Serum Soluble Urokinase Plasminogen Activator Receptor as a Prognostic Biomarker in Children with Chronic Kidney Disease Due to Congenital Anomalies of the Kidney and Urinary System

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Abstract

Aim: This study aimed to evaluate the relationship between serum soluble urokinase plasminogen activator receptor (suPAR) levels and chronic kidney disease (CKD) stages in children with CAKUT, the leading cause of pediatric CKD.

Methods Forty-two children with CAKUT-related CKD and 42 healthy controls were prospectively enrolled. Serum suPAR levels, estimated glomerular filtration rate (eGFR), and other laboratory parameters were compared. ROC analysis was used to assess the diagnostic performance of suPAR.

Results: Serum soluble urokinase plasminogen activator receptor levels were significantly higher in the patient group ($p<0.001$) and increased with CKD severity. A suPAR cut-off of ≥3.38 ng/mL predicted CKD with 85.7% sensitivity and 78.6% specificity. Serum suPAR showed a negative correlation with eGFR and a positive correlation with creatinine and potassium levels.

Conclusions: Elevated suPAR levels may reflect CKD progression in children with CAKUT and serve as a non-invasive biomarker for early detection and disease monitoring.

***Keywords**: Soluble Urokinase Plasminogen Activator Receptor (suPAR); Child, Chronic Kidney Disease (CKD); Estimated Glomerular Filtration Rate (eGFR); Congenital Kidney and Urinary System Anomalies (CAKUT).*

1. Introduction

Chronic kidney disease (CKD) is defined as the persistence of pathological, laboratory or imaging evidence of kidney damage for 3 months or more, or a decrease in glomerular filtration rate (GFR) below 60 ml/min/1.73 m^2 for 3 months or more, regardless of the presence of kidney damage.[1] The prevalence of CKD worldwide is estimated to be 8-16%.[2] In Türkiye, the incidence in the pediatric population was determined to be 10.9 per million.[3] The etiology of CKD in children differs from that in adults, with congenital anomalies of the kidney and urinary system (CAKUT) being the most common cause in children.[4] CAKUT, less commonly accompanied by hypertension or proteinuria, accounts for approximately 40–50% of CKD cases in children worldwide.[5] While hypertension and proteinuria are key modifiable indicators of CKD progression, they are not present in all cases and often emerge in later stages of the disease. Identifying novel biomarkers that appear earlier could enable earlier intervention, enhance risk stratification for high-risk individuals, and uncover new therapeutic targets, potentially leading to better patient outcomes.

Serum suPAR is a soluble biomarker released into the circulation following the proteolytic cleavage of the cell surface-bound urokinase-type plasminogen activator receptor (uPAR), which is expressed on podocytes, endothelial cells, and immune cells.[6] SuPAR has been identified as an immune-mediated mediator of kidney injury. The urokinase receptor system plays a key regulatory role in the interaction between inflammation, immunity, and coagulation.[7] Studies over the past 10 years have identified suPAR as a unique inflammatory mediator for the kidney. Automatically, it has been shown to activate podocytes in glomerular diseases.[8] It is an independent marker for the incidence and progression of kidney diseases. In pediatric CKD patients, the absence of common adult comorbidities such as hypertension, diabetes, smoking, and aging allows for clearer evaluation of disease-specific biomarkers. Therefore, this study focused on analyzing the link between suPAR concentrations and CKD stages in children with CAKUT, a condition responsible for the majority of pediatric CKD cases.

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2. Materials and Methods

This study was prospective, case-control, single-center on children diagnosed with CAKUT etiology to evaluate the levels of suPAR in children with CKD. The study took place between February 2024 and July 2024 in the Division of Pediatric Nephrology at the Faculty of Medicine at Harran University, Sanliurfa, Türkiye. The study was approved by the Harran University Local Ethics Committee (Approval No: HRÜ/23.24.36; Date: 25 December 2023). The study was conducted in compliance with the Declaration of Helsinki of the World Medical Association. Informed consent was obtained in writing from the legal guardians of all participating children.

