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Original Article

Clinical features of children with multicystic dysplastic kidney

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Abstract ***Background*:** To evaluate the clinical features of patients with multicystic dysplastic kidney (MCDK).

***Methods*:** The medical files of children diagnosed with MCDK between January 2008 and November 2015 were retrospectively reviewed. The demographic, clinical, laboratory and radiological data were evaluated.

***Results*:** Of 128 children with MCDK enrolled in the study, 82 (64.1%) were male, and 46 (35.9%) were female ($P < 0.05$). MCDK were located on left and right sides in 66 (51.6%) and 62 children (48.4%), respectively ($P > 0.05$). Antenatal diagnosis was present in 64 patients (50%). The mean age at diagnosis was 2.8 ± 2.7 years (range, 0–8 years), and follow-up duration was 4.5 years. Fifteen patients (20.8%) had vesicoureteral reflux. Of these, four underwent endoscopic surgical correction. Other associated urological anomalies were ureteropelvic junction obstruction ($n = 6$), hypospadias ($n = 1$), and kidney stones ($n = 1$). On technetium-99 *m* dimercaptosuccinic acid scintigraphy, which was performed in all patients, no significant association between grade of reflux and presence of scarring was seen. Hypertension was diagnosed only in one child (0.8%) who required antihypertensive treatment. The prevalence of unilateral undescended testicle in children aged <1 year in the 82 male patients was 4.9%. Seventy-six patients (59.4%) developed compensatory hypertrophy in the contralateral kidney during a 1 year follow-up period. Of the total, only seven children (5.5%) had undergone nephrectomy.

***Conclusions*:** MCDK follows a benign course with relatively few sequelae, and therefore these patients should be closely followed up and conservatively managed.

Key words morbidity, multicystic dysplastic kidney, renal cystic disease, urinary tract malformation, vesicoureteral reflux.

Multicystic dysplastic kidney (MCDK) is one of the most commonly identified congenital anomalies of the urinary tract. It is a form of renal dysplasia characterized by the presence of multiple, non-communicating cysts of varying size that are separated by dysplastic parenchyma, and the absence of a normal pelvicalyceal system. It is the most common cause of abdominal mass in the neonatal period, and is the most common cystic malformation of the kidney in infancy.[1–3] The incidence ranges from 1 in 1,000 to 4,300 live births. Although the number of diagnoses of MCDK has increased exponentially in the past 20 years due to widespread use of antenatal and postnatal ultrasonography (US), it is usually asymptomatic and could be remained undetected till adulthood.[4,5]

The etiology of MCDK is still unclear, but there are two leading theories for the etiology of MCDK. One is the obstruction theory, which proposes that MCDK results from severe fetal obstructive hydronephrosis, and the other suggests that it is due to abnormal interaction between the ureteric bud and metanephric mesenchyme during renal development.[2,6] Ultrasonography represents the first-line imaging modality for diagnosis and follow up of MCDK in routine practice. Renal scintigraphy provides additional information such as the functioning of renal cortical tissue in the affected kidney, involvement of contralateral kidney, and/or presence of any complicating vesicoureteral reflux (VUR). Therefore, it is suggested to perform technetium-99 *m* dimercaptosuccinic acid (^{99m}Tc DMSA) scintigraphy at least once for each patient with MCDK.[7] ^{99m}Tc mercaptoacetyltriglycine could be replaced with ^{99m}Tc DMSA because it allows better assessment of the contralateral kidney and permits full assessment of any complicating hydronephrosis accompanying MCDK.[8] Voiding cystourethrogram (VCUG) may also be indicated due to the high probability of reflux in the contralateral kidney.[9]

The management of MCDK including nephrectomy has been changed given the low rates of hypertension, infection and malignant transformation.[3,10] A non-surgical approach has become more popular, and is mostly based on spontaneous involution of the dysplastic kidney.[11] Large case series on the clinical features of pediatric MCDK are sparse, hence the aim of this study was to evaluate and describe the clinical features of pediatric MCDK.

Methods

After ethical committee approval was obtained from our institution (08/12/2016-19/01), we retrospectively reviewed the medical records of patients with MCDK in the registry system

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of Firat University Hospital, Elazig, Turkey. A total of 150 children with MCDK were identified between January 2008 and November 2015. Twenty-three patients were excluded from the study because of incomplete medical records and irregular follow-up visits. Hence, the remaining 128 patients diagnosed with MCDK were enrolled in the present study.

