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Is aortic elasticity associated with hydration status in stage of chronic renal disease in children?

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ABSTRACT

Objective: We aimed to evaluate cardiovascular risks and influencing factors by measuring aortic elasticity parameters and carotid intima thickness in children with cronic kidney disease (CKD), and also evaluated the hydration status of patients with bioimpedance spectroscopy (BIS) measurements and investigated the effect of hydration status on vascular functions.

Patients and Methods: The study group included an average of 13.3 ± 3.7 years (16 girls and 22 boys, 38 CKD patients), control group on average 12.1 ± 2.9 (16 girls and 15 boys, 31 healthy children). Systolic and diastolic diameters of the aortic annulus and aorta at each level were obtained; z-scores, aortic strain, distensibility, stiffness index were calculated. Carotid intima-media thickness and flow – mediated dilatation were studied. Bioimpedance spectroscopy was performed to all patients.

Results: Interventricular septum and left atrial (p=0.002, p=0.013), sinus valsalva and sinotubular junction z scores (p=0.009, p=0.012) were found to be higher and distensibility and strain decreased, stiffness index increased in the abdominal aorta of patients with CKD (p=0.007, p=0.002, p=0.004). Patients with CKD had statistically significant over-hydration.

Conclusion: Vascular wall changes that affect the elastic properties of the aortic wall begin to develop in childhood in patients with CKD.

Keywords: Chronic kidney disease, Aortic elasticity, Bioimpedance spectroscopy

1.INTRODUCTION

In chronic kidney disease (CKD), cardiovascular mortality and morbidity is higher than in the normal population due to changes in cardiac structure and function. In patients with CKD, left ventricular hypertrophy, dilatation, and systolic and diastolic dysfunction develop in relation to hypertension and volume overload. Many studies have reported that a decrease in aortic elasticity or an increase in aortic stiffness caused cardiovascular side effects and increased mortality in adult patients with CKD [1,2]. Arterial calcification and aortic stiffness have also been reported to be independent predictors of all-cause and cardiovascular mortality in adult CKD patients [3, 4].

The aim of this study was to evaluate cardiovascular risks and influencing factors by measuring parameters of aortic elasticity and carotid intima media thickness in children with CKD. In addition, the hydration status of patients was evaluated with bioimpedance spectroscopy (BIS) measurements and the effect of hydration status on vascular function was investigated.

2. PATIENTS and METHODS

The study group consisted of patients with CKD who were being followed-up at Pediatric Nephrology Outpatient Clinic. The control group consisted of healthy children without

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cardiovascular disease, who were referred to the pediatric cardiology outpatient clinic due to an innocent murmur or non-cardiac chest pain, and who had no cardiac anomaly on echocardiography. Physical examination findings, weight and height measurements, blood pressure and heart rate of all patients were recorded, and body surface area and age-appropriate height and weight percentiles were calculated. In addition, the duration of kidney failure in the patient group and the duration of dialysis in those who underwent dialysis were also recorded. Blood samples were drawn from patients with CKD and evaluated for complete blood count, kidney function tests, electrolytes, and blood gas analysis. Bioimpedance spectroscopy was applied to both the patient and control groups before echocardiography was performed, and the measurements were recorded.

Echocardiography was performed using a Philips Epic 7C echocardiography machine (Release 2.0.1 Philips Healthcare 3000, Minuteman Road, Andover, MA 01810, USA) equipped with 5.2 and 8.3 MHz transducers. All patients underwent M-Mode, two-dimensional and Doppler echocardiographic examinations, and these measurements were obtained using standard techniques according to the recommendations of the American Society of Echocardiography. All measurements were performed by a single investigator and M-Mode z scores were obtained using Detroit Data [5].

•^aSystolic and diastolic diameters of the aortic annulus, the sinus valsalva, the sinotubular junction, the ascending aorta 3 cm distal to the aortic annulus, the proximal aortic arch between truncus brachiocephalicus and left carotid artery, the descending aorta 1 cm distal to the origin of the left subclavian artery, and the abdominal aorta at the level of the diaphragm were measured with two-dimensional echocardiography. Aortic strain, aortic distensibility, and aortic stiffness index was calculated for each subject using the formulas below:

- Aortic strain = (Systolic dimension-Diastolic dimension) / Diastolic dimension
- Aortic stiffness index = logarithm (Systolic blood pressure/ Diastolic blood pressure) / [(Systolic dimension-Diastolic dimension/Diastolic dimension]
- Aortic distensibility = 2 x (Systolic dimension-Diastolic dimension) / [(Systolic blood pressure – Diastolic blood pressure) x Diastolic diameter]

Z-scores of aortic measurements were obtained using Halifax Data [6].

