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Assessment of total kidney volume by computed tomography in autosomal dominant polycystic kidney disease and its correlation with estimated glomerular filtration rate

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ABSTRACT

Background: Autosomal dominant polycystic kidney disease (ADPKD) is characterised by progressive renal cyst enlargement, leading to increased total kidney volume (TKV), declining kidney function, and eventual end-stage renal disease. **Aims and Objectives:** This study aimed to estimate TKV using computed tomography (CT) and assess its correlation with the estimated glomerular filtration rate (eGFR) as a structural marker of disease progression. **Materials and Methods:** This prospective study included 25 patients with ADPKD aged 15–46 years who had preserved renal function. The TKV was measured using contrast-enhanced CT through freehand manual planimetry. Renal function was assessed using serum urea, creatinine, and eGFR (Chronic Kidney Disease Epidemiology Collaboration) at baseline and 12-month follow-up. The association between TKV and eGFR changes was analysed using Pearson's correlation and linear regression. **Results:** At baseline, the mean eGFR was 90.72 mL/min/1.73 m^2, which significantly decreased to 81.52 mL/min/1.73 m^2 after 1 year ($P = 0.001$). The TKV increased significantly from 847.6 mL at baseline to 933.84 mL at 1 year ($P = 0.001$). A moderate positive correlation was observed between the percentage increase in TKV and the percentage reduction in eGFR ($r = 0.53$). Linear regression analysis demonstrated that for each 1% increase in TKV, there was a corresponding 0.89% increase in the percentage reduction in eGFR ($\beta = 0.893$, 95% Confidence interval: 0.27–1.52, $P = 0.007$). **Conclusion:** This study demonstrates a significant association between increasing TKV and decreasing renal function in ADPKD, supporting the use of TKV as a marker of disease progression. CT-based volumetric assessment may aid in the early identification of patients at risk of rapid functional deterioration.

Key words: Autosomal dominant polycystic kidney disease; Total kidney volume; Estimated glomerular filtration rate; Computed tomography; Renal function dysfunction

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INTRODUCTION

Autosomal dominant polycystic kidney disease (ADPKD) is described by the advancement of fluid-filled cysts resulting in progressive kidney volume (KV) magnification regardless of seemingly normal renal function from 40 to 50 years of age.[1] The augmentation in KV, which is associated with renal function deterioration only at a later

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stage of life, is broadly acknowledged as the leading feature of ADPKD development and offers a metric of disease advancement.[2] Total KV (TKV) relates to the advancement of end-stage renal disease (ESRD) and considerably influences the quality of life.[3]

Kidney size is smaller in women; therefore, progression to ESRD occurs later than in men.[4] Patients with polycystic kidney disease 1 (PKD1) have larger kidneys with extra cysts and acquire ESRD earlier than PKD2 patients.[5] These results suggest that TKV predicts the later advancement of renal insufficiency. In ADPKD, the glomerular filtration rate (GFR) remains normal for an extended period before it begins to drop inevitably.[6]

Attempts to intervene at the stage when the GFR begins to decline have largely been unsuccessful, and the rate of GFR reduction appears to be more rapid than in many other kidney disorders.[7] However, even when GFR remains within normal limits, the expansion of renal cysts and kidney enlargement continues to progress. The precise mechanism that triggers the initial decline in GFR remains unclear. Inferences from three new randomized controlled trials have not proven an association between changes in TKV and GFR, suggesting that TKV values are an indicator of ADPKD.[8] Hypertension is commonly observed in patients with ADPKD, even when the GFR remains normal. By the age of 30, nearly 70% of individuals with ADPKD develop elevated blood pressure.

Hypertension is an important determinant of ESRD; however, whether hypertension in ADPKD plays a part in the quicker progression is chiefly an indicator of more severe disease resulting in ESRD, or both, is unspecified. The present study observed that patients with ADPKD with proteinuria have a bigger annual rise in renal volume as well as a bigger annual decrease in GFR than those without proteinuria.[9] Associations between renal enlargement, hypertension, sex, proteinuria, and renal dysfunction are multifaceted and have not been scrutinized in a large longitudinal study.

