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Assessment of Hypertension in Children with Autosomal Dominant Polycystic Kidney Disease; Single-Center Experience

Author(s)

Alper Uygun[1], Hülya Nalçacıoğlu[2], Özlem Aydoğ[2]

Affiliation(s)

[1]Ondokuz Mayıs University Faculty of Medicine, Department of Pediatric, Samsun, Turkey
[2]Ondokuz Mayıs University Faculty of Medicine, Department of Pediatric Nephrology, Samsun, Turkey

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Abstract

Autosomal dominant polycystic kidney disease (ADPKD) is one of the most common hereditary renal cystic diseases. Although its clinical manifestations usually occur in adulthood, hypertension (HT) is known to develop in most patients before the decline in renal function and it is associated with faster progression to end stage kidney disease (ESKD). We investigated ambulatory blood pressure monitoring (ABPM) results of 23 patients with ADPKD, followed up in the Pediatric Nephrology Clinic of Ondokuz Mayıs University Medical Faculty Hospital. Patients' demographic characteristics, laboratory and ultrasonography (US) results, office blood pressure, and ABPM measurements were evaluated. The parameters of gender, age, increased kidney size, proteinuria, glomerular filtration rate (GFR) was compared in hypertensive and non-hypertensive group. Twenty three patients (13 girls, ten boys) with a mean age of 11.94±4.01 (min-max: 4.6-18) years and a female/male ratio of 1.3/1 were examined. Ultrasound revealed increased kidney sizes in 12 patients (52.2%) and multiple cysts in the bilateral kidneys in 20 patients (87%). Mild to moderate proteinuria was detected in 7 patients (30.4%). The HT ratio of patients was 52.2% and 39.1% when assessed with office blood pressure (BP) measurement and ABPM respectively. A non-dipper pattern was established in 14 patients (60.9%). Gender, age, increased kidney size, proteinuria, GFR did not differ significantly between ADPKD patients with and without ambulatory HT. This study shows that nearly half of children with ADPKD have HT by ABPM. BP should be regularly screened by ABPM in all pediatric ADPKD patients.

Keywords: Ambulatory blood pressure monitoring, autosomal dominant polycystic kidney disease, childhood, hypertension, kidney disease

Correspondence: Hülya Nalçacıoğlu, Ondokuz Mayıs University Faculty of Medicine, Department of Pediatric Nephrology, Samsun, Turkey
E-mail: hulyanalcacoglu@hotmail.com **ORCID:** 0000-0002-0686-9714

Introduction

Autosomal dominant polycystic kidney disease (ADPKD) is one of the most common hereditary cystic disease characterized by the progressive development of kidney cysts resulting in kidney enlargement and decline in renal function with developing end-stage kidney disease (ESKD).[1-3] Although ADPKD seems to be a kidney-specific disorder, it is a multisystem hereditary disease with cyst formation in other organs, primarily the liver and pancreas, and associated with cardiovascular abnormalities.[3-5]

Highlights

- Hypertension, ambulatory blood pressure measurement, autosomal dominant polycystic kidney disease.

Early identifying the risk factors which is associated with more rapidly progression to ESKD remains the base of the management of ADPKD. Male sex, *Polycystic kidney disease 1* (*PKD1*) gene, episodes of hematuria, and hypertension (HT) play a vigorous role in the progression of renal disease to ESKD.[2,3,5-7] In children with ADPKD, HT affects 10-35% of children, associated with cardiovascular disease and decline in glomerular filtration rate (GFR) over time.[6-11]

In this study, we investigated the demographic characteristics, clinical findings, and blood pressure (BP) values using office and ambulatory monitoring in children diagnosed with ADPKD.