Flow-mediated dilatation of the right brachial artery was performed using an L11 MHz linear array transducer after resting for 10 minutes in the supine position. The straight segment of the brachial artery was identified in the antecubital fossa and basal measurement was made. A pneumatic cuff on the forearm was then inflated to 50 mmHg above systolic blood pressure for 5 minutes. After the cuff was deflated, the brachial artery diameter was measured every 30 seconds in the end-diastolic phase, every three minutes. Flow-mediated dilatation was calculated as the percent change in diameter from baseline to maximum after the cuff was deflated.

Carotid intima-media thickness was measured from the right carotid artery with the patient in the supine position and neck rotated 45° using a Philips IE33 Echocardiography machine (Philips Medical Systems, Bothell, WA, USA) equipped with an 11 MHz linear array probe. The neck vein was found in a section plane and then the transducer was rotated clockwise to a longitudinal plane. Carotid intima-media thickness was measured on the distal wall of the carotid artery. Carotid intima-media thickness was the distance between the two bright lines measured end-to-end. In the electrocardiogram, images of the end-diastolic phase were taken simultaneously with the end of the R wave. Three different measurements were made, and the average value was used.

Measurement of body composition

Bioimpedance spectroscopy was performed with the Body Composition Monitor (BCM) (Fresenius Medical Care GmbH, Bad Homburg, Deutschland). Electrodes were placed on the right hand and foot with the patient in a supine position. All measurements were computed automatically after the patient's height, weight, age, gender and blood pressure data were entered into the monitor. Bioimpedance spectroscopy was applied to dialysis patients before dialysis, non-dialysis patients and control group before echocardiography and other evaluations.

The study was approved by the Marmara University, School of Medicine, Clinical Research Ethics Committee. (date: 4.9.2020, approval number: 1015). All the patients or parents gave written consent for participating in the research.

Statistical Analysis

Statistical analysis was performed using SPSS for Windows, version 25.0 (IBM Corp., Armonk, NY, United States of America). Data are presented as mean ± SD for continuous variables and median (range) for non-continuous variables. For comparisons between groups, t-test, $\chi 2$ test, and Mann–Whitney U were used, as appropriate. Correlation between variables was expressed using the Spearman rank correlation coefficient. A p-value <0.05 was considered statistically significant.

3.RESULTS

Patient characteristics

The study group consisted of 38 patients (16 girls, 22 boys) with CKD, between the ages of 6-20 years, with a mean ± SD of 13.3 ± 3.7 (median 14.2) years. The control group consisted of 31 healthy children (16 girls, 15 boys) between the ages of 7-16.5 years, with a mean ± SD of 12.1± 2.92 (median 12.1) years. Duration after diagnosis of CKD in patients ranged between 0.25-17 years (mean ± SD= 5.5 ± 4.05; median: 5 years). Eighteen (47.4%) of the patients with CKD were under renal replacement therapy, nine of whom were on peritoneal dialysis and nine on hemodialysis. The mean±SD duration of dialysis treatment in these patients was 3.5± 2.3 years with a median of 3.25 years.

When the control group and patients with CKD were compared in terms of age, weight, height, and body surface area there

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was no statistically significant difference (Table I). BMI was significantly less in patients compared to the control group (p=0.039). There was no significant difference in terms of systolic blood pressure while the diastolic blood pressure of patients with CKD was higher than the control group (p=0.039).

Table I. *Clinical features of patients*

	Chronic kidney disease(n:38) (mean ± SD)	Control (n:31) (mean ± SD)	P
Age (years)	13.3 ± 3.7	12.1 ± 2.9	0.131
Weight (kg)	39.2 ± 17.3	48.6 ± 22.2	0.061
Height (cm)	141.0± 22.8	148.4± 19.2	0.150
BSA (m^2)	1.2 ± 0.3	1.3± 0.3	0.075
BMI (kg/m^2)	18.6 ± 3.1	20.9 ± 6.3	**0.039**
SBP (mmHg)	117.1± 15.1	112.9± 11.7	0.193
DBP (mmHg)	74.6 ± 12.1	69.0 ± 9.9	**0.039**

BSA:Body surface area, BMI: Body mass index, SBP: Systolic blood pressure, DBP:Diastolic blood pressure

Echocardiographic features of patients

When both groups were compared in terms of echocardiographic parameters, mitral A (p=0.013) and pulmonary velocity (p=0.047), interventricular septum and left atrial z scores (p=0.002 and p=0.013, respectively) were found to be higher in patients with CKD. There was no significant difference between the two groups in terms of isovolumetric relaxation time (IVRT), deceleration time (DT), left ventricular end-diastolic diameter (LVDd), left ventricular end-systolic diameter (LVDs), left ventricular posterior wall (LVPW), ejection fraction (EF), fractional shortening (FS), or aortic velocity (Table II).

