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GFR measurements and ultrasound findings in 154 children with a congenital solitary functioning kidney

Cecilie Siggaard Jørgensen [a,b,*,1], Ronja Carstensen [a,1], Hanifa Awneh [c], Anne Mette Schmidt Frattari [d], Luise Borch [e,f], Lise Bols Toustrup [d], Søren Hagstrøm [c], Konstantinos Kamperis [a,b], Søren Rittig [a,b], Stephanie Dufek-Kamperis [a,g,**]

[a]Department of Pediatrics and Adolescent Medicine, Aarhus University Hospital, Aarhus, Denmark
[b]Department of Clinical Medicine, Aarhus University, Aarhus, Denmark
[c]Department of Paediatrics and Adolescent Medicine and Steno Diabetes Center North Denmark, Aalborg University Hospital, Aalborg, Denmark
[d]Department of Paediatrics and Adolescent Medicine, Regional Hospital Central Jutland, Viborg, Denmark
[e]Department of Paediatrics and Adolescent Medicine, Gødstrup Hospital, Herning, Denmark
[f]NIDO | Centre for Research and Education, Gødstrup Hospital, Herning, Denmark
[g]Department of Pediatrics and Adolescent Medicine, Randers Regional Hospital, Randers, Denmark

* Correspondence to: Department of Paediatrics and Adolescent Medicine, Aarhus University Hospital Palle Juul-Jensens Boulevard 99, DK-8200 Aarhus N, Denmark. Tel.: +45 61161606

** Correspondence to: Department of Paediatrics and Adolescent Medicine, Aarhus University Hospital Palle Juul-Jensens Boulevard 99, DK-8200 Aarhus N, Denmark. Tel.: +45 61314116
cecisi@rm.dk (C.S. Jørgensen)
ronjcars@rm.dk (R. Carstensen)
h.awneh@rn.dk (H. Awneh)
anscid@rm.dk (A.M.S. Frattari)
luise.borch@rm.dk (L. Borch)
lban@rm.dk (L.B. Toustrup)
soha@rn.dk (S. Hagstrøm)
konskamp@rm.dk (K. Kamperis)
soren.rittig@skejby.rm.dk (S. Rittig)
sdufek@clin.au.dk (S. Dufek-Kamperis)

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Summary

Background
Multicystic dysplastic kidney (MCDK) and unilateral renal agenesis (URA) are the most common reasons for a congenital solitary functioning kidney (SFK). We aimed to assess the presence of abnormalities in the congenital SFK and evaluate kidney function using chrome EDTA (CrEDTA) measurements.

Methods
We retrospectively reviewed the medical records of 154 children with MCDK and URA in the period from 2005 to 2022 to analyze results from ultrasound scans and CrEDTA glomerular filtration rate (GFR) examinations.

Results
Of 154 children with a solitary kidney due to MCDK (62%) or URA (38%), abnormalities on the congenital SFK were found in 13 children (8%). The abnormalities spontaneously resolved in 6 children (46%). The most common abnormality was hydronephrosis. Compensatory hypertrophy was found in 17% of the children within the first 6 months of life. 116 children (90%) had a standard GFR (sdGFR) above 75% of expected for the age. Out of those with a sdGFR below 75% of expected, 3 (23%) had abnormalities in the congenital SFK. There was no difference in sdGFR between children with MCDK and URA.

Conclusions
Our study is the first using CrEDTA for GFR measurements and suggests that most children with a congenital SFK due to MCDK or URA have a kidney function within expected for the age. Compensatory hypertrophy of the SFK is found in a minority of children within the first six months of life, suggesting that this process is developing over time. The prevalence of abnormalities in the SFK seems low, however those with abnormalities (e.g. hydronephrosis) are at higher risk of reduced sdGFR.

[1] Shared first authors.