The diagnosis of MCDK was made on the US findings of multiple round non-communicating cysts of varying sizes, randomly distributed throughout the kidney with no parenchymal tissue. ^{99m}Tc DMSA scintigraphy was performed in all patients to confirm the diagnosis, obtain additional information about the function of the affected kidney, and investigate the accompanying abnormalities in the contralateral kidney. VCUG was performed in the patients who had US abnormalities in the contralateral kidney (e.g. hydronephrosis or hydroureteronephrosis, increased echogenicity in the renal parenchyma). VUR on VCUG was graded according to the international classification.[12] All patients were followed up by experienced pediatric nephrologists.

The data were analyzed with respect to patient characteristics including gender, age at diagnosis, side of MCDK, renal function tests (serum urea and creatinine), estimated glomerular filtration rate (eGFR) using the Schwartz formula,[13] and the presence of additional anomalies in the ipsilateral and/or contralateral urinary tract, hypertension, recurrent urinary tract infection (UTI), malignancy, undescended testicle in male subjects, additional non-urological anomalies, and also nephrectomy indications in the patients who had undergone nephrectomy, and their final outcome. Creatinine in the antenatally diagnosed patients was measured after 72 h postnatal age. On follow-up visits, control US for urinary system, arterial blood pressure measurement, testing for serum urea and creatinine, and urine analysis were performed. Control visits were made every 6–9 months for the first 5 years, and then every 12–15 months. Prehypertension was defined as systolic and/or diastolic blood pressure (SBP and/or DBP) ≥90th percentile but <95th percentile for age, gender, and height. Hypertension was defined as SBP and/or DBP ≥95th percentile for age, gender, and height, measured on three or more separate occasions.[14]

Statistical analysis

SPSS version 22.0 (IBM, Armonk, NY, USA) was used to analyze the data. Descriptive statistics are presented as *n* (%), parametric data as mean ± SD, and non-parametric data as median (range). Mann–Whitney *U*-test was used for two-group comparison, and the chi-squared test was used for comparison of categorical data. $P < 0.05$ was defined ass statistically significant.

Results

The characteristics of 128 children with MCDK are listed in Table 1. Of the patients, 82 (64.1%) were male and 46 (35.9%) were female (M/F, 1.78; $P < 0.05$). The rate of involvement of the left and right kidneys ($n = 66$, 51.6% vs $n = 62$, 48.4%) in MCDK was not significantly different ($P > 0.05$). A total of 64 patients (50%) had antenatal diagnosis. The remaining patients were diagnosed incidentally on US for UTI or non-urinary problems. The mean age at diagnosis was 2.8 ± 2.7 years (range, 0–8 years): 2.1 ± 1.0 years for girls, and 3.2 ± 1.5 years for boys ($P > 0.05$).

Table 1 Unilateral pediatric MCDK: Subject characteristics

Characteristics	*n*	%	*P*-value
Gender			
Male	82	64.1	<0.05
Female	46	35.9	
Diagnosis time			
Antenatal	64	50	
Postnatal (incidental)	64	50	
Side of MCDK			
Right	62	48.4	>0.05
Left	66	51.6	
VUR			
Ipsilateral/Contralateral	6/7	8.1/9.5	>0.05
Unilateral/bilateral	13/2	17.6/2.7	<0.05
Scar on ^{99m}Tc DMSA	3	2.3	
Recurrent UTI	5	3.9	
Hypertension	1	0.8	
Contralateral compensatory hypertrophy	76	59.4	
Nephrectomy	7	5.5	
Undescended testicle	4	4.9	

^{99m}Tc DMSA, technetium-99*m* dimercaptosuccinic acid; MCDK, multicystic dysplastic kidney; UTI, urinary tract infection; VUR, vesicoureteral reflux.

Follow-up duration was 4.5 years (range, 2–7 years) and mean age at last control visit was 5.8 ± 2.3 years. Hypertension was diagnosed only in one child (0.8%) who required antihypertensive treatment in the absence of reflux, scarring or renal failure. Mean serum creatinine was 0.42 ± 0.24 mg/dL (range, 0.1–0.7 mg/dL) and serum urea was 23.9 ± 10.0 mg/dL (range, 2–40 mg/dL). Mean eGFR was 151.5 ± 56.0 mL/min/1.73 m^2 (range, 56–337 mL/min/1.73 m^2), hence none of the patients had renal failure (the lower range of GFR was measured in newborn babies).

