$, respectively) BMI ($r=0.48$; $p=0.007$) with LVMI. In patients with CKD, in the various stages of the disease, disappears relationship effect of age and LVMI.

Table 2. Results of multiple analysis with interventricular septal thickness in diastole (IVSd) as a dependent variable (Model 1) and left ventricular mass index (LVMI) as a dependent variable (Model 2) in patients with CKD1–3.

	Beta	Beta standard error	*p*
Model 1			
$R=0.64$; $R^2=0.41$; Adjusted $R^2=0.38$			
$F=13.95$; $p=0.0000$			
BMI	0.33	0.09	0.0004
Number of antihypertensive drugs	0.26	0.09	0.008
eGFR CKD-EPI	−0.27	0.09	0.005
Hemoglobin	0.24	0.09	0.008
Model 2			
$R=0.54$; $R^2=0.29$; Adjusted $R^2=0.27$			
$F=11.63$; $p=0.0000$			
Number of antihypertensive drugs	0.38	0.10	0.0002
Hemoglobin	0.25	0.09	0.009
SBP ABPM	0.20	0.10	0.037

Discussion

The present study of patients with CKD stage 1–3 aimed at analyzing which of the subclinical indicators of cardiovascular damage changes first. According to our knowledge such data are published for the first time.

CKD is a health problem of widespread prevalence. The disease afflicts approximately 600 million people worldwide and over 4 million Polish citizens.[15] Its development is associated with the occurrence of such complications as AH, LVH, heart failure and accelerated progression of arterial atherosclerosis.[4,5,16] Interplay between kidney and heart function begins prior to the development of symptomatic heart failure and kidney disease.[17] The mechanisms of association between impaired kidney function and CVD are not fully understood: a chronic activation of the renin–angiotensin system (RAAS) is a hallmark of CKD. A persistent activation of RAAS directs damaging effects on cardiac function and contributes to the progression of heart failure *via* promotion of cardiac remodeling and myocardial fibrosis.[18] Changes in the structure and functioning of the heart and blood vessels occur in the early stages of chronic nephropathies.[19] This was confirmed by Stefański et al., who examined patients with IgA nephropathy – without hypertension and with normal glomerular filtration – and found left ventricular structural changes and diastolic function impairment.[19]

Adverse cardiovascular prognosis accompanies the progression of CKD.[2] In a 5-year observation period of patients with CKD1–4 it was found that the population was more susceptible to the risk of cardiovascular events than to their health status requiring renal replacement therapy.[20] In this study, it was recommended to take actions focused on treatment and prevention of coronary heart disease, congestive heart failure, diabetes and anemia, with the aim of reducing mortality rates in patients with early stages of CKD.

The prevalence of LVH in patients with CKD3–5 was 45.2–78%,[4,5,21] whereas in patients with CKD1–2 it was 51%.[21] In other studies investigating the prevalence of LVH in patients with CKD2–3, evidence of LVH was found in 43.2%

Table 3. Duration of hypertension and antihypertensive therapies and statin therapy in groups of patients with CKD1–3.

	CKD1 ($n=30$)	CKD2 ($n=33$)	CKD3 ($n=27$)	Test chi-square
Duration of hypertension median (range)	Median 3 (range: 0–30)	Median 10.5 (range: 0–35)	Median 15 (range: 0–35)[1,2]	<0.001
ACEI *n* (%)	12 (40.0%	14 (42.4)	17 (63.0%)	NS
ARB *n* (%)	5 (14.3%)	6 (18.2%)	4 (14.8%)	NS
Calcium antagonists *n* (%)	6 (20.0%)	13 (39.4%)	15 (55.6%)	<0.05
Beta blocker *n* (%)	4 (13.3%)	15 (45.5%)	11 (44.7%)	<0.05
Diuretic *n* (%)	4 (13.3%)	9 (27.3%)	8 (30.0%)	NS
Other medication for high blood pressure *n* (%)	0 (0%)	4 (12.1%)	6 (22.2%)	<0.05
Statin *n* (%)	5 (16.7%)	11 (33.3%)	11 (40.7%)	NS

of subjects,[22] similarly to the present study, where 41.1% of patients with CKD1–3 showed evidence of LVH, and 20.0% showed left ventricular concentric remodeling, which also is of adverse prognostic significance.[23] The number of patients diagnosed with LVH may be attributed to varying criteria for LVH diagnosis adopted by individual researchers.