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Introduction

Congenital anomalies of the kidney and urinary tract (CAKUT) represent one of the most frequent developmental defects in humans (birth prevalence of 1%) and are the cause for approximately 40% of paediatric and 8% of adult end-stage kidney disease worldwide [1]. One important condition is the absence or cystic malformation of one kidney, leading to the picture of a solitary functioning kidney (SFK). Multicystic dysplastic kidney (MCDK) occurs in ~1:1000 to 1:4000 births and unilateral renal agenesis (URA) in ~1:2000 births [2–4]. MCDK is characterized by the presence of multiple, non-communicating cysts varying in size and separated by dysplastic parenchyma, and with absence of a normal pelvocaliceal system and ureter. The latter, unilateral renal agenesis (URA), is defined by unilateral nonformation of the kidney. The cause of these malformations lies within disturbances of very early kidney development, where complex and well-orchestrated interactions between the ureteric bud and the metanephric mesenchyme are necessary to provide normal kidney development. The unilateral absence or cystic malformation of one kidney can usually be seen antenatally or after birth on ultrasound scans. Naturally, the remaining SFK overtakes the overall kidney function and is solely responsible for renal outcome [5].

The clinical relevance of a SFK is influenced by the presence of abnormalities in the SFK, and if these affect the overall kidney function. Several studies have investigated renal outcome and complications in these children [6–8], however, the majority do not focus on congenital SFK (in contrast to acquired solitary functioning kidney) and more importantly use estimated glomerular filtration rate (GFR) for evaluation of kidney function rather than gold standard chrome EDTA GFR measurements [9].

The two largest studies to date are the SOFIA study and the KIMONO study, which included 715 and 223 children with congenital SFK, respectively. The KIMONO study showed that a significant number (26%) had abnormalities of the congenital SFK and 31% had signs of kidney injury [10]. The SOFIA study confirmed that 39% of patients with a congenital SFK have indicators of severe kidney injury at the age of 18, and that CAKUT in the SFK is associated with reduced eGFR [8].

We aimed to study a selected cohort of children with a congenital SFK to investigate abnormalities of the congenital SFK and to evaluate kidney function by using golden standard (CrEDTA) GFR measurements. We further wanted to compare sdGFR between the two cohorts of congenital SFK (MCDK and URA).

Methods and material

We retrospectively reviewed the medical records of children with MCDK or URA treated at four different hospitals in Denmark covering a population of approx. 2 million, during the period 2005 to August 2022. Patients were identified by ICD codes and lists of performed GFR measurements using chrome EDTA. MCDK was defined as the ultrasound finding of a kidney with parenchyma completely substituted by large non-communicating cysts of varying size. URA was defined as the absence of one kidney. Technetium-99m-dimercaptosuccinic acid (DMSA) scintigraphies or MAG3 renographies were used to confirm the diagnosis of MCDK and URA.

Results from available chrome EDTA (CrEDTA) GFR measurements were collected and standardized to body surface area (1.73 m^2) and are expressed as standard GFR throughout the paper (sdGFR). From the clinical patient records, we reviewed findings of the first available renal ultrasound scan (US1). Size and abnormalities of the congenital SFK were noted. Hydronephrosis (HN) was defined as a pelvis anterior-posterior diameter of >12 mm. In patients with abnormalities in the SFK, we collected data on a follow-up scan (US 2).

Compensatory enlargement of the congenital SFK was noted in US1 and defined as a kidney length above the 95th centile of normal kidney length for age according to Han et al. [11]. In those patients, where a follow-up US was available, compensatory enlargement was also noted in this US (US 2).

Data analysis was performed with Excel and SPSS version 23 (IBM Corp). The study received ethical approval according to Danish law.

Results

General data

In total, 154 children (67% males) with congenital SFK were included in the study of which 96 (62%) had MCDK and 58 (38%) URA. The right and the left kidney were equally affected in both groups (50/50% and 50/50%). In eight children an associated syndrome was identified (VACTERL association n = 3, Brachiootorenal syndrome n = 1, Mowat-Wilson syndrome n = 1, DiGeorge syndrome n = 1, 2q13 deletion syndrome n = 1, chr7 deletion n = 1). Table 1 presents demographic and clinical information.

Ultrasound data

Ultrasound findings (US 1) were available in all children (n = 154). Age at US 1 was <3 months in 86 (56%) children, 3–6 months in 27 (18%) children, 6–12 months in 10 (6%) children and >12 months in 31 (20%) children. A follow-up ultrasound was collected in those children where an abnormality of the SFK was found in US 1 (n = 13). In one patient (with a single cyst in the SFK) no follow-up US was available. Time difference between US 1 and follow-up US was between 11 and 22 months.