TKV can be computed on either magnetic resonance imaging (MRI) or computed tomography (CT) with a technique that varies in difficulty, time required, accuracy, and meticulousness. The most frequently used methods comprise the entire kidney. As these methods are quicker, a mid-slice approach or an ellipsoid equation has been projected to assess TKV. Previous studies evaluated the strength of a single or a couple of TKV evaluation methods in comparison with either manual planimetry or stereology.[10]

Building on this background, the present study aimed to evaluate the potential association between TKV and estimated GFR in individuals diagnosed with ADPKD.

Aims and objectives

To estimate TKV using CT-based freehand manual tracing planimetry and assess its association with eGFR in patients with ADPKD.

MATERIALS AND METHODS

This prospective study enrolled 25 patients with a confirmed diagnosis of ADPKD, conducted at the Barnard Institute of Radiology, Madras Medical College, and Rajiv Gandhi Government General Hospital, Chennai, between March 2019 and September 2020. Ethical approval was secured from the institutional ethics committee, and written informed consent was obtained from all participants before inclusion in the study.

Inclusion criteria

Patients between 15 and 46 years of age with a diagnosis of ADPKD and preserved renal function, defined as a creatinine clearance of ≥70 mL/min and serum creatinine levels ≤1.6 mg/dL in males and ≤1.4 mg/dL in females, were included in the study.

Exclusion criteria

Patients were excluded if they had other medical conditions besides hypertension that could affect renal function (e.g., diabetes mellitus).

Methods

The sample size (n=25) was calculated using a 95% confidence level and an expected proportion of 93% based on previous Indian studies. Using the normal approximation for the binomial distribution, the required sample size was estimated to be 22, and 25 participants were included to account for potential dropouts.

Data collection included baseline demographic and clinical details, TKV assessment using contrast-enhanced CT, and renal function evaluation. TKV was calculated by summing the cross-sectional areas of the outlined kidney slices multiplied by the slice thickness, while eGFR was derived using the CKD-EPI equation.

All imaging was conducted by blinded radiologists using standardised protocols, and the measurements were reviewed and averaged for accuracy. Follow-up assessments at 12 months included repeat CT for TKV and laboratory evaluation of serum urea, creatinine, and eGFR. The percentage change in TKV was correlated with changes in eGFR to assess the disease progression.

Statistical analysis

Descriptive statistics were expressed as mean±standard deviation for continuous variables and as frequencies with percentages for categorical data. Independent sample t-tests were used to compare continuous variables between groups, while paired t-tests assessed changes in TKV and estimated GFR from baseline to the 12-month follow-up. The relationship between TKV and eGFR was analysed using Pearson's correlation coefficient. A two-tailed P=<0.05 was considered statistically significant. All statistical analyses were conducted using the IBM Statistical Package for the Social Sciences Statistics for Windows, version 25.0.

RESULTS

Among the patients, the highest proportion of males was in the 41–45 years age group (70%), followed by equal gender distribution in the 36–40 years group (50% males), and the lowest in those aged <35 years (42.9%).

At baseline, the mean urea level was 29.16 mg/dL, which significantly increased to 31.8 mg/dL at 1 year (P=0.030). The mean serum creatinine increased from 0.95 to 1.07 mg/dL (P=0.001), whereas the mean eGFR significantly decreased from 90.72 to 81.52 mL/min/1.73 m^2 (P=0.001). In addition, the mean TKV significantly increased from 847.6 mm^3 to 933.84 mm^3 over the same period (P=0.001) (Table 1).

The correlation coefficient (r) was 0.527, indicating a moderate, positive correlation. Linear regression analysis demonstrated that each unit increase in the % of KV was associated with a 0.89-unit increase in the % decrease in eGFR (β=0.893; 95% confidence interval: 0.27–1.52; P=0.007) (Table 2 and Figure 1).