This study included pediatric patients aged 1 month to 18 years who were diagnosed with CKD due to CAKUT. Primary anomalies in the number, size and/or morphology of kidneys including unilateral renal agenesis, multicystic dysplastic kidney, horseshoe/ectopic pelvic kidney, renal hypoplasia/dysplasia, ureteropelvic junction stenosis, vesicoureteral reflux, double collecting system and posterior urethral valves, and additional malformations of the urinary tract were accepted as CAKUT categories. Individuals with kidney calcification, neurogenic bladder, multiple birth defects, blood in the urine, current urinary tract infections, a history of extensive bladder reconstruction, dependency on urinary catheterization, or notable heart, lung, digestive, or nervous system disorders were not included in the study. Healthy children, aged and sex-matched with the CAKUT group and without any acute or chronic diseases, particularly with normal blood pressure and renal function, were randomly selected from the medical files of the Pediatric Outpatient Clinics to constitute the Control Group in a 1:1 ratio. Ultimately, the study comprised 42 children diagnosed with CKD secondary to CAKUT, alongside a control group of 42 healthy children matched for age and gender.

For this study, children's demographic such as age and gender, CAKUT type and CKD stages and laboratory test results were collected prospectively. Laboratory tests administered to the children included complete blood count, neutrophil, lymphocyte, platelet, serum creatinine, C-reactive protein (CRP), albumin, sodium, potassium, calcium, phosphorus, blood urea nitrogen tests and urine protein/creatinine ratio. Venous blood and random urine samples were collected from participants in both groups.

Serum suPAR levels were measured using a sandwich-type enzyme-linked immunosorbent assay (ELISA) method. Then, the Microelisa strip plate provided in the kit was pre-coated with antibodies specific to each analyte. Standards or samples were added to the wells, where the analyte was bound to the specific antibody on the plate. Subsequently, a horseradish peroxidase (HRP)-conjugated antibody specific to the analyte was included to each well and incubated. The quantitative analyzes of SuPAR was conducted using specific kits ((Human suPAR ELISA kits (cat. no. E-EL-H2584), produced by Elabscience Biotech Co., Ltd. using Elabscience enzyme-linked immunosorbent assay (ELISA) kits. No financial support or sponsorship was received for the procurement of the kits used in this study. The authors purchased the kits using their own resources.

Additionally, the estimated glomerular filtration rate (eGFR) was calculated for each child according to the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) formula.[9]

Table 1

Distribution of Demographic and Laboratory Measurements in Patient and Control Groups

Variables	All Patients (n=84)	CAKUT (n=42)	Control (n=42)	p
Age (years)	5.00 (2.00-8.00)	4.50 (2.00-8.25)	5.00 (2.00-8.00)	0.709*
Gender (female)	45 (53.6)	23 (54.8)	22 (52.4)	0.827**
Weight (kg)	18.00 (13.00-27.50)	17.50 (12.00-28.00)	20.00 (14.00-26.50)	0.211*
Height (cm)	110.00 (90.00-127.50)	106.00 (88.25-126.25)	110.00 (92.00-128.50)	0.332*
BMI (kg/m^2)	16.20 (15.09-17.72)	15.61 (14.73-17.44)	16.57 (15.37-17.72)	0.128*
GFR (ml/dk)	115.03 (77.69-131.78)	77.95 (52.27-119.77)	122.50 (110.56-141.95)	<0.001*
BUN (mg/dL)	23.54 (18.63-28.70)	24.61 (17.28-34.06)	21.40 (19.26-26.21)	0.335*
Uric acid (mg/dL)	3.70±0.87	3.69±0.93	3.72±0.83	0.893***
Creatinine (mg/dL)	0.42 (0.30-0.75)	0.73 (0.30-0.81)	0.34 (0.28-0.46)	<0.001*
Urine protein/creatinine (mg/mg)	0.65±0.83	0.65±0.83	-	-
Neutrophil (10^3/µL)	3.76 (2.60-5.47)	3.62 (2.31-5.17)	4.09 (2.70-5.97)	0.231*
Lymphocyte (10^3/µL)	4.52 (2.86-6.16)	4.13 (2.66-5.69)	5.13 (3.31-6.45)	0.067*
Thrombocyte (10^3/µL)	353.00 (296.25-403.75)	345.50 (298.75-410.25)	358.00 (279.50-404.50)	0.792*
CRP (mg/dL)	0.05 (0.01-0.11)	0.05 (0.05-0.48)	0.01 (0.01-0.01)	<0.001*
Albumin (mg/dL)	4.50 (4.30-4.60)	4.50 (4.30-4.60)	4.50 (4.30-4.60)	0.272*
Sodium (mEq/L)	138.00 (137.00-140.00)	138.50 (137.00-140.00)	138.00 (137.00-140.00)	0.745*
Potassium (mEq/L)	4.28 (4.10-4.57)	4.40 (4.20-4.70)	4.21 (4.00-4.45)	0.015*
Calcium (mEq/L)	9.80 (9.50-10.00)	9.80 (9.50-10.00)	9.80 (9.50-10.02)	0.812*
Phosphorus (mEq/L)	4.93±0.76	5.03±0.77	4.84±0.75	0.261***
Su-PAR ng/ml	3.65 (1.79-5.08)	5.07 (3.90-6.06)	2.30 (0.60-3.31)	<0.001*