Abnormalities of the congenital solitary functioning kidney

Information on the congenital SFK was available in all children (n = 154). Abnormalities on the congenital SFK were found in 13 (8%) children on US 1 (in 7 (7%) children with MCDK and 6 (10%) children with URA). Details of these abnormalities are presented in Table 2. Out of those 13 children, the abnormalities resolved in 6 (46%) children

Table 1 Demographic, clinical and laboratory features of 154 children with a solitary functioning kidney.

	MCDK n = 96	URA n = 58	Total n = 154
Sex (male/female), n (%)	62/34 (65%/35%)	41/17 (71%/29%)	103/51 (67%/33%)
Location of affected kidney, n (%)			
Right kidney	48 (50%)	29 (50%)	77 (50%)
Left kidney	48 (50%)	29 (50%)	77 (50%)
Abnormalities of the SFK, n (%)			
US 1 (n = 154)	7 (7%)	6 (10%)	13 (8%)
Compensatory enlargement of the SFK, n (%)			
US 1 (n = 113)			19 (17%)
US 2 (n = 99)			22 (22%)
Associated syndrome, n (%)			8 (5%)
MCDK nephrectomy, n (%)	4 (4%)		4 (4%)

Legend: SFK: solitary functioning kidney, *US 1: Ultrasound scan 1. US 2: Ultrasound scan 2.*

Table 2 Abnormalities of the solitary functioning kidney.

	Number (N = 13)	%
Hydronephrosis (> 12 mm)	7	54%
Single cysts	3	23%
Hydroureter	1	8%
Pelvic kidney	1	8%
Increased echogenicity/dysplasia	1	8%

until the follow-up US (US 2). In 6 (46%) children, the abnormality was still found at a later point (four children with hydronephrosis, one patient with cysts and one patient with pelvic kidney). In one patient with a single cyst in the SFK, no follow-up US was performed.

Size of the congenital solitary functioning kidney

In total, 113 (73%) children had their first ultrasound within the first six months of live. Distribution of kidney length of the congenital SFK is shown in Fig. 1. Only one child had a congenital SFK size under the 5th centile, and 19 (17%) children had a compensatory hypertrophy of the congenital SFK already in the first 6 months of life (defined as kidney length over the 95th centile) [11]. Kidney length of the congenital SFK after the first six months of life was available in 99 children, aged between 6 and 81 months at time

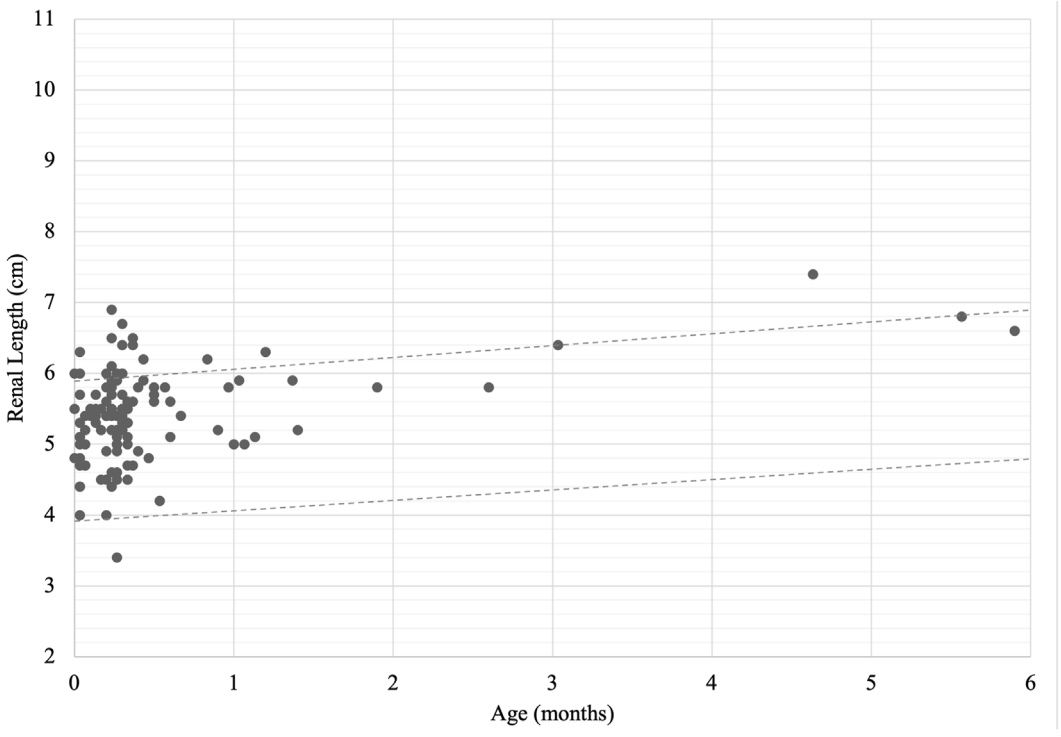

Fig. 1 Length of the congenital solitary functioning kidney during the first 6 months of life. Each dot indicates one patient. The black dotted line indicates the 5th and 95th percentile of normal kidney length [11].

