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Paper Accepted* **ISSN Online 2406-0895**

Original Article / Оригинални рад

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Renal injury in children with a congenital solitary kidney – a single center experience

Повреда бубрега код деце са урођеним солитарним бубрегом – искуство у једном центру

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Received: November 14, 2024
Revised: February 28, 2025
Accepted: March 11, 2025
Online First: March 13, 2025
DOI: https://doi.org/10.2298/SARH241114025G

Paper accepted

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SUMMARY

Introduction /Objective Reduced kidney length, low birth weight, obesity, and ipsilateral congenital anomalies of the kidney and urinary tract (CAKUT) are risk factors for renal injury (hypertension, proteinuria, and chronic kidney disease) in single-functioning kidneys. Our study aimed to investigate the risk factors for renal injury and outcome in children with congenital solitary kidney (CSK).

Methods We collected data from the medical records of 95 children with CSK.

Results Children with CSK were predominantly male (61%). An abnormal ultrasound (US) view of a solitary kidney was found in 9 (9%) and renal length below the 75th percentile in eight (11%) children. Seven (7%) children had low birth weight, 18 (20%) were obese, 26 (28%) had urinary tract infections, 24 (25%) had CAKUT and 28 (29%) were treated with an angiotensin-converting enzyme (ACE) inhibitor. Decreased glomerular filtration rate was found in 3, proteinuria in 14 (15%), and arterial hypertension in 10 (11%) children. 23 (24%) children met the criteria for renal injury. In multiple logistic regression, only US abnormalities approached significance (OR 5.6, p = 0.08). Compared to other studies, we had a higher percentage of an ACE inhibitor prescribed for renal protection. This could be the reason for the low percentage of renal injuries in our study.

Conclusion Monitoring blood pressure, proteinuria, and renal function might be of utmost importance, especially in children with CSK and abnormal US appearance. Additionally, further studies are needed to confirm the possible beneficial effect of renoprotective treatment in patients with CSK.

Keywords: solitary kidney; renal injury; renoprotective treatment; ACE inhibitor; children; abnormal ultrasound appearance

САЖЕТАК

Увод /Циљ Фактори ризика за повреде бубрега (хипертензија, протеинурија и хронична болест бубрега) у једном функционалном бубрегу су смањена дужина бубрега, ниска порођајна тежина, гојазност и ипсилатералне конгениталне аномалије бубрега и уринарног тракта (ЦАКУТ). Циљ наше студије био је да истражимо факторе ризика за повреде бубрега и исход код деце са урођеним солитарним бубрегом (ЦСК).

Методе Прикупили смо податке из медицинске документације 95 деце са ЦСК.

Резултати Деца са ЦСК су претежно мушки (61%). Абнормални ултразвучни (САД) поглед на усамљени бубрег пронађен је код 9 (9%) и дужине бубрега испод 75. перцентила код осморо (11%) деце. Седморо (7%) деце је имало ниску порођајну тежину, 18 (20%) је било гојазно, 26 (28%) је имало инфекције уринарног тракта, 24 (25%) је имало ЦАКУТ и 28 (29%) је лечено инхибитором ангиотензин-конвертујућег ензима (АЦЕ). Смањена брзина гломеруларне филтрације пронађена је у 3, протеинурија у 14 (15%) и артеријска хипертензија код 10 (11%) деце. 23 (24%) деце испунило је критеријуме за повреде бубрега. У вишеструкој логистичкој регресији, само су се америчке абнормалности приближиле значају (ОР 5.6, p = 0.08). У поређењу са другим студијама, имали смо већи проценат АЦЕ инхибитора прописаног за заштиту бубрега. То би могао бити разлог за низак проценат повреда бубрега у нашој студији.

Закључак Праћење крвног притиска, протеинурије и бубрежне функције може бити од највећег значаја, посебно код деце са ЦСК и абнормалним изгледом у САД-у. Поред тога, потребне су даље студије како би се потврдио могући благотворни ефекат ренопротективног третмана код пацијената са ЦСК.