: Mann Whitney U Testi, **: Chi-square Test, *: Independent Groups T Test*

2.1. Statistical analyses

Data were analyzed with SPSS 25.0 package program. In the descriptive analysis, categorical variables were presented as counts (n) and percentages (%), while continuous variables were expressed as mean ± standard deviation or as median with interquartile range (25th–75th percentile, IQR). Categorical data distribution was assessed using the Pearson Chi-square test, while the normality of numerical data was tested with the Shapiro-Wilk test. For comparisons between two independent groups, normally distributed data were analyzed using the t-test, while non-normally distributed data were assessed with the Mann-Whitney U test. The distribution of numerical data across more than two independent groups was analyzed using the Kruskal-Wallis test. For variables showing statistical significance, post-hoc comparisons were performed with the Mann-Whitney U test, applying the Dunn-Bonferroni correction for multiple testing. The association between two non-normally distributed numerical variables was evaluated using Spearman's correlation analysis. In the evaluation of correlation relationships; if r=0.05-0.30, it was accepted as low correlation, if r=0.30-0.40, it was accepted as low-moderate correlation, if r=0.40-0.60, it was accepted as moderate correlation, if r=0.60-0.70, it was accepted as good correlation, if r=0.70-0.75, it was accepted as very good correlation, and if r=0.75-1.00, it was accepted as perfect correlation.

The diagnostic decision-making properties of the parameters in predicting the diagnosis of CAKUT were tested with ROC (Receiver Operating Characteristics) Curve Analysis. The statistical significance level for all tests was accepted as $p<0.05$.

3. Results

The study comprised 42 children diagnosed with CKD secondary to CAKUT, along with 42 age- and sex-matched healthy controls. Table 1 presents the distribution of demographic and laboratory characteristics for the entire study population as well as for the patient and control groups separately. The median age of the patient group was 4.50 years (interquartile range: 2.00–8.25), and 54.8% (n=23) of the patients were female. Age, sex, weight, height, and body mass index (BMI) were comparable between the patient and control groups, with no statistically significant differences were observed ($p>0.05$). Among the laboratory findings, the patient group showed significantly lower eGFR levels compared to controls, whereas serum creatinine, CRP, potassium, and suPAR levels were significantly elevated ($p<0.001$; $p<0.001$; $p<0.001$; $p=0.015$; $p<0.001$). No significant differences were observed in the remaining laboratory parameters ($p>0.05$).

ROC analysis was performed for the suPAR parameter to predict the diagnosis of CKD. It was found that values of 3.38 ng/ml and above for suPAR could predict the diagnosis of CKD with 85.7% sensitivity and 78.6% specificity ($p<0.001$; Area under the curve: 0.895 (0.829-0.961)) (Table 2. Figure 1).

The distribution of suPAR levels in the patient group according to CAKUT type and CKD stages is demonstrated in Table 3. No statistical difference was recorded in the distribution of suPAR according to CAKUT types ($p=0.065$). A statistically significant difference was observed in suPAR levels across different CKD stages ($p<0.001$). Post hoc analysis revealed that this difference was primarily driven by significantly lower suPAR concentrations in stage 1 CKD patients compared to those in stage 3 ($p=0.001$). No statistically significant difference was found between stage 1 and stage 2 patients, suggesting similar suPAR levels in the early stages of CKD. In stage 3, suPAR concentrations were markedly higher compared to stage 1, indicating progression-related elevation. Although an increasing trend in suPAR levels was observed in stages 4 and 5, the limited number of patients in these groups prevented a statistically robust comparison.

The relationship between suPAR parameter and demographic and other laboratory parameters in CKD patients is presented in Table 4. A moderate positive correlation was found between suPAR levels and serum creatinine, while a negative correlation was observed between suPAR levels and GFR ($r=-0.596$; $p<0.001$; $r=0.548$; $p<0.001$). A low-moderately significant positive correlation was recorded between SuPAR and serum potassium level ($r=0.360$; $p=0.019$).