Table 3 sdGFR during infancy for children with a solitary functioning kidney (129 children).

Age group	GFR ≥ 90 (n)	GFR = 60–89 (n)	GFR = 30–59 (n)	GFR ≤ 29 (n)	Mean GFR
<28d (n = 1)			1		32
1–6m (n = 13)	0	6	7	0	61
6–12m (n = 30)	12	17	1	0	88
12–19m (n = 60)	38	21	1	0	93
20m-12yr (n = 25)	19	5	1		97

GFR is displayed as ml/min/1.73^2.
d: days; m: months; yr: years.

of ultrasound. None had a kidney size under the 5th centile and 22 (22%) children had compensatory hypertrophy.

Chrome EDTA standardized GFR measurements

Results of GFR measurements using CrEDTA were available in 129 (84%) children.

Because of varying age at the time of the CrEDTA test, it was not possible to make a general conclusion with 90 ml/min/1.73^2 as a cutoff value defining normality [12]. Therefore, we grouped the patients according to age as suggested by Heilbron, D.C. et al. [13] and display the results in Table 3. Further, we calculated the mean sdGFR for each age group and compared these mean sdGFR values in our study population to average normal sdGFR values published by Heilborn et al. [13]. We found that our population had lower than normal mean sdGFR values.

We further calculated the percentage of measured sdGFR in relation to age-dependent normal mean sdGFR [14]. Results are displayed in Fig. 2. 116 children (90%) had a sdGFR above 75% of expected sdGFR for age. Two children had a sdGFR below 50% of expected for age. One of them had hydronephrosis and parenchymal defects on the DMSA scan of the SFK. The other one had a normal US and DMSA scan. Eleven children (9%) had a sdGFR between the 50% and 75% of expected for age. Two of them had hydronephrosis (with one also having a megaureter needing reimplantation). The remaining nine children had a normal US and no obvious reason for the reduced sdGFR could be found. Overall, three of the 13 children (23%) with a sdGFR below the 75% of expected had abnormalities in the