Material and Method

This single-centre, observational study was conducted at the Pediatric Nephrology Division at Ondokuz Mayıs University. A total of 23 children with an diagnosis of ADPKD who had undergone an ambulatory blood pressure monitoring (ABPM) were recruited. The inclusion criteria were:

- Diagnosis of ADPKD adequate diagnostic criteria would be considered as based on positive family history of ADPKD in a parent combined with the presence of at least two kidney cysts,[10] unknown family history case, a patient with bilaterally enlarged kidneys and innumerable cysts, without other findings to suggest a different cystic disease, most likely has ADPKD,
- ≥2 visits in our pediatric nephrology center,
- Fully completed laboratory and clinical investigation (kidney ultrasound, clinic blood pressure, urinalysis, and serum creatinine).

In total, 23 children were included in the study. The median time of follow-up was 5.6 years (min-max: 2-14).

The Ondokuz Mayıs University Clinical Research Ethics Committee approved this study (27.12.2019 with the decision number OMÜ KAEK 2019/879). Informed consent was obtained from parents and children for patients aged 11-18 years and from parents only for those ten years of age and under.

Demographic data, physical examination findings, laboratory and ultrasonography (US) examinations were accessed from the patient registry system. The presence of parental consanguinity, a family history of cystic kidney disease, and whether undergoing dialysis were reached from the patient files. The US of the abdomen evaluated kidney and liver sizes, parenchyma echogenicity, cyst number, and cyst sizes. Urine collected over 24h for quantitative measurement of protein. If protein excretion was <4 mg/m^2/hour, 4-40 mg/m^2/hour, and >40 mg/m^2/hour, it was considered normal, mild-moderate proteinuria, and nephrotic (massive) proteinuria, respectively. GFR values of the patients were calculated by the 24-hour creatinine clearance method. A mercury sphygmomanometer measured clinic office BP. After the patient had rested for at least 10 minutes in a sitting position, at least three measurement values were obtained at different times with an appropriately sized sleeve attached to the right arm. Calculated mean systolic and diastolic BP values were evaluated based on the 2017 American Academy of Pediatrics guidelines for childhood HT.[12] According to the guideline, HT was defined as systolic and diastolic BP ≥95 the percentile. ABPM was performed on all children with a Physio-Port Up brand device. As long as the machine was attached, our patients were asked to keep a diary and record hours of sleeping, eating, stressing, exercising, and studying. By attaching a cuff to the patient's non-dominant arm, ABPM reports, where measurements were made every 20 minutes during the day and every 30 minutes at night and at least 40 valid measurements were recorded, were evaluated. Daily, night (sleep), and day (wakefulness) mean, minimum and maximum systolic and diastolic BP values, night, and day mean arterial BP values, and daily, night, and day BP loads (Calculation of the BP values above the 95th percentile as percentages of all measurements) were obtained automatically from the computer data. ABPM results were evaluated according to the report, where the daily, day, and night average arterial pressure mean arteriel blood pressure (MABP) measurements were obtained with ABPM out of 1,000 Central European children, updated by American Heart Association in 2014 and published by Anti Müllerian Hormone in 2016.[13,14]

Patients who had daily, day, or night MABP values >95th percentile with ABPM and systolic and/or diastolic BP load >25th percentile were considered ambulatory HT. Patients who had mean arterial BP values <95th percentile with ABPM and systolic and/or diastolic BP load >25th percentile were accepted as prehypertension. In the office measurements, patients with a mean systolic and/or diastolic BP >95th percentile and diagnosed with HT, but those who had mean arterial BP values >95th percentile with ABPM and systolic and/or diastolic BP load <25th percentile were considered to have white coat HT. The 10-15% decline in night (sleep) measurements compared to daytime (wakefulness) measurements with ABPM was considered "dipper pattern," less than 10% decline "non-dipper pattern," and more than 20% decline "forward-dipper pattern."[13]

Statistical Analysis

Analyses were done using Statistical Package for the Social Sciences 22.0 (SPSS IBM Corp, Armonk, New York, USA). The compatibility of variables was

investigated using the analytic (Shapiro-Wilk) test. The characteristics of patients were determined using descriptive statistics. Parameters compatible with normal distribution were defined as mean standard deviations, and parameters that did not fit normal distribution were described as medium and distribution (lower-upper limit). The comparisons of proportions were performed with the chi-square tests. For the comparisons between the groups, the independent samples t-test was used for the parameters with normal distribution, and the Mann-Whitney U-test was used for the parameters with non-normal distribution. P<0.05 is considered to be statistically significant.