Of the 74 patients who underwent VCUG, VUR was detected in 15 (20.8%). Of these, two patients had bilateral VUR. There were also two cases of suspected high grade ipsilateral reflux/mega-ureter (Fig. 1). The characteristics of VUR in the children with MCDK are listed in Table 2. US at presentation was false negative in one patient who was later diagnosed with right MCDK, and indicative of bilateral MCDK in another patient who later diagnosed with left MCDK, in which both were scarless on ^{99m}Tc DMSA. ^{99m}Tc DMSA was performed in all patients, three of whom had scarring in the contralateral kidney. Two of them each had grade I and III reflux in the contralateral kidney, while the child with grade III reflux had recurrent UTI. No significant association between grade of reflux and scar formation was found. Five children in total (3.9%) had recurrent UTI, only one of whom had grade III VUR in the contralateral kidney and also scarring on ^{99m}Tc DMSA.

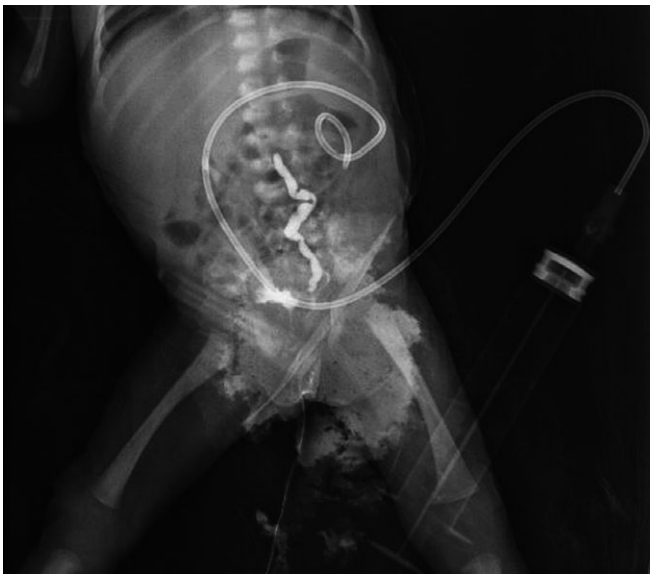

Fig. 1 Voiding cystourethrogram suggestive of high-grade ipsilateral vesicoureteral reflux.

Table 2 VUR in pediatric unilateral MCDK

Side of VUR	*n*	%
Unilateral VUR	13	17.6
Ipsilateral	6	8.1
Grade I	3	4.1
Grade II	1	1.4
High-grade reflux/mega-ureter	2	2.8
Contralateral	7	9.5
Grade I	4	5.4
Grade III	3	4.1
Bilateral VUR	*2*	*2.7*
Grade I	1	1.4
Grade II	1	1.4

MCDK, multicystic dysplastic kidney; VUR, vesicoureteral reflux.

Other associated urologic anomalies were ureteropelvic junction obstruction (UPJO; $n = 6$), hypospadias ($n = 1$), and kidney stones ($n = 1$). Anal atresia and myelomeningocele were the most common non-renal anomalies. The incidence of accompanying non-renal anomalies listed in Table 3. The prevalence of unilateral undescended testicle in the 82 male children was 4.9%. Three of them had right MCDK with additional anomalies: UPJO; anal atresia; and tetralogy of Fallot in one each.

No surgical intervention was performed for VUR in 11 children who had low-grade reflux without complication and no scar formation on ^{99m}Tc DMSA. Two children underwent endoscopic surgical correction for reflux: one of them had grade V VUR, but no scar formation on ^{99m}Tc DMSA. Three children with grade III reflux in the contralateral kidney, complicated by recurrent UTI and multiple scarring on ^{99m}Tc DMSA, underwent surgical intervention for VUR. Of the 128 patients, only seven children (5.5%) had undergone nephrectomy at another center. None of the remaining patients underwent nephrectomy later at the present hospital.

Table 3 Incidence of associated non-renal anomalies in pediatric MCDK

Patient characteristics	*n*	%
Anal atresia	4	3.1
Myelomeningocele	3	2.3
Agenesis of the corpus callosum	2	1.6
Ventricular septal defect	2	1.6
Esophageal atresia and TEF	1	0.8
Inguinal hernia	1	0.8
Congenital deafness	1	0.8
Mental retardation	1	0.8
Pulmonary stenosis	1	0.8
Tetralogy of Fallot	1	0.8
Congenital talipes equinovarus	1	0.8
Congenital hypothyroidism	1	0.8

MCDK, multicystic dysplastic kidney; TEF, tracheoesophageal fistula.