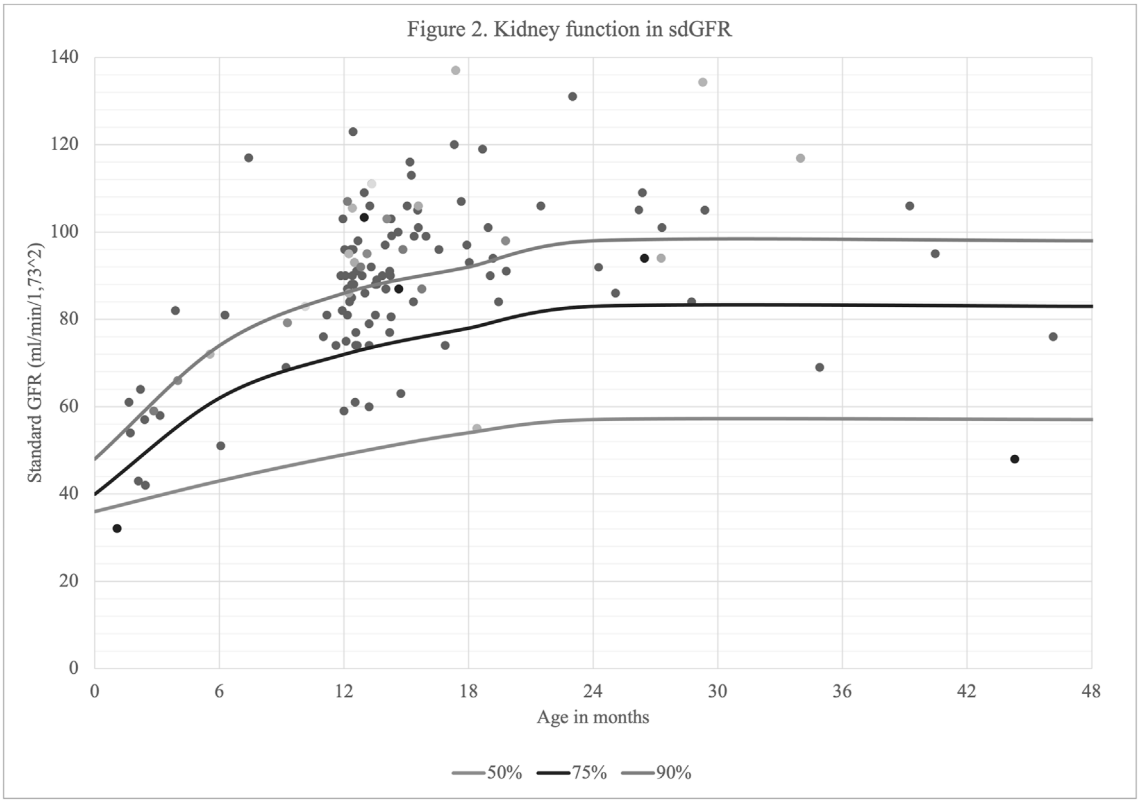

Fig. 2 Chrome EDTA standardized glomerular filtration rate as a function of age for 129 children with a solitary functioning kidney. 90%, 75% and 50% of age-dependent normal mean standard GFR is shown [14]. Four patients are not displayed, because of an age above 48 months at GFR measurement. All four had a sdGFR above 75% of expected.

congenital SFK (hydronephrosis n = 1, hydronephrosis and parenchymal defects n = 1, hydronephrosis and megaureter n = 1). This is nearly the threefold of the overall abnormality rate (8%) found in the congenital SFK in our population.

Comparison of sdGFR between MCDK and URA

To evaluate if there was a difference in the risk for developing impaired kidney function depending on the underlying cause for congenital SFK, we compared sdGFR measurements between children with MCDK and URA. We only included children older than 6 months at time of the GFR measurement, because of the rapid increase in GFR during the first six months of life. Furthermore, we excluded children with a known abnormality on the congenital SFK. We ended up comparing 59 children with MCDK with 33 children with URA. Mean sdGFR was 92 (SD 14.4) vs 93 (SD 17.5) ml/min/1.73^2. There was no significant difference in sdGFR between the two groups ($P = 0.70$).

Discussion

In this study, we describe a large cohort of children with congenital SFK due to MCDK and URA examine abnormalities in the congenital SFK and kidney function using CrEDTA measurements. With 154 children from four centers across Denmark, this is one of the largest studies to date focusing on congenital SFK. In contrast to previous studies, our data suggest that only a low number of children presents with abnormalities in the SFK on US and most of these children have a normal kidney function. Furthermore, we could not find a difference in sdGFR between children with MCDK and URA.

Nowadays, MCDK or URA is often diagnosed prenatally. Consequently, these children receive an early postnatal ultrasound and often scintigraphy/renography to confirm diagnosis and evaluate abnormalities in the congenital SFK [15]. In our study population, only 8% of children had abnormalities on the congenital SFK, with a higher prevalence within children with URA (10% vs 7%). This reflects a lower prevalence than previously reported: The SOFIA study reports that 46% of patients with congenital SFK had any kind of CAKUT in the SFK and 21% had severe CAKUT. However, this study includes not only patients with MCDK and URA as cause for congenital SFK, but also hypodysplasia, PUV, VUR and more. The KIMONO study reports that 26% of children with a congenital SFK had abnormalities on the SFK. The KIMONO study also found a higher prevalence of abnormalities in the SFK in children with URA compared to children with MCDK (20% vs 16%) [6]. La Scola et al. [7] reported an incidence of associated CAKUT abnormalities in 21% of 146 children with congenital SFK. Similar findings were reported by Rudnik-Schöneborn et al. [16], where 27% (n = 130 children) had abnormalities in the SFK, primarily vesicoureteral reflux (VUR) or obstructive changes.