Results

Demographic Characteristics

The mean age of the study group was 11.94±4.01 (min-max: 4.6-18), years of which 13 (56.5%) were female, and 10 (43.5%) were male. The mean follow-up period of the patients was 5.67±2.79 (min-max: 2-14) years. The median age at diagnosis was 5.1 (6 months -14.2 years). There was consanguinity between the parents in only one of the 23 patients (4.3%). While a family history of cystic kidney disease was present in 17 patients (73.9%), it was not present in 6 patients (26.1%). The family history was positive on the mother's side in 8 patients, on the father's side in 6, on their siblings in 2, and both on the mother's and father's side in a patient whose parents were relatives. A total of 9 patients (39.1%) in their family histories had at least a relative who developed end stage renal disease (ESRD) and underwent dialysis; these patients had a positive family history on the mother's side in 3 patients and on the father's side in 6 patients. Demographic and clinical features of the patients were shown in **Table 1**.

US Findings

The mean right kidney vertical length of the patients was 107,09±20.9 (min-max: 80-160) mm, mean left kidney vertical length was 109,43±24.09 (min-max: 80-170) mm. Patients' mean renal vertical length standard deviation score (SDS) was calculated as 1.40±2.65 mm for the right kidney and 1.69±2.99 mm for the left kidney. Twelve of the patients (52.2%) had an increase in kidney sizes; 20 (87%) and 3 (13%) had multiple cysts and two or fewer cysts, respectively, in the bilateral kidneys. The diameter of the largest cyst detected in the patients was calculated as 21±14.05 mm. Extrarenal cysts located in the liver were found in only one patient (4.3%); none of the patients had stones in the urinary system. The largest cyst size of 20 patients was determined as 20.65±13.67 mm, while the largest cyst length of 12 patients was 21±14.05 mm (**Table 2**).

Laboratory Findings

The mean total leukocyte count, hemoglobin value, and platelet count were found to be 6844,09±1523,8 (min-max: 4,970-11,190)/mm^3, 13.1±1.43 (min-max: 9.7-16.1) g/dL, and 280,363±64,727 (min-max: 168,000-421,000)/mm^3, respectively. The mean blood urea nitrogen, serum creatinine, GFR, plasma sodium, plasma potassium, serum calcium, serum phosphorus, and serum albumin values of the patients were observed as 12.40±3.97 (min-max: 8.3-22) mg/dL, 0.59±0.2 (min-max: 0.32 -1.16) mg/dL, 126,71±40.94 (min-max: 40.65-196,50) mL/minute/1.73 m², 140,48±2.31 (min-max: 136-146) [miliEkivalan (mEq)/L], 4.41±0.29 (min-max: 3.8-4.9) mEq/L, 9.91±0.34 (min-max: 9.2-10.8) mg/dL, 4.25±0.60 (min-max: 3.26-5.47) mg/dL, and 4.73±0.26 (min-max: 4.2-5.18) gr/dL, respectively. Liver function tests of all patients were detected to be normal. The mean spot urine protein-creatinine ratios of the patients were measured as 0.23±0.50 (min-max: 0.03-1.33); this ratio was greater than 0.2 in only one patient. 24-hour urine was collected in 22 patients, accordingly, daily protein excretion in urine was calculated as the mean 3.22±1.57 (min-max: 0.99-7.32) mg/m²/hour; while 15 patients (65.2%) had no proteinuria, 7 patients (30.4%) had mild proteinuria. Microalbumin excretion in 24-hour urine of the patients was measured as the mean 24.22±34.39 (min-max: 10-124) mg/day; while there was no microalbuminuria in 16 patients (69.9%), microalbuminuria was detected in 6 patients (26.1%) **Table 2** showed the US and laboratory data of the patients.