Table II. *Echocardiographic features of patients*

	Chronic kidney disease(n:38) (mean ± SD)	Control (n:31) (mean ± SD)	P
Mitral E (m/sn)	1.0 ± 0.2	1.0 ± 0.1	0.394
Mitral A (m/sn)	0.70± 0.2	0.5 ± 0.1	**0.013**
Mitral E/A	1.5 ± 0.3	1.7 ± 0.3	**0.010**
IVRT (msn)	69.0± 19.1	62.0 ± 13.3	0.081
DT (msn)	125.8± 68.7	144.4 ± 33.4	0.172
IVSd (z scores)	1.66± 1.1	0,8 ± 0.8	**0.002**
LVDd (z scores)	– 0.3 ± 0.9	– 0.1 ± 0.9	0.441
LVDs (z scores)	– 0.6 ± 1.0	– 0.4 ± 1.0	0.334
LVPW (z scores)	1.3 ± 1.0	0.8 ± 0.9	0.055
LA (z scores)	1.3 ±1.0	0.7 ± 1.0	**0.013**
EF(%)	72.1 ± 5.4	71.5 ± 5.2	0.640
FS(%)	41.0 ± 4.5	40.6 ± 4.6	0.729
Aort velocity(m/sn)	1.3 ± 0.2	1.3 ± 0.1	0.408
Pulmonary velocity (m/sn)	1.2 ± 0.2	1.1 ± 0.1	**0.047**

IVRT:Isovolumetric relaxation time, DT: Deceleration time, IVSd: Interventricular septum diastolic diameter, LVDd: Left ventricular diastolic diameter, LVDs: Left ventricular systolic diameter LVPW: Left ventricular posterior wall diameter, LA: Left atrium diameter

Aortic z scores of patients with CKD and control group were compared. Sinus valsalva and sinotubular junction z scores (p=0.009 and p=0.012, respectively) were higher in patients with CKD, while there was no significant difference in ascending, arcus and abdominal aorta diameters (Table III).

Table III*. z scores of aorta*

	Chronic kidney disease(n:38) (mean ± SD)	Control (n:31) (mean ± SD)	P
Anulus	0.1 ± 1.0	0.1 ± 0.8	0.783
Sinus Valsalva	0.6 ± 1.2	– 0.03 ± 0.8	**0.009**
Sinotubuler junction	0.8 ± 1.05	0.2 ± 0.9	**0.001**
Ascending aorta	1.8 ± 1.3	1.02 ± 0.9	0.066
Arcus aorta	0.3 ± 1.1	0.35 ± 1.0	0.766
Abdominal aorta	– 0.32± 1.3	– 0.2 ± 0.9	0.739

Elasticity parameters of aorta

Abdominal aortic distensibility and strain decreased, and stiffness index increased in the patients with CKD compared to the control group (p=0.007, p=0.002 and p=0.004, respectively) indicating that the abdominal aorta was less distensible and stiffer in patients with CKD. There was no difference between the two groups in terms of sinutubular junction, ascending aorta and arcus aorta strain, distensibility, and stiffness index (Table IV).

Table IV. *Aortic elasticity parameters*

	Chronic kidney disease(n:38) (mean ± SD)	Control (n:31) (mean ± SD)	P
Sinus Valsalva			
SI	3.4 ± 0.6	3.60± 0.9	0.350
DIS	5.5 ± 4.1	6.7 ± 11.5	0.568
Strain	6.34± 4.1	7.42 ± 11,71	0.628
Sinutubuler junction			
SI	3.1± 0.8	3.1 ± 0,8	0.991
DIS	8.6 ± 7.2	8.9 ± 8.4	0.875
Strain	9.7 ± 7.1	10.3 ± 9.8	0.784
Ascending aorta			
SI	3.2 ± 0.9	3.3 ± 0,7	0.589
DIS	7.7± 5.9	6.4 ± 5.5	0.329
Strain	9.0 ± 7.0	7.2 ± 6.05	0.253
Arcus aorta			
SI	3.0 ± 0.8	3.1 ± 0.8	0.829
DIS	8.0 ± 5.8	9.3 ± 8.1	0.463
Strain	9.7 ± 6.4	10.4 ± 8.1	0.678
Abdominal aorta			
SI	3.1 ± 0.7	2.6 ± 0.7	**0.004**
DIS	7.60± 6.3	12.2 ± 7.1	**0.007**
Strain	8.6 ± 6.1	14.1 ± 7.6	**0.002**

SI = stiffness index, DIS = distensibility

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Carotid intima-media thickness

There was no statistically significant difference when comparing carotid intima thickness between the patients with CKD and the control group (p=0.490).