During an average 4.5 years of follow up, it was not possible to obtain any information about the involution rates of the affected kidneys due to insufficient medical records related to US on the control visits. Compensatory hypertrophy in the contralateral kidney was detected in all children: in 76 children (59.4%) this occurred during a 1 year follow-up period. Malignant transformation was not observed in any patient who had not undergone nephrectomy.

Discussion

Multicystic dysplastic kidney is the most common cause of renal cystic disease in children, resulting from the malformation of the kidney during fetal development, in which the kidney consists of many irregular cysts of varying sizes.[1–4] In this study, we have described the clinical features, accompanying additional urological anomalies and long-term outcomes of pediatric MCDK patients who were managed and followed up at the present institution. The frequency of diagnosis of MCDK has increased in the past two decades because of the widespread use of antenatal and postnatal US. Moralıoğlu *et al.* reported a prenatal diagnosis rate of 94.1%,[15] whereas 50% of the present MCDK patient had an antenatal diagnosis. This might be due to the low socioeconomic status and education level of the present patients' parents, who had had inadequate antenatal visits. In addition, the retrospective nature of this study, which was also a limitation of the study, might have been associated with the low rate of antenatal diagnosis. Hence, it is clear that an appropriate pregnancy monitoring program would increase the rate of antenatal diagnosis.

The present study involved 128 children with a male:female ratio of 1.78. A meta-analysis of 14 studies, which reported on 340 patients with unilateral MCDK, noted a male:female ratio of 1.48.[16] Although left-predominant MCDK has been previously reported in the literature,[17,18] left- and right-side involvement in the present study were similar (51.6% vs 48.4% in the left and right kidneys, respectively).

Renal US is recommended as a preliminary diagnostic imaging modality,[19] and will show a kidney containing multiple cysts of variable size that are randomly arranged and separated by little or no echogenic parenchyma.[2,4] In all of the present patients, the diagnosis of unilateral MCDK was made on renal US, except for two patients, in whom the diagnosis was later confirmed on ^{99m}Tc DMSA scintigraphy. Renal scintigraphy is not usually performed in the first 3 months of life, but it would be necessary if US does not show the classic features of multicystic dysplasia of the kidney, as occurred in two of the present patients. Additionally, scintigraphy may be indicated in order to evaluate the function of affected kidney and rule out the probability of accompanying severe UPJO.

Urinary malformations seen in MCDK patients consist of VUR, UPJO, kidney stones and hypospasdias. Contralateral VUR is the most common urological anomaly in MCDK, and ipsilateral VUR might also be present.[2,6,20,21] The rate of VUR in the contralateral kidney ranges between 4.4% and 43%.[2,11,15,22,23] VCUG could be performed to evaluate VUR in MCDK patients, but its use has been increasingly questioned, given that VCUG is an invasive technique, and that VUR is usually low grade and resolves early in life.[20] Two successive normal renal US in infants clinically rule out the significant contralateral anomalies, and thereby VCUG may not be necessary. In the present study, VCUG was performed in 74 patients in total with parent permission, and indicated VUR in 15 patients (20.8%). Of these, two had high-grade ipsilateral reflux. In these patients, reflux did not reach the kidney, and hydronephrosis was not seen on US or VCUG. In addition, scintigraphy showed that these kidneys were not functioning. The contralateral UPJO rate in MCDK patients ranges between 1.1% and 13%.[2,3,15,22,24] Moralıoğlu *et al.* noted contralateral urological anomalies in 10 of 68 patients with MCDK (14.7%).[15] In the present study, the main urological anomalies consisted of contralateral UPJO, kidney stones and hypospasdias, which were present in six (4.7%); one; and one patient, respectively.