Table 1.
Demographic and clinical features of the patients with ADPKD

General Features	Value
No of patients (boys/girls)	23 (10/13)
Age, mean ± SD, year	11.94±4.01
Family history, n (%)	17 (73.9)
Affected mother, n (%)	8 (47)
Family history of ESRD, n (%)	9 (39.1%)
Duration of follow-up, mean ± SD, year	5.67±2.79

ADPKD; Autosomal dominant polycystic kidney disease, SD; Standard deviation, ESRD; End stage renal disease

Table 2.
Radiological and laboratory data of the patients with ADPKD

Data	Value
Radiological findings in admission	
Right kidney size, SDS median (min-max)	1.36 (-1.78 -8.65)
Left kidney size, SDS median (min-max)	1.10 (-1.56 -9.92)
Enlarged kidneys, n (%)	12 (52.2)
Renal cyst size, median (min-max) mm	19 (5- 58)
Bilateral renal cyst, n (%)	20 (87)
Bilateral >4 cysts	20 (87)
Urolithiasis/cyst wall calcification	
Laboratory findings in admission mean ± SD	
Hemoglobin (g/dL)	13.1±1.43
Leukocyte, × 10^3/mm^3	6844,09±1523,8
Platelets, × 10^3/mm^3	280.363±64727
Creatinine (mg/dL)	0.59±0.2
GFR, mL/min/1.73 m^2	126,71±40.94
GFR ≥140 mL/min/1.73 m^2, n (%)	9 (39.1)
Proteinuria, n (%)	7 (30.4)

ADPKD; Autosomal dominant polycystic kidney disease, SDS; Standard deviation score, SD; Standard deviation, GFR; Glomerular filtration rate

Office BP Measurements

The mean systolic BP, diastolic BP, and heart rate were found to be 118,83±16.55 millimeters of mercury (mmHg), 80.87±15.88 mmHg, and 101,61±26.02 respectively. Eight patients (34.8%) were considered normotensive, 3 patients (13%) prehypertensive, and 12 patients (52.2%) hypertensive. Of the 12 hypertensive patients, 6 were girls, and 6 were boys; when HT was staged, 5 (41.7%) were classified as stage 1 HT, 7 (58.3%) as stage 2 HT.

Ambulatory BP Measurements

Patients whose more than 90% of the measurements were appropriate were included in the study. **Table 3** summarizes the office and ABPM data. The mean systolic and diastolic BP was 108,48±8.27, 69.22±7 mmHg, respectively. In the daytime measurements, the mean systolic BP was 109,83±7.20, while its minimum and maximum values were 78.70±6.81 and 153,87±15.13 mmHg. Besides, the mean diastolic BP was 71.26±6.54; its minimum and maximum values appeared to be 43.30±5.06 mmHg and 111,30±11.83 mmHg. During night measurements of patients, while the mean systolic BP was 104,09±12.73, its minimum and maximum values were 85.22±12.25 mmHg and 130,13±21.13 mmHg. The mean diastolic BP was 62.22±9.20; its minimum and maximum values were detected 44.87±7.75 mmHg ve 87.30±17.41 mmHg. Considering ABPM measurements; nine patients (39.1%) were diagnosed with ambulatory HT, four patients (17.4%) prehypertension, and one patient (4.3%) white coat HT. Two of 5 patients with stage 1 HT in the office measurement were diagnosed as HT with ABPM, two as prehypertension, and one as white coat HT. Fourteen of the patients (60.9%) had a non-dipper pattern and ten of the patients (43.4%) had isolated nocturnal HT (**Table 3**).