Flow-mediated dilatation

Flow-mediated dilatation of the brachial artery was 1.94±0.46 in patients with CKD and 2.30±0.85 in the control group. There was no statistically significant difference between the two groups in terms of flow-mediated dilatation (p=0.490).

Bioimpedance spectroscopy measurements

Patients with CKD had statistically significant over-hydration compared to the control group (p=0.005). The fatty tissue index (p=0.013), fat ratio (p=0.007), and adipose tissue mass (p=0.044) of the patients with CKD were statistically lower than the control group. There was no significant difference between the two groups in terms of other parameters (Table V).

Table V. *Bioimpedance spectroscopy measurements*

	Chronic kidney disease(n:38) (mean ± SD)	Control (n:31) (mean ± SD)	P
Over-hydration (L)	0.6 ± 1.7	– 0.4 ± 1.4	**0.005**
Over-hydration (%)	4.6 ± 11.5	– 1,9 ± 11.0	**0.027**
Urea distribution volume (L)	22.1 ± 9.3	25.9 ± 9.6	0.108
TBW (L)	28.5± 23.5	26.2 ± 9.5	0.596
ECW (L)	9.8 ± 4.0	10.8 ± 3.9	0.309
ICW (L)	13.0 ± 5.6	15.2 ± 5.7	0.127
E/I	0.8± 0.1	0.7 ± 0.05	0.134
LTI (kg/m^2)	14.2 ± 2.4	14.6 ± 2.6	0.469
FTI (kg/m^2)	4.2± 3.0	6.8 ± 5.1	**0.013**
LTM	29.2± 12.7	33.3 ± 11.7	0.180
Fat (kg)	5.7 ± 4.5	11.1 ± 10.4	**0.007**
ATM (kg)	8.9 ± 8.7	15.1 ± 14.2	**0.044**
BCM (kg)	16.8 ± 7.6	19.2± 7.3	0.208

TBW: Total body water, ECW: Extracelluler body water, ICW: Intracelluler body water, E/I: Extracelluler-intracelluler water ratio, LTI: Lean tissue index, FTI: Fatty tissue index, LTM: Lean tissue mass, ATM: Adipose tissue mass, BCM: Body celluler mass

Correlations

Systolic blood pressure positively correlated to total body water (r= 0.366**, p**=0.030), extracellular body water (r= 0.426, p=0.011), intracellular body water (r= 0.404, p=0.016), body mass index (r= 0.560, p=0.016), lean tissue mass (r= 0.370, p=0.029), adipose tissue mass (r= 0.398, p=0.018) and body cellular mass (r= 0.367, p=0.030). No correlation was found between diastolic blood pressure and BIS measurements. In patients with CKD, overhydration was negatively correlated to abdominal aortic stiffness index (SI) (r= – 0.475, p=0.003) and positively correlated with abdominal aorta distensibility (r= 0.363, p=0.027) and strain (r= 0.486, p=0.002).

Diastolic blood pressure positively correlated with interventricular septum diameter (r=0.335, p=0.040), aortic diameter (r=0.291, p=0.015), mitral A velocity (r=0.341, p=0.004) and negatively correlated with abdominal aortic distensibility (r= – 0.255, p=0.015). Additionally, diastolic blood pressure and carotid intima media thickness were positively correlated (r= – 0.386, p=0.017).

In addition, abdominal aorta distensibility negatively correlated with serum urea value (r= – 0.335, p=0.043), and positively correlated with serum calcium levels (r= 0.335, p=0.043), and negatively correlated with serum sodium levels (r= – 0.481, p=0.003).

Ascending aorta SI negatively correlated with HDL (r= – 0.407, p=0.011), LDL (r= – 0.325, p=0.046) and total cholesterol (r= – 0.442, p=0.005).