Кључне речи: усамљени бубрег; повреда бубрега; ренопротективни третман; АЦЕ инхибитор; деца, абнормални ултразвук

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INTRODUCTION

Congenital solitary kidney (CSK) is a kidney's anatomical or functional absence from birth. It results from abnormal or incomplete kidney development in utero leading to a non-functioning

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kidney, as in multicystic dysplastic kidney and renal aplasia, or from unilateral renal agenesis [1]. Dysplastic kidneys can regress spontaneously either prenatally or within the first few years of life. Additionally, other congenital anomalies of the kidney and urinary tract (CAKUT), in particular vesicoureteral reflux (VUR), are often associated with CSK [1].

Unilateral renal agenesis is adequately confirmed in most cases with a neonatal abdominal ultrasound (US) performed by a pediatric radiologist. Further imaging with renal scintigraphy with dimercaptosuccinic acid (DMSA) or dimercaptoacetyltriglycine (MAG3) is only recommended if the diagnosis is uncertain (e.g. in the absence of compensatory renal hypertrophy) [2].

In the majority of CSK, there is increased renal growth, which is initiated prenatally. A reduced number of nephrons leads to glomerular hyperfiltration, which results in renal injury, such as high blood pressure, proteinuria, and chronic kidney disease [3]. In a large group of children with a single functioning kidney (SFK), the median age at which renal injury occurred was around 15 years [4]. Risk factors for renal injury in SFK were found to be ipsilateral CAKUT and insufficient kidney length, obesity, and low birth weight [4–6]. The best indicator of renal function in children and adolescents is the glomerular filtration rate (GFR) and as such is used as the most reliable marker of a functioning kidney mass [7]. Angiotensin-converting enzyme (ACE) inhibitors slow the progression of chronic kidney disease in children with renal hypodysplasia [8].

In this study, we sought to evaluate the risk factors for renal injury and outcome in children with SFK compared to other similar studies.

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METHODS

The study is retrospective, conducted at the University Children's Hospital in Ljubljana, Slovenia.

The Patients. We reviewed the medical records of 310 children diagnosed with solitary kidney disease between January 1980 and December 2017. Following the inclusion criteria (e.g. an abdominopelvic US examination without evidence of renal tissue on one side (empty renal fossa, no renal tissue in the retroperitoneum or pelvis) and confirmatory renal scintigraphy) and the exclusion of children with a surgically removed kidney, 95 children were finally included in the study.

We collected the following data: Gender, reasons for first US examination, appearance of kidneys at US examination, measurement of kidney length, duration of follow-up, additional risk factors (low birth weight, obesity, urinary tract infections (UTI), CAKUT (other than CSK)), use of renoprotective medications (ACE inhibitors), GFR, proteinuria, and blood pressure.

Ultrasound. US examinations of the abdomen and pelvis were performed by various examiners with children in the supine position using a 3.5 to 5 MHz probe, usually with the Toshiba US devices (Eccocce, Ecusson, SSA 140A or Power Vision 6000). Compensatory hypertrophy of the CSK was defined in children in the prone position as kidney length in the maximal renal longitudinal section exceeding the 95th percentile value for normal kidney length, as described by Akhavan et al [9]. The US appearance was considered normal if echogenicity, structure, and thickness of the kidney parenchyma were normal, normal corticomedullary differentiation without calyceal dilatation was present and 10 mm was considered the upper limit of normal anterior-posterior renal pelvis diameter [10], otherwise, it was considered abnormal.

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Scintigraphy. Renal scintigraphy with DMSA (57/95; 60%) or MAG3 (38/95; 40%) was performed in all children to confirm CSK and exclude obstruction or parenchymal scarring.

Additional risk factors. Low birth weight is defined as ≤ 2500 g and obesity is diagnosed with a BMI ≥ 95th percentile for age and sex.

CAKUT. Because children with CAKUT had a higher proportion of renal injury on the CSK side [11], we divided the children into those with and without CAKUT. CAKUT was identified by renal US and scintigraphy (performed in all patients) and by a cystourethrogram performed in 56/95 patients (58.9%) when VUR was suspected, mainly in children with recurrent UTI.