The mean percentage increase in KV was higher among patients with eGFR <60 mL/min/1.73 m^2 (15.43%) than among those with eGFR >60 mL/min/1.73 m^2 (8.86%), and the difference was significant (P=0.017) (Table 3).

Table 1: Comparison of kidney profile at baseline and after 1 year

Parameter	Mean±SD		P-value
	Baseline	After 1 year	
Urea (mg/dL)	29.16±6.64	31.80±5.97	0.030
Creatinine (mg/dL)	0.95±0.14	1.07±0.19	0.001
eGFR (mL/min/1.73m^2)	90.72±18.12	81.52±19.94	0.001
TKV (mm^3)	847.60±600.36	933.84±669.94	0.001

SD: Standard deviation, TKV: Total kidney volume, eGFR: Estimated glomerular filtration rate

Table 2: Distribution of % fall in eGFR

Predictor for % fall in eGFR	Correlation coefficient "r"	B (95% CI)	P-value
% increase in kidney volume	0.527	0.893 (0.27–1.52)	0.007

eGFR: Estimated glomerular filtration rate, CI: Confidence interval

Table 3: Distribution of % increase in KV with eGFR after 1 year

eGFR	Mean±SD	P-value
eGFR after 1 year (%)		
eGFR <60	15.43±4.95	0.017
eGFR >60	8.86±4.08	

KV: Kidney volume, eGFR: Estimated glomerular filtration rate, SD: Standard deviation

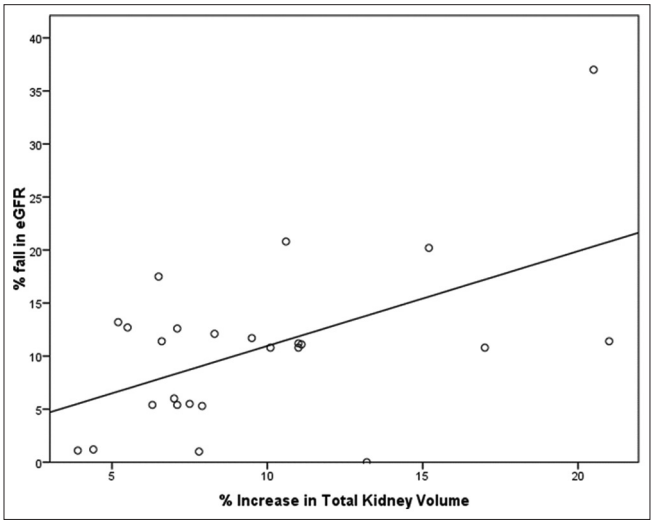

Figure 1: Scatter plot of % increase in kidney volume versus % reduction in estimated glomerular filtration rate

DISCUSSION

This study aimed to assess the correlation between TKV and eGFR in patients with ADPKD. The findings demonstrated a significant inverse relationship between increasing TKV and declining eGFR over time, highlighting the role of TKV as a marker for monitoring disease progression in ADPKD. The mean age of participants was 38.12 years, with the highest number of patients in the 41–45-year age group. This is comparable to the study by Chapman et al., in which 241 ADPKD patients had an age range of 15–45 years and a mean age of 24.5 years.[11] In our study, more than half of the patients were male; similarly, a study by Yu et al., in the Consortium for Radiologic Imaging Studies of Polycystic Kidney Disease (CRISP) study. Across various studies, male patients were slightly more prevalent than female patients, supporting the observation that ADPKD tends to be more severe in males than in females.[12]

Our study found a significant increase in TKV and a decrease in eGFR over the follow-up period, supporting the correlation between TKV and functional dysfunction. This is concluded in the study by Chen et al., the study of 541 Chinese patients with ADPKD, the mean TKV at baseline was 1265 mL, with an average increase of 5% over approximately 14.3 months. eGFR decreased significantly over this period, supporting the use of TKV as a predictive biomarker for renal function.[13]