Ascending aortic distensibility was positively correlated with HDL (r= 0.334, p=0.041), whereas with ascending aortic strain, LDL (r= 0.436, p=0.006) and total cholesterol had positive correlation (r= 0.437, p=0.006).

There was no significant correlation between abdominal aortic stiffness, distensibility and strain with respect to other parameters (Table VI).

Table VI. *Correlation of abdominal aorta stiffness index, distensibility, strain*

	Abdominal aorta SI		Abdominal aorta distensibilty		Abdominal aorta strain	
	r	P	r	P	r	P
Urea	0.228	0.175	**– 0.335**	**0.043**	– 0.319	0.054
Creatinine	0.051	0.762	– 0.066	0.696	– 0.102	0.548
Calcium	– 0.221	0.209	**0.330**	**0.046**	0.298	0.073
Phosphorus	– 0.005	0.974	– 0.107	0.529	– 0.081	0.624
PTH	0.029	0.862	– 0.010	0.954	– 0.038	0.823
SBP	0.111	0.364	0.057	0.640	– 0.089	0.468
DBP	0.070	0.569	– 0.255	0.035	– 0.189	0.121
CKD phase	0.124	0.457	– 0.243	0.142	– 0.208	0.211
CKD duration	– 0.042	0.801	0.008	0.961	0.059	0.724
Dialysis type	– 0.184	0.270	0.115	0.490	0.123	0.461
Dialysis duration	– 0.020	0.993	0.055	0.818	– 0.015	0.951

PTH:Parathormone, SBP: Systolic blood pressure, DBP:Diastolic blood pressure

4. DISCUSSION

Structural arterial changes and accelerated arterial stiffness increase the risk of cardiovascular disease in CKD. Studies in adults have shown that the development of arterial stiffness is associated with increased mortality [1-4, 7-9].

The elastic properties of the great arteries provide the buffering function of the arterial tree. When the buffer capacity of the elastic arteries is reduced, damage to the delicate microcirculatory bed occurs. As arterial stiffness increases, arterial compliance and distensibility decrease. Eventually, increased cardiac afterload causes left ventricular remodeling and hypertrophy. In addition,

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increased arterial stiffness may cause impaired coronary perfusion and myocardial hypoperfusion. As a result, diastolic and systolic left ventricular dysfunction develops [9-14]. In the present study, children with CKD had thicker interventricular septa and larger left atrial diameters, suggesting left ventricular hypertrophy and left ventricular diastolic dysfunction.

Arterial stiffness is the most important parameter that indicates early changes in vascular structures. Studies evaluating the arterial stiffening of patients with CKD were performed in adult patients, and a different technique from ours was used for the measurement of stiffness. Pulse wave velocity measurement from the aorta or its branches has been reported as the gold standard for arterial stiffness [7,9,12]. Therefore, studies in adults have also measured pulse wave velocities of the ascending aorta, aortic arch, common carotid artery, femoral artery, or brachial artery [3,8,14,15]. In the study of Sayın et al., aortic stiffness was calculated by measuring only the diameters of the ascending aorta in systole and diastole [1]. Later, echocardiography was used in the evaluation of aortic elasticity in many studies [16,17]. Arterial stiffness is associated with arterial dilatation and arterial wall hypertrophy [10,18]. For this reason, we hypothesized that dilatation at the level of sinus valsalva and sinotubular junction in children with CKD is a precursor to arterial stiffness. The results of our study suggested that arterial stiffness in CKD begins in childhood and occurs first in the abdominal aorta, and then affects the sinus valsalva and sinotubular junction regions of the aorta. We found that the stiffness index increased, and distensibility and strain decreased in the abdominal aorta in children with CKD. There was no significant difference in the stiffness index, distensibility and strain at the level of sinus valsalva, sinotubular junction, ascending aorta and aortic arch compared to healthy children. However, patients with CKD had higher aorta z scores at the level of sinus valsalva and sinotubular junction.

The mechanism and chronology of the development of arterial stiffness in CKD is not fully understood. Many studies have shown that diabetes, hypertension, obesity and dyslipidemia are risk factors for premature vascular aging in children. Several studies have reported that uremia is an important factor affecting vascular aging. Uremia, mineral bone disease, hypertension, over-hydration, inflammation and oxidative stress all cause significant changes in vascular structure and functions in children with CKD [9-11].