Patients with and without ambulatory HT were compared in terms of gender, age, increase in kidney size, 24-hour urinary protein excretion, kidney size SDS, and GFR values. No statistical difference was detected

Table 3.
Blood pressure measurement data and stage of hypertensive patients with ADPKD

	Data	Value
Office blood pressure	Systolic blood pressure, mean ± SD, mmHg	118,83±16.55
	Diastolic blood pressure, mean ± SD, mmHg	80.87±15.88
	Heart rate mean ± SD, /min	101,61±26.02
	Stage of hypertension, n (%)	15 (65.2)
	Pre-hypertensive	3 (13)
	Hypertensive	12 (52.2)
Ambulatory blood pressure	Daytime systolic BP, mean ± SD, mmHg	109,83±7.20
	Daytime systolic BP, SDS	-0.7 (-2.36 -3)
	Daytime diastolic BP, mean ± SD, mmHg	71.26±6.54
	Daytime diastolic BP, SDS	-0.29 (-1.87 -3.49)
	Nighttime systolic BP, mean ± SD, mmHg	104,09±12.73
	Nighttime systolic BP, SDS	0.51 (-1.72-5.16)
	Nighttime diastolic BP, mean ± SD, mmHg	62.22±9.20
	Nightime diastolic BP, SDS	1.11 (-1.57-5.08)
	Systolic HT, n (%)	4 (17.4)
	Diastolic HT, n (%)	9 (39.1)
	Stage of hypertension, n (%)	
	Pre-hypertensive	4 (17.4)
	Hypertensive	9 (39.1)
	Non-dipper patern	14 (60.9)
	Isolated nocturnal hypertensive	10 (43.4)

ADPKD; Autosomal dominant polycystic kidney disease, SD; Standard deviation, mmHg; Millimeters of mercury, BP; Blood pressure, SDS; Standard deviation score, HT; Hypertension

Table 4.
Comparision of hypertensive and non-hypertensive ADPKD patients

Data	Hypertension present (+)	Hypertension absent (-)	p
Number of patients Sex (female/male)	6/3	7/7	0.43
Age, years	11.03± 4.35	12.53±3.83	0.394
Right kidney size, SDS	2.08 (-1.78-8.65)	0.53 (-1.48-2.62)	0.159
Enlarged kidneys, number of patients	6	6	0.265
24 hour urine protein excretion (mg/m^2/hour)	4.03 ±1.85	2.76±1.23	0.068
GFR, mL/min/1.73 m^2	124.6±47.3	127.7±39.2	0.873

ADPKD; Autosomal dominant polycystic kidney disease, SDS; Standard deviation score, GFR; Glomerular filtration rate

(**Table 4**), also when these two groups compared in terms of 24-hour urinary protein excretion and right kidney size SDS there are no statistical difference was found. (**Figure 1, 2**) In addition to dietary and dynamic aerobic exercise recommendations as conservative treatment, antihypertensive drug therapy was started diagnosed with prehypertension and ambulatory HT. The angiotensin-converting enzyme (ACE) inhibitor was also initiated for antiproteinuric effect in two patients with proteinuria but without HT.

Discussion

Autosomal dominant polycystic kidney disease, characterized by multiple cyst formation in the kidneys, increased kidney sizes, and progression to ESRD over time, mainly in the 5th-6th decade.[1-3] Structural changes, HT, proteinuria about 2-5% start in childhood, ranging from severe neonatal presentation to the incidental finding of kidney cysts on imaging.[2] There are limited reports of predictors of kidney disease progression in ADPKD.[8,15-20]

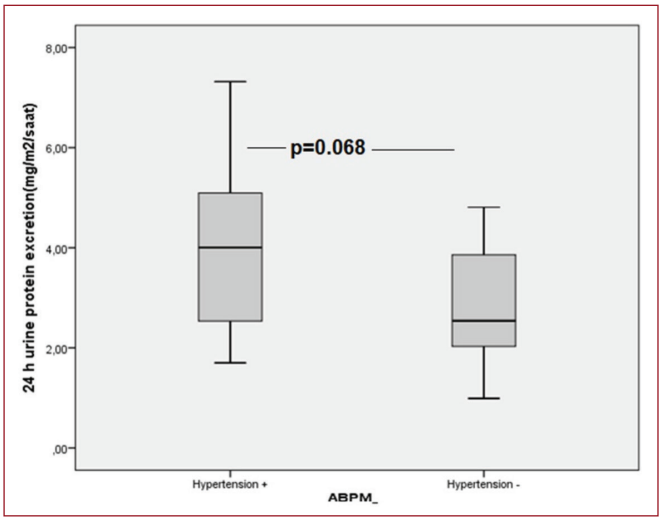

Figure 1. Comparision of 24 hour urine protein excretion in hypertensive and non hypertensive ADPKD patients.