Renoprotective treatment. In our study, ACE inhibitor treatment was prescribed not only to children with proteinuria and hypertension, but also to some children with prehypertension, CAKUT, and kidney length below the 75th percentile for renoprotection. Therefore, we did not include the ACE inhibitor as a possible marker of renal injury as was the case in the KIMONO study [4].

Renal injury. Renal injury was defined as the persistent presence of one or more of the following: significantly impaired GFR, proteinuria, and hypertension.

The Schwartz formula with adjustment to the Slovenian population (k= 40 children ≤3 years; k = 48 girls >3 years; k=58 boys >3 years) [12] was used to calculate the GFR. According to Hellerstein [13], the threshold for significantly impaired GFR in solitary kidney was 78 mL/min/1.73m2 in 1–2-year-old children, 73 mL/min/1.73m2 in girls over 2 years and boys 2-13 years, and 70 mL/min/1.73m2 in boys over 13 years. Proteinuria was defined as urinary protein excretion >100 mg/m2/24h or spot urine protein/creatinine ratio > 0.2 g/g Cr [14]. Blood pressure was determined with a 24- or 48-hour ambulatory blood pressure measurement.

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A blood pressure ≥ 95th percentile for sex, age, and height was defined as arterial hypertension [15].

Statistical Analysis

Statistical analysis was performed using the SPSS package for Windows (IBM Corp. Released 2022. IBM SPSS Statistics for Windows, Version 29.0. Armonk, NY). Numeric variables are expressed as median or mean with standard deviation (SD), depending on the distribution of the data. Descriptive variables are expressed as percentages. Multiple logistic regression analysis was performed to determine the predictive risk factors for renal injury. The results are expressed as odds ratios (OR) with 95% confidence intervals (CI). A p-value of less than 0.05 was considered statistically significant.

Ethical Approval

The study was approved by the National Medical Ethics Committee of the Republic of Slovenia (0120-374/2017/7). It complies with the 1964 Helsinki Declaration and its subsequent amendments or comparable ethical standards. Informed consent was obtained from all individual participants included in the study.

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RESULTS

Patient Characteristics

Among the children with CSK, there were more boys (61%), and CSK was predominantly right-sided (56%).

The solitary kidney was detected prenatally in 10/95 (10.5%), immediately after birth during newborn screening in 31/95 (32.6%) children, by US examination due to UTI in 8/95 (8.4%), and by US examination during screening for other congenital malformations, enuresis, urinary incontinence or abdominal pain in 31/95 (32.6%) children. For the remaining 15/95 (15.8%), no data was found at the time of diagnosis.

The last US examination showed an abnormal kidney appearance in 9 (10%) children (Table 1).

CAKUT (other than CSK) was detected in 24 (25%) children, among those more than one anomaly was found in 8/24 (33%). The most common anomaly was VUR, followed by Hydronephrosis and/or hydroureter with or without ureterovesical junction obstruction (Table 2).

Impaired GFR for solitary kidney according to Hellerstein [13] was found in 3 patients. In 14/91 (15%) children proteinuria was present and in 10/95 (11%) arterial hypertension. The mean age of children with a diagnosis of hypertension was 12.4 ± 2 years. Twenty-eight (29%) of the children were treated with an ACE inhibitor for renal injury or for renal protection itself (Table 3). All children with renal injury were recommended to receive treatment with an ACE inhibitor; however, only 9 of them were taking the medication. The mean age at the start of treatment with an ACE inhibitor was 10.2 years (SD 3.2 years).

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Renal injury risk factors

Twenty-three (24%) patients met the criteria for renal injury, defined as significantly impaired GFR and/or the presence of proteinuria and/or hypertension. In the multiple logistic regression analysis (Table 4), an abnormal US appearance is almost significant (OR 5.6).