In our study, each unit increase in % KV resulted in a 0.89-unit increase in the % fall in eGFR (P=0.007). Volumetric changes in the kidneys are directly related to decreased renal function. This finding is consistent with the study by Grantham et al., a linear relationship between kidney growth and functional loss, estimating that each unit of TKV increase could predict a corresponding GFR decrease.[2] In a study by Torres et al., the patients had a mean serum creatinine of ~1.0–1.2 mg/dL and blood urea nitrogen within the upper limit of normal at baseline, but with gradual worsening over time in untreated individuals.[14] This is similar to our study findings that an increase in creatinine (0.95→1.07 mg/dL) and urea (29.16→31.80 mg/dL) levels over 1 year is consistent with the early signs of renal decrease.

Our study showed a significant increase in TKV and a corresponding decrease in eGFR over 1 year, with a positive correlation (r=0.53) between TKV growth and eGFR. This is aligned with the study of Rule et al., which demonstrated a strong inverse relationship between TKV and eGFR. CRISP-I found that total cyst volume, age-adjusted TKV, and percentage of cyst volume were all significantly inversely correlated with GFR. In CRISP-II, this correlation strengthened over time, rising from r=−0.22 at baseline to r=−0.65 at 8 years.[15]

Our study supports the use of urea as a valid renal marker for chronic kidney disease, including ADPKD. This is similar to a study by Hossain et al., which confirmed that serum urea is independently associated with decreasing kidney function and can be used alongside creatinine for assessing disease progression.[16]

Our study found a significant increase in TKV and a decrease in eGFR over the follow-up period, demonstrating that TKV growth closely correlates with decreasing renal function in patients with ADPKD. These findings support a study by Thomsen et al., which demonstrated that KV increases as renal function decreases in PKD, highlighting TKV as a marker for disease progression.[17]

Irazabal et al. found a strong inverse association between increases in TKV and decreases in renal function, reinforcing the utility of TKV in assessing disease progression. Similarly, our study demonstrated a significant increase in TKV and a corresponding decrease in eGFR over 1 year, with a positive correlation (r=0.53) between TKV growth and the extent of eGFR decrease. These findings further support the clinical relevance of TKV measurement in predicting renal outcomes in ADPKD.[18]

Given the significant correlation between TKV and eGFR, CT-based TKV measurement may be implemented as a routine structural biomarker in the early evaluation of ADPKD, especially in resource-limited settings where MRI access is constrained. Future studies should validate these findings in larger and more diverse populations.

Limitations

As the sample was recruited from a single hospital, the findings may not be generalisable to the broader population, and the small sample size limits the strength of the conclusions. Although CT imaging is cost-effective and rapid, it exposes patients to radiation and potentially nephrotoxic contrast agents, making it less ideal for long-term follow-up in ADPKD. In addition, severe cystic changes can hinder accurate kidney delineation because of imaging artefacts. MRI, while more accurate and radiation-free, is less accessible because of its higher cost and longer scan times.

CONCLUSION

Our study identified a significant inverse correlation between increased TKV and reduced eGFR in patients with ADPKD, indicating that kidney enlargement is closely associated with declining renal function. Both serum urea and creatinine levels increased, whereas eGFR decreased, indicating worsening renal function. TKV measurements using CT showed progressive kidney enlargement, with a positive correlation between the percentage increase in TKV and the percentage decrease in eGFR. These findings highlight the potential of TKV as a reliable structural biomarker for tracking disease progression and anticipating renal function decline in patients with ADPKD. Given the limitations of CT imaging and the need for long-term assessment, future studies with larger cohorts and longer follow-ups using MRI-based volumetric techniques may further refine the utility of TKV in clinical practice and therapeutic monitoring.

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PRG- Manuscript preparation, data collection, data analysis; **SN-** Protocol review, review manuscript; **TMM-** Literature review, editing manuscript; **RD-** Review manuscript.

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