Several studies have indicated that VUR is the most common abnormality seen in children with MCDK with a prevalence varying from 10% to 50% [17–19]. We do not routinely perform VCUG investigations in our centers, which may account for the differences in prevalence of abnormalities in the congenital SFK found in our study compared to previous studies. Otherwise, abnormalities of the congenital SFK visible on US were comparable to what is described in literature, such as hydronephrosis inclusive hydroureter, positional and shape abnormalities, and cysts [20]. Interestingly, at the follow-up US, 46% of these abnormalities had resolved. Only six children had a persistent, significant abnormality of the congenital SFK.

CrEDTA examination is the gold standard to evaluate overall kidney function and calculate GFR [9]. In children with MCDK or URA, the overall GFR reflects the function of the remaining SFK. In our study, most of the children (90%) had a kidney function within the normal range (defined as above 75% of expected for the same age group). However, when comparing sdGFR measurements with a healthy population, the average kidney function for children with a solitary kidney was slightly lower than in healthy children (Table 3). Similar findings are reported in previous studies. La Scola et al. [7] reported a decreased estimated GFR in 12% of these children.

When looking at children with an abnormal GFR in our study, 23% had abnormalities of the SFK. This is nearly threefold of the overall abnormality rate found in our study. The SOFIA study [8], the KIMONO study [6] and La Scola et al. [7] report similar findings, with a higher prevalence of abnormalities on the congenital SFK in those with lower GFR values. Indeed, not surprisingly, children with a normal congenital SFK on ultrasound are more likely to gain full compensatory kidney function. These findings underline the importance of the discovery of abnormalities of the SFK to provide adequate follow-up and counselling of affected children. Nevertheless, in ten children (77%) with a low GFR value, no apparent abnormalities were found. We therefore recommend long-term follow up of all children with abnormalities in the congenital SFK additionally to those with reduced sdGFR values. Previous studies suggested that children with MCDK are at higher risk of developing impaired kidney function [16,21]. In our population, we could not find a difference in sdGFR between children with MCDK and URA when excluding those examined at age<6 months and those with abnormalities in the congenital SFK ($p = 0.695$).

In children with a SFK, compensatory hypertrophy of that kidney may reflect an increased number of nephrons and is associated with a preserved normal GFR-level. We report that 17% of the children had a compensatory enlargement of the congenital solitary kidney already in the first 6 months of life. Gaither et al. [22], demonstrated in 443 children with MCDK, that up to 90% had undergone hypertrophy of the congenital SFK by ten years. Hence compensatory hypertrophy of the congenital SFK appears to be a process that may take several years.

In our study, we are not able to comment on long-term risk of hypertension and proteinuria as the follow-up time was rather short. Westland et al. [6] reported that four out of 62 children (6%) with MCDK developed hypertension during childhood, and Webb et al. [23] concluded that the general risk of developing hypertension due to MCDK is underestimated. Several studies describe that hypertension is seen to cease with treatment of nephrectomy [23–25], however, other studies showed no effect on blood pressure levels after nephrectomy [26]. Whether children with

solitary kidney are at risk of hypertension is still debated, and more research is needed to clarify this. In a recent review of studies published between 1985 and 2014, Cochat et al. [27] concluded lifelong monitoring is necessary, but also that more long-term follow up studies are needed in order to make final recommendations for a follow-up program for children with a solitary kidney. The Italian society of pediatric nephrology has also published guidelines on follow -up [28].

The limitations of this study are the retrospective design and the short follow-up time, which varies between children. Furthermore, VCUG is not performed routinely in our centers. This might mask some children with VUR on the congenital SFK, although the clinical significance of VUR identification in these children is debatable. A strength of this study is that we use CrEDTA which is a more precise method compared to the estimated GFR based on serum creatinine measurements [9]. Further we evaluate the two cohorts (MCDK and URA) separately and compare results.

In conclusion, we report a low prevalence of congenital SFK abnormalities (8%), when evaluating children with congenital SFK due to MCDK and URA. Children with URA are at higher risk of having abnormalities in the congenital SFK. Kidney function for children with a SFK was found to be slightly lower compared to normal average sdGFR values, with no differences between children with MCDK and URA. Only a minority (10%) had a GFR below 75% of expected, with a quarter of those children having abnormalities on the congenital SFK.

These children with abnormalities of the congenital SFK need regular follow up to monitor the overall kidney function. The optimal follow-up regime for children with SFK and normal sdGFR should be evaluated through large follow-up studies.

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Conflicts of interest

The authors declare that there is no conflict of interests regarding the publication of this paper.

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