DISCUSSION

In our retrospective unicentric study, 95 children with CSK were evaluated. The median age at diagnosis was 2 months, with most cases diagnosed either prenatally or immediately after birth, like in the KIMONO study (both 43%) [4]. We had a higher proportion of males like the study by Jorgensen et al (61% vs. 67%) [16]. When considering abnormal kidney appearance, our data is also like the study by Jorgensen et al (10% vs. 8%) [16]. In addition, 25% of our children were found to have CAKUT, mainly VUR, which occurred in 15% of cases. This number is lower than the 24% of patients with VUR in the meta-analysis by Westland et al [1]. Siomou et al [17], on the other hand, report an even lower number of VUR. Since low-grade VUR can be self-limiting, but high-grade VUR may severely impair renal function, it is important to be aware of the latter. As noted in the Westland meta-analysis, these findings highlight the need for further validation to avoid routinely using voiding cystourethrography in children with a normal sonographic appearance of CSK and no recurrent UTIs, especially given the increasing availability of non-invasive methods for detecting VUR [1].

In our study, a significantly higher percentage of children were treated with an ACE inhibitor compared to the KIMONO study (29% vs. 17%, respectively). In most of our children, treatment with an ACE inhibitor was started due to renal protection (19/28 (68%)), whereas in the KIMONO study, the indications for an ACE inhibitor were mostly proteinuria or

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hypertension. The mean age at treatment initiation is approximately the same in both our and the KIMONO study (10.2 years (SD 3.2 years) and 9.8 years (SD 5.5 years), respectively). However, the main difference between these two studies was that the KIMONO study included not only children with CSK but also those with acquired SFK [4].

Impaired GFR, proteinuria, and hypertension were found in 3%, 15%, and 11%, respectively. In comparison, Westland et al. found impaired GFR in 10%, microalbuminuria in 21% and hypertension in 16% in their meta-analysis [1] of more than 2500 unilateral renal agenesis patients. In contrast, in the KIMONO study of more than 400 patients with SFK (congenital and acquired), 4% had impaired GFR, 13% proteinuria, and 22% hypertension [4]. We can speculate that the lower prevalence of hypertension among CSK patients in our study, compared to other studies, may be attributed to the use of ACE inhibitors as renoprotective agents, rather than solely being a consequence of the smaller sample size or the exclusive inclusion of CSK patients in our cohort.

In our study, 23 patients (24%) met the criteria for renal injury, defined as significantly impaired GFR, and/or the presence of proteinuria, and/or hypertension. This is lower than the KIMONO study, where renal injury was observed in 37% of patients [4]. Although in the KIMONO study, ACE inhibitor was used as a criterion for renal injury, we can speculate that the renoprotective use of the ACE inhibitor in our study could reduce the percentage of patients with proteinuria and hypertension.

According to our data, no statistical significance of risk factors for renal injury was found, in contrast to other previous studies that found ipsilateral CAKUT, insufficient kidney length [4], obesity [5], and low birth weight [6] as risks factors for renal injury in SFK. In multiple logistic regression, only the US abnormalities approached significance (OR 5.6, p-value 0.08). The reason for this discrepancy between our study and other studies could be the smaller number

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of patients included in our study compared to other studies [4, 18], although a possible positive effect of an ACE inhibitor as a renoprotective medication cannot be completely excluded, especially in children with prehypertension and US abnormalities. To date, there is limited data on this topic. In 2017, a systematic literature review was conducted, concluding that anti-RAAS drugs (renin-angiotensin-aldosterone system inhibitors) may also provide renoprotective benefits in patients with an SFK. The use of direct renin inhibitors and angiotensin receptor blockers appears to be particularly suitable, especially in children [18]. Additionally, studies in animal models suggest that early administration of ACE inhibitors in cases of SFK is associated with impaired glomerular hyperfiltration-mediated kidney disease [19]. However, further studies are needed to draw more definitive conclusions.

Most studies highlight the importance of lifelong, regular follow-up for CSK patients, focusing on monitoring proteinuria, blood pressure, and kidney function. The KIMONO study recommended at least annual follow-up for children with CSK until adulthood [4, 15]. However, some studies suggest a less intensive follow-up program for children without associated CAKUT and with adequate compensatory renal hypertrophy [3, 4]. Our study demonstrates that patients with CSK and abnormal US appearances require close monitoring.