Multicystic dysplastic kidney is usually asymptomatic. Abdominal or flank pain and respiratory distress are uncommon symptoms because of the pressure effect of the abnormal kidney.[17] None of the present patients, however, had abdominal or flank pain. The complications of MCDK include UTI, hypertension and renal malignancy. Although the rate of UTI in children with MCDK varies between 5.0% and 34.7%,[15,17,22–24] only 3.9% of the present patients developed UTI, which was not higher than the overall incidence of pediatric UTI.[25] In the present study, there was no difference in the incidence of UTI or of renal scarring in the contralateral kidney according to VUR status, which is consistent with the literature.[20,26] It is recommended that children with MCDK and contralateral VUR should receive antibiotic prophylaxis during infancy and early childhood in order to avoid the risk for scarring secondary to pyelonephritis. In the present study, however, six children who had low-grade reflux without any complication of scar formation on ^{99m}Tc DMSA did not receive prophylaxis. Three children, however, were given antibiotic prophylaxis: one of them had grade V reflux, and the other two had grade III VUR in the contralateral kidney complicated by recurrent UTI, and with accompanying multiple scars on ^{99m}Tc DMSA.

In MCDK patients, blood pressure should be measured on an annual basis, given that hypertension risk in MCDK ranges from 0% to 17.7%.[27–30] The Multicystic Kidney Registry reported mild hypertension in four of 260 individuals with MCDK (1.5%).[23] Radiologically, most MCDK have no demonstrable blood flow. A non-functional bloodless kidney is an unlikely cause of hypertension in infancy and early childhood. The presence, however, of any accompanying congenital urinary abnormality such as UPJO or renal dysplasia and development of a pyelonephritic scar secondary to VUR, or the effects of hyperfiltration over time in the contralateral kidney, is a potential causes of hypertension.[17] In the present study, hypertension was diagnosed only in one child (0.8%) who required antihypertensive treatment, but that girl had no reflux, scarring or renal failure. Therefore, blood pressure should be checked at least once each year in the case of MCDK, and if hypertension is detected it should be treated and closely followed up.

In MCDK the stroma might not completely involute, and might provide a focus for malignant transformation. Because, however, of the retrospective nature of the present study, first kidney size on initial US could not be obtained in all patients, therefore the MCDK involution rate could not be calculated on Kaplan–Meier analysis. Malignancy associated with MCDK has been reported.[31] Determination of renal malignancy in children with MCDK is usually based on US. Individuals with MCDK should undergo renal US every 6–12 months until the age of 5 years or until involution is noted. An earlier study by the American Multicystic Kidney Disease Registry found no cases of renal neoplasia in 260 patients with MCDK.[23] Recently, Eickmeyer *et al.* reported on 301 patients with MCDK over a 30 year follow-up period, and no malignancy was identified in their cohort.[30] Similarly, during approximately 4.5 years of follow up, no renal malignancy was detected in the present series.

Nephrectomy for MCDK is a controversial issue. Prior to modern US, nephrectomy was often required to establish diagnosis, and also it was the standard treatment to avoid complications of UTI, abdominal and flank pain, hypertension, and probable malignant transformation. In recent years, treatment options have been modified to a non-invasive, conservative approach.[11] In the present series, only seven children (5.5%) had undergone nephrectomy before admission to the present hospital. No patients underwent nephrectomy later at the present clinic. Hence, we supposed that nephrectomy might be performed possibly due to a potential risk of malignant transformation and pressure effect of the affected kidneys on adjacent organs.

The contralateral solitary kidney often undergoes compensatory hypertrophy. In the literature, the rate of compensatory hypertrophy in MCDK patients ranges from 43.9% to 89.8%.[20,22,24,27] Compensatory hypertrophy of the functional

kidney begins *in utero* and continues throughout childhood.[6,11] In the present study, 76 patients (59.4%) had compensatory hypertrophy in the functional kidney during the 1 year follow-up period.

Multicystic dysplastic kidney is the most common cause of renal cystic disease in children. It has a good prognosis with conservative management. Routine VCUG or prophylactic antibiotics may not be required. The development of UTI is infrequent, and commonly does not cause damage in the contralateral kidney; thus renal function generally is well preserved in these cases. In order to avoid the potential development of hypertension or hyperfiltration injury, periodic follow up every 6 months, and then annually, in patients diagnosed with simple MCDK, is judicious.

Disclosure

The authors declare no conflict of interest.

Author contributions

A.K. and Z.P.K. designed the study; A.K. and Z.P.K. collected data; A.K. analyzed data, performed the statistical analysis and drafted the manuscript; M.A., M.K.G. gave technical support and conceptual advice, reviewed the manuscript and supervised the whole study process. All authors read and approved the final manuscript.

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