In the present study, concentric hypertrophy was prevalent in patients with CKD 2 and 3. More frequent occurrence of concentric hypertrophy in both early 1–2 and advanced 3–5 stages of the disease was also determined by other researchers.[22]

Among factors related to LVM increase in CKD patients, apart from decreased creatinine clearance, emphasis is also placed on age, occurrence and duration of AH and the progression of anemia.[3,24,25]

It was also found that LVM was higher in CKD1 patients with AH than in CKD1 patients without AH (222.9 ± 44.8 vs. 162.4 ± 36.8 g; $p = 0.003$). Increase in LVM in patients with CKD1 and AH, as compared with patients in the same stage of disease but without AH, confirms the contribution of AH in LVH pathogenesis.

The relationship of LVM with AH is confirmed by positive correlations of LVMI with the number of antihypertensive drugs found in patients with CKD1, positive correlations of LVMI with BMI and AH duration found in patients with CKD2 and positive correlations of LVMI with SBP ABPM, MAP ABPM PP ABPM and number of antihypertensive drugs found in patients with CKD3.

Increased arterial IMT constitutes a parameter which reflects both early atherosclerotic lesions and arterial tunica media hypertrophy.[26] Increased IMT allows assessing the risk of cardiovascular events.[27]

In the present study, IMT value was significantly statistically higher in patients with CKD3 than in patients with CKD1 ($p = 0.0265$). The hereby determined tendency of IMT value to increase in subsequent groups of patients along with a decrease in eGFR value may indicate that CKD is conducive to the progression of vascular lesions. Similar results were obtained in another study of patients with CKD1–5.[28] Other researchers found a statistically significant negative correlation between eGFR and IMT values in a group of patients with CKD1–3.[29] Similarly, in the present study of patients with CKD1–3, a statistically significant negative correlation between eGFR and IMT was discovered. However, Konings et al.,[30] who investigated predialysis CKD patients and patients treated with hemodialysis or peritoneal dialysis did not find differences in IMT values between individual groups of patients with CKD and the control group.

The parameter reflecting the elastic properties of vessels is PWV. Its increase is indicative of increased vascular stiffness. The prognostic value of PWV measurement was determined in the population of patients with AH.[31] According to the European Society of Cardiology and the European Society of Hypertension 2013 guidelines, the value of PWV >10 m/s is accepted as an indicator of subclinical organ damage in AH.[13]

According to the literature, the following predictive factors for increased PWV in CKD patients are enumerated, age, male gender, black race, concomitant diabetes, glucose concentration in blood serum, waist circumference, blood pressure and the presence of vascular calcification.[6,32]

The tendency of PWV to increase in subsequent stages of CKD was identified by Wang et al.,[6] who investigated a group of patients with CKD1–5 and found a statistically significant negative correlation between PWV and eGFR. In the present study, arterial stiffness assessed by measuring PWV did not increase along with renal excretory function impairment. In the study by Wang et al., subjects were older than those participating in the present study (60 vs. 56 years old), 30.4% of patients were diagnosed with diabetes, and male gender prevailed (63%). According to the literature, diabetes is one of the key factors determining increased arterial stiffness in CKD patients.[32] In the present study, diabetes was a criterion for exclusion from the investigation, while male gender constituted 53% of the subjects. Mean values of SBP and DBP in the study by Wang et al. were comparable to those obtained in the present study.

Another report concerning a group of 104 patients with CKD1–5, 25% of whom had diabetic kidney disease, found a correlation of PWV with the progression of such organ complications as LVH, increased arterial IMT and arterial calcification.[29] Similarly, other authors[33] investigating a group of patients with CKD3–5 found statistically significant positive correlations of PWV with LVMI and IMT with PWV. In the present study, it was determined that IMT increased along with an increase in LVMI.

Available literature highlights the significance of the relationship between atherosclerotic plaque and vascular elastic properties determining vascular stiffness. This is confirmed by results of a study whereby the distensibility of the internal carotid artery narrowed by atherosclerotic plaque was measured ultrasonographically and compared with the normal contralateral artery.[34] It was found that both the stenosed internal carotid artery and the morphologically unchanged ipsilateral common carotid artery showed lesser distensibility than the contralateral vessels.

To conclude, it has to be stated that a decrease in GFRs corresponds with an increase in the prevalence of subclinical markers of organ damage, such as LVH and IMT, of the carotid arteries. In the course of CKD, the left ventricle undergoes remodeling earlier than large arterial vessels. Echocardiographic assessment of LVH in early stages of CKD may identify patients at increased cardiovascular risk.

Conclusions

In the course of CKD, the left ventricle undergoes remodeling earlier than large arterial vessels. Echocardiographic assessment of LVH in early stages of CKD may identify patients at increased cardiovascular risk.

Declaration of interest

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