ADPKD; Autosomal dominant polycystic kidney disease

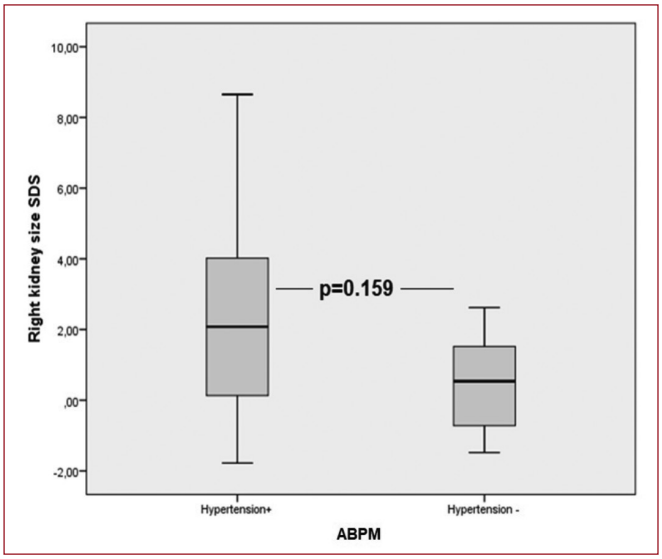

Figure 2. Comparision of right kidney size standart deviation score in hypertensive and non hypertensive ADPKD patients.

ADPKD; Autosomal dominant polycystic kidney disease

We reported 23 pediatric patients with ADPKD investigating the demographic, laboratory, radiological data, with both office and ambulatory BP measurements. Also, we compared the hypertensive /non-hypertensive patients according to the clinical predictors. Based on the studies, age at presentation is associated with the progression of kidney disease.[2,6,15] Early diagnosis of ADPKD (in utero or infancy) are more likely to develop HT, nephromegaly, or decreased GFR.[15] In this study, two patients had an early diagnosis of ADPKD, and both were hypertensive and had nephromegaly. In addition, one patient had a lower GFR; the other had a hyperfiltration of more than 140 mL/min/1.73 m^2. Hyperfiltration is the other precursor or the risk factor for ADPKD progression. In a study, children with glomerular hyperfiltration at baseline have been determined as the underlying cause of faster kidney growth and GFR decline.[20] In the current study, nine patients had glomerular hyperfiltration, HT detected in two of the nine patients.

Proteinuria is the earliest progression marker in both forms of chronic kidney disease. The presence of proteinuria has been associated with the development of HT and more severe kidney cystic disease.[10,15] An international consensus on ADPKD recommended screening proteinuria and treating ACE inhibitors or angiotensin receptor blockers (ARB) if proteinuria is present.[21] In our study, proteinuria was present in almost 30.4% of children with ADPKD similar rate of study by Fick-Brosnahan et al.[19]