The typical phenotype of uremic arteriopathy is arterial wall hypertrophy, decreased elastin, and arterial calcification. The hallmark of CKD-related arterial disease is vascular calcification in the intima or media layer of the arterial wall [10,11]. Elastic lamellar calcifications and increased calcium content are observed in the arteries of uremic patients. In young patients with CKD, either during predialysis or the dialysis period, medial calcification and accumulation of hydroxyapatite deposits occur within the arterial wall. In addition, hyperphosphatemia also increases osteoblastic activity, induces apoptosis in vascular muscle cells and causes deposition of mineralized apoptotic bodies in the arterial wall [9,10]. There are also significant elastic lamellar calcifications suggesting a potential role of parathyroid hormone [10,15]. In the present study, decreased abdominal aortic elasticity was associated with increased urea level and decreased calcium level in patients with CKD. This suggests that arterial calcification occurs in uremic patients, as reported in previous studies. High urea levels and accumulation of calcium in the vessel wall appear to be the causes of decreased aortic distensibility in pediatric or young adult patients with CKD.

Anecdotally, we had noticed that our patients with CKD had higher diastolic blood pressure, thicker interventricular septum (IVS) and wider left atrium, and high diastolic blood pressure was associated with IVS thickness and left atrial diameter. Furthermore, we found that high diastolic blood pressure was also associated with decreased abdominal aortic distensibility and increased carotid intima-media thickness. Measurement of carotid intima-media thickness in adults is used as a well-defined marker for atherosclerotic status. Adult studies have reported an increase in carotid intima-media thickness in patients with CKD, dialysis patients, and after kidney transplantation [10]. We found no difference in carotid intima-media thickness measurements in pediatric subjects in the present study.

The increase in mitral A velocity and decrease in mitral E/A in our patients with CKD suggested diastolic dysfunction. Left ventricular diastolic dysfunction and left ventricular hypertrophy were also found in previous studies in pediatric CKD patients [8,19-22]. Children with end-stage renal disease and diastolic dysfunction are thought to be at increased risk of ventricular systolic dysfunction, particularly leading to congestive heart failure and premature cardiac death. Arterial stiffening raises myocardial oxygen consumption for a given stroke volume, and ventricular systolic stiffening amplifies this effect. Moreover, arterial stiffening could influence diastole by elevating systolic load to prolong relaxation, compromise filling, and raise end-diastolic pressure. It has been suggested that vascular stiffness tends to increase and may contribute to the pathophysiology of diastolic heart failure [21,23,24].

Bioelectrical impedance analysis is the gold standard method recommended for objective evaluation and monitoring of hydration status [25,26]. Chronic volume overload in CKD patients can induce changes in mechanical forces and lead to changes in the geometry and composition of the vessel walls. Kwan et al., reported that patients with over-hydration were usually asymptomatic, and that bispectral index (BIS) was important in patients on peritoneal dialysis [25]. In this study, over-hydration was associated with high blood pressure and arterial stiffness, and may contribute to increased cardiovascular risk in this group of patients.

In individuals with normal elastic functions, the increased volume causes stretching of the arterial walls, affecting the aortic diameters, resulting in an increase in arterial strain and distensibility and a decrease in the stiffness index. In patients with CKD, on the other hand, elastic functions are impaired and the increased volume increases the pulse pressure without making a difference in aortic diameters due to higher arterial stiffness. This leads to a deterioration in the elastic parameters of the aorta and causes a decrease in arterial strain and distensibility and an increase in the stiffness index [1,25].

The children with CKD in the present study were overhydrated compared to normal children. However, unlike other studies, overhydration did not correlate to diastolic blood pressure, which is related to peripheral vascular resistance rather than volume overload. In the study cohort of patients with CKD, it was observed that, although the abdominal aorta had stiffened, distensibility and strain were decreased. This increased distensibility of the abdominal aorta correlated with over-hydration, indicating a still normal response to increased cardiac output in children and in contrast to earlier reports of studies in adults. This may be due to slowly progressing calcification of vascular wall, which has not reached the degree seen in adult CKD patients.

Conclusion

These results suggest that vascular wall changes effecting the elastic properties of the aortic wall start to develop in childhood in patients with CKD and are related to severity and the hydration status of the patients. Further larger studies are necessary to confirm these findings of vasculopathy in children with CKD. Close monitoring of hydration status in pediatric patients with CKD is also of cardiac importance. We suggest that cardiovascular functions should be monitored and vascular functional parameters should be measured in pediatric patients with CKD in order to recognize cardiovascular complications of CKD early.

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Compliance with Ethical Standards

Ethical approval: The study was approved by the Marmara University, School of Medicine, Clinical Research Ethics Committee. (date: 4.9.2020, approval number: 1015). All the patients and parents gave written consent for participating in the research.

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