Figure 1

Distribution of Demographic and Laboratory Measurements in Patient and Control Groups

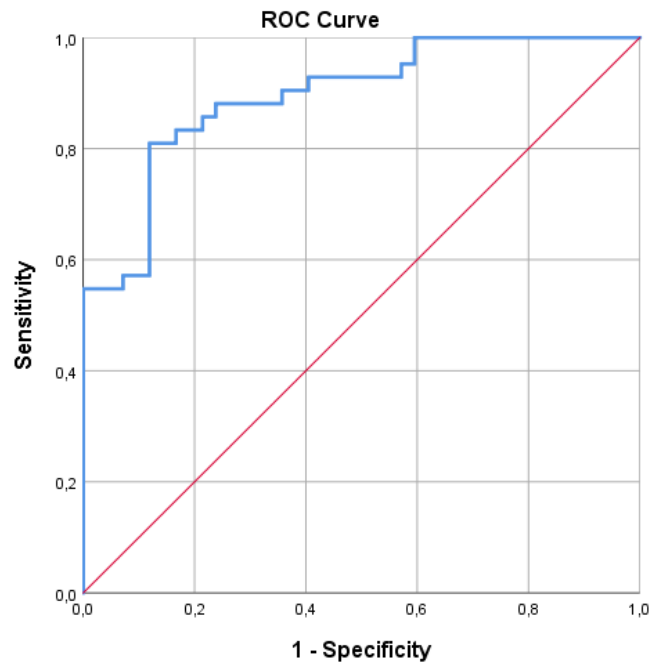

Table 2

ROC Analysis Table for Su-PAR

	Su-PAR
Cut-off value	≥3.38
AUC (95% CI)	0.895 (0.829-0.961)
Sensitivity (95% CI)	85.71 (71.46-94.57)
Specificity (95% CI)	78.57 (63.19-89.70)
PPV (95% CI)	80.00 (68.87-87.85)
NPV (95% CI)	84.62 (72.06-92.14)
Accuracy (95% CI)	82.14 (82.26-89.65)

CI: Confidence Interval, AUC: Area Under the Curve, PPV: Positive Predictive Value, NPV: Negative Predictive Value

Table 3

Distribution of suPAR in the Patient Group according to CAKUT Type and CKD Stages

Variables		suPAR	p	Post -hoc
CAKUT type	Multicystic dysplastic kidney (n=2)	3.30 (2.71-)	0.065*	
	Unilateral renal agenesis (n=6)	6.50 (5.17-8.56)		
	Renal hypoplasia/dysplasia (n=3)	5.69 (4.11-)		
	Posterior urethral valve (n=4)	4.75 (2.36-5.91)		
	Horseshoe kidney (n=6)	5.40 (3.61-8.30)		
	Ureteropelvic junction stenosis (n=5)	5.50 (4.03-6.43)		
	Vesicoureteral reflux (n=13)	4.13 (3.09-5.11)		
	Double collector system (n=3)	6.96 (3.91-)		
CKD stage	Stage-1 CKD (n=17)[a]	4.09 (3.09-4.76)	<0.001*	a-c: 0.001**
	Stage-2 CKD (n=10)[b]	5.02 (3.95-5.58)		
	Stage-3 CKD (n=12)[c]	6.89 (5.29-9.18)		
	Stage-4 CKD (n=2)[d]	7.21 (6.12-)		
	Stage-5 CKD (n=1)[e]	1.73		

**: Kruskal Wallis Test, **: Mann Whitney U Test*

Table 4

Relationship between Water-PAR and Demographic and Laboratory Parameters in the Patient Group

Variables	SuPAR	
	r	p*
Age (years)	-0.049	0.756
Weight (kg)	-0.023	0.887
Height (cm)	-0.048	0.762
BMI (kg/m^2)	0.025	0.876
GFR (ml/dk)	-0.596	<0.001
BUN (mg/dL)	0.043	0.789
Uric acid (mg/dL)	-0.229	0.145
Creatinine (mg/dL)	0.548	<0.001
Urine protein/creatinine (mg/mg)	0.051	0.750
Neutrophil (10^3/µL)	0.222	0.158
Lymphocyte (10^3/µL)	0.014	0.932
Thrombocyte (10^3/µL)	-0.080	0.613
CRP (mg/dL)	-0.069	0.664
Albumin (mg/dL)	-0.083	0.601
Sodium (mEq/L)	0.033	0.834
Potassium (mEq/L)	0.360	0.019
Calcium (mEq/L)	-0.030	0.849
Phosphorus (mEq/L)	0.003	0.985