HT is one of the early warning signs associated with decreased GFR over time and higher kidney volumes in patients with ADPKD. About 50-80% of adult and 27-37% of pediatric ADPKD patients have HT.[15,22,23] In our cohort, 39.1% of patients have HT, which corresponds well with the studies in children with ADPKD.[8,15,16,24] A survey by Shamshirsaz et al.[15] assessed 199 pediatric ADPKD patients according to age presentation. Forty-six children in the first 18 months were defined as very early-onset (VEO) and 153 children after 18 months as not very early-onset (non-VEO). 43% of VEO cases and 29% of non-VEO patients were found hypertensive. In the VEO group, this frequency increased up to 52% during the follow-up. Seeman et al.[8] reported that ambulatory HT is present in 35% of children with ADPKD. While Tee et al.[24], in their study from Canada, found the prevalence of HT in children with ADPKD to be 46%. Reed et al.[16] has shown that HT is associated with kidney size and the number of kidney cysts in children with ADPKD. We found no significant change in kidney growth or presence of proteinuria in the hypertensive group. ABPM was performed in all children with ADPKD in our center, a more sensitive form of BP measurement. 39.1% of patients have ambulatory HT, of whom 14 patients had a non-dipper pattern, and three had isolated nocturnal HT.

In different studies, HT has been reported to appear in the non-dipper pattern, disrupting the circadian rhythm in ADPKD.[23-25] Nocturnal HT is also prevalent in children with ADPKD. In a study of 310 children, 52% of the patients had non-dipper, and 18% had isolated nocturnal HT.[23] Another study in patients with ADPKD

has also pointed out an association between BP and renal size. Early initiation of HT might relate to bilateral renal ischemia developed secondary to cyst altering the renal vasculature leading to increased kidney size.[8,18,19,22] There is a significant correlation between kidney length and both daytime and nighttime blood pressure.[8] In contrast to studies, we failed to observe any meaningful relationship between HT and renal function or increased kidney size.

Due to potential kidney protective effects, antihypertensive therapy using ACE inhibitors and ARB blockers might retard the progression of ADPKD.[26,27] Another study by Schrier et al.[22] found that close BP control (<90p) was associated with slower kidney growth, improved left ventricular mass index, and lower proteinuria. However, a study in children with hypertensive ADPKD found no effect on kidney growth under the ACE inhibitor over five years. Still, it showed a potential impact on preventing an increase in left ventricular mass index and loss of renal function in patients with borderline HT (75th to 95th percentile).[10] Therefore, as well as the dietary and dynamic aerobic exercise recommendations, the ACE inhibitor was started on 14 patients (60.9%) with pre-HT and HT with ABPM in the current study.

The limitations are mainly the retrospective design, the lack of kidney volume measurement, and the long-term follow-up. On the other hand, the strengths of this study are the relatively large number of patients in a single center, and ABPM was evaluated in all patients. Therefore, we would recommend a longitudinal survey using ABPM in children with ADPKD and follow up the long term of ACE inhibitor on kidney growth and renal survival.

Conclusions

This study has shown that children with ADPKD suffer from HT with a rate of 39.1% by ABPM. Our study results point out HT that starts at younger ages in children with ADPKD. We can conclude that HT should be regularly screened in all pediatric ADPKD patients.

Author Contributions: Uygun A: Constructing the hypothesis or idea of research and/or article, planning methodology to reach the conclusions, organizing, supervising the course of progress and taking the responsibility of the research/study, taking responsibility in patient follow-up, collection of relevant biological materials, data management and reporting, execution of the experiments, taking responsibility in logical interpretation and conclusion of the results, taking responsibility in necessary literature review for the study, taking responsibility in the writing of the whole or important parts of the study, reviewing the article before submission scientifically besides spelling and grammar. Nalçacıoğlu H: Planning methodology to reach the conclusions, taking responsibility in the writing of the whole or important parts of the study. Aydoğ O: Constructing the hypothesis or idea of research and/or article, taking responsibility in logical interpretation and conclusion of the results, reviewing the article before submission scientifically besides spelling and grammar.

Conflict of Interest: On behalf of all authors, the corresponding author states that there is no conflict of interest. This study was presented as an oral presentation in the 64th Turkish National Pediatric Congress, December 2020.

Ethics Committee Approval: The study was conducted in accordance with the Declaration of Helsinki and approved by the Clinical Research Ethics Committee of Ondokuz Mayıs University (27.12.2019 with the decision number 2019/879).

Financial Disclosure: The authors have no conflicts of interest to declare.

Informed Consent: Informed consent was obtained from the parents of the patients.

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