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Limitations of study

An important obstacle in a retrospective study is missing data, a large difference in follow-up time, the number of outpatient visits, and data that was not collected systematically and longitudinally. Due to the retrospective study, albuminuria was not routinely measured and the results found were far too low to include the parameter in the study. In our study, we used proteinuria as a parameter, even though urinary albumin measurement is recognized as a more specific and sensitive indicator of changes in glomerular permeability compared to total urinary

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protein [20]. On the other hand, the protein-to-creatinine ratio has been demonstrated to be a relevant diagnostic biomarker in clinical trials involving children with glomerular diseases [21].

CONCLUSION

According to our results, a significant percentage of children with CSK and an abnormal US appearance exhibit renal injury compared to those with a normal US. Therefore, close monitoring of blood pressure, protein levels, and kidney function in these children is critically important. Moreover, further research is needed to explore the benefits of renoprotective treatments for individuals with CSK.

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ACKNOWLEDGMENTS

Both authors have made substantial contributions to the article and take full responsibility for its content. All individuals who have contributed significantly to the article are listed as authors.

Funding Sources: No financial or non-financial benefits have been received or will be received from any party directly or indirectly related to the subject of this article.

Conflict of interest: None declared.

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Table 1. Patient characteristics

Characteristic	**No. of patients (%)**
Male gender	58/95(61.1)
Right side CSK	53/95 (55.8)
Abnormal Kidney appearance (last US)	9/93* (9.7)
Kidney length (last US) according to Akhavan	
< 75%	8/74* (10.8)
75–95%	22/74* (29.7)
> 95%	44/74* (59.5)
Low birth weight	7/95 (7.4)
Obesity	18/89* (20.2)
UTIs	26/93* (28.0)
CAKUT	24/95 (25.3)
Extrarenal anomalies	23/95 (24.2)
Median age at first diagnosis, months	2 (2-61) **
Median follow-up, months	106 (3-230) **

CSK – congenital solitary kidney; US – ultrasound; UTI – urinary tract infection; CAKUT – congenital anomalies of the kidney and urinary tract;

*****there are a few missing data results due to retrospective studies; ******range

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Table 2. Congenital anomalies of the kidney and urinary tract (CAKUT)

Parameters	**No. of patients (%)**
CAKUT (total)	24/95 (25.3)
VUR	14/95 (14.7)
Hydronephrosis and/or hydroureter with or without ureterovesical junction obstruction	9/95 (9.5)
Pielon duplex	4/95 (4.2)
Ectopic CSK	2/95 (2.1)
Cystic dysplasia of CSK	1/95 (1.1)
Hypospadias	1/95 (1.1)

VUR – vesicoureteral reflux; CSK – congenital solitary kidney.

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Table 3. Renal injury and the use of ACE inhibitor

Parameters	**No. of patients (%)**
Renal injury (total)	23/95 (24.2)
Impaired GFR according to Hellerstein	3/91* (3.3)
Proteinuria	14/91* (15.4)
Arterial hypertension	10/95 (10.6)
ACE inhibitor (total)	28/95 (29.5)
for Renal injury	9/95 (9.5)
for Renoprotection	19/95 (20)

ACE – angiotensin-converting enzyme; GFR – glomerular filtration rate;
*****there are a few missing data results due to retrospective studies

Paper accepted

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Table 4. Multiple logistic regression analysis of risk factors

Risk Factor	OR (95% CI)	p-value
CAKUT	0.40 (0.08–1.94)	0.254
Low birth weight	0.64 (0.06–5.62)	0.637
UTI	0.50 (0.10–2.43)	0.394
Obesity	1.44 (0.36–5.75)	0.604
Renal length < 95%	1.33 (0.39–4.56)	0.647
Abnormal US appearance	5.57 (0.81–38.04)	0.080

CAKUT – congenital anomalies of the kidney and urinary tract; UTI – urinary tract infection; US – ultrasound

Paper accepted

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