*r= Spearman correlation coefficient, *: Spearman Correlation Analysis*

4. Discussion

In this study, serum suPAR levels were found to increase progressively with advancing CKD stages. While patients in stages 1 and 2 exhibited relatively similar and lower suPAR concentrations, a significant rise was observed in stage 3, suggesting that suPAR may serve as a marker reflecting the transition from early to moderate CKD. This elevation could be explained by increased glomerular and tubulointerstitial damage as kidney function deteriorates. Although stages 4 and 5 demonstrated a trend toward further elevation in suPAR, the small number of patients in these advanced stages limited the statistical power of the analysis. Nevertheless, these findings highlight the potential role of suPAR as a biomarker that mirrors disease severity and may help predict CKD progression in children with CAKUT.

Serum suPAR is a biomarker of inflammation that contributes to kidney damage via the immune system. Recent studies have highlighted that increased suPAR levels may independently contribute to the progression of CKD.[10] It has also been proposed that suPAR serves as a renal-specific biomarker involved in the pathophysiological link between acute kidney injury and the development of CKD.[11,12]

In our study, it was shown that serum suPAR levels were significantly higher in children diagnosed with CKD due to CAKUT and these levels increased as the CKD stage progressed. Additionally, suPAR levels were found to be inversely associated with GFR, and directly associated with serum creatinine and potassium concentrations. These findings indicate that suPAR may be a potential biomarker in childhood CKD. In recent years, suPAR has been recognized as a biomarker reflecting glomerular and tubulointerstitial damage in adult populations and has been implicated in the advancement of chronic kidney disease.[13,14] However, data on this subject are limited in the childhood age group. Our study contributes to the literature by showing that suPAR levels increase in line with the severity of CKD in pediatric patients.

The suPAR threshold value of 3.38 ng/mL determined as a result of ROC analysis was able to predict the diagnosis of CAKUT with high sensitivity (85.7%) and specificity (78.6%). This finding indicates that suPAR is valuable not only in identifying the presence of disease but also in enhancing diagnostic precision. Previous research has shown that suPAR serves as an indicator of inflammation and immune system activation and may play a role in the development of fibrosis within kidney tissue.[15,16] In 2011, suPAR was initially recognized as a permeability factor involved in the pathogenesis of chronic glomerulonephritis.[17] In the study of Sharma et al.[18], it was shown that sera taken from FSGS patients could cause proteinuria in rats.

In our research, suPAR levels did not significantly vary among the different CAKUT subtypes; however, a notable variation was observed across the various stages of CKD. The significantly higher suPAR levels, especially in stage 3 patients, suggests that this biomarker may also reflect disease progression. In addition, the negative correlation between suPAR and GFR (r=-0.596; p<0.001) is parallel to the relationships previously described in pediatric cohorts.[19] In a separate pediatric cohort, suPAR proved to be a more reliable indicator than eGFR alone for predicting the progression of CKD, particularly among patients with mild to moderate disease (eGFR between 40 and 80 mL/min/1.73 m^2).[20] Elevated suPAR levels could serve as a useful clinical biomarker for risk assessment in patients with stage 3 CKD, which is a critical phase when secondary complications often develop and kidney-protective treatments are most effective.

5. Conclusion

This study demonstrated that children with CKD caused by CAKUT had notably elevated suPAR levels, which rose progressively with advancing stages of CKD. Serum suPAR stands out as a potential biomarker in both disease diagnosis and staging. In addition, its negative correlation with GFR and positive correlation with creatinine and potassium that it may be a reflection of renal dysfunction. In line with these findings, it is thought that suPAR may be a non-invasive and clinically valuable marker that can be used in monitoring CKD progression in pediatric CAKUT patients. Nevertheless, these findings require validation through additional research involving larger sample sizes.

Statement of ethics

This study was carried out in accordance with the ethical principles outlined in the Declaration of Helsinki and the study received approval from the local Ethics Committee of Harran University (Ethic no: HRÜ/23.24.36).

genAI

No artificial intelligence-based tools or generative AI technologies were used in this study. The entire content of the manuscript was originally prepared, reviewed, and approved by both authors.

Funding

This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

Conflict of interest statement

The authors declare that they have no conflict of interest.

Availability of data and materials

This Data and materials are available to the researchers.

Author Contributions

Concept/ A.G., S.T.; Literature Review / A.G., S.T.; Design / A.G., S.T.; Data collection / A.G.

Analysis and interpretation / A.G., S.T.; Writing / A.G., S.T.; Critical review / S.T.

All authors reviewed the results and approved the final version of the manuscript.

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