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Contribution of polycystic kidney disease to the development of chronic renal dysfunction in cats

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Abstract

Polycystic kidney disease (PKD) is a hereditary disorder characterized by progressive renal cyst formation, leading to compromised kidney function and an increased risk of chronic kidney disease (CKD). This prospective, 18-month longitudinal study evaluated diagnostic and prognostic markers in 12 PKD-affected cats, utilizing ultrasound imaging alongside biochemical analysis. The study documented a 14.00% increase in cyst volume, a 7.00% enlargement in kidney length, and significant elevations in plasma creatinine, phosphorus, and blood urea nitrogen levels, underscoring their correlation with disease progression. Hematological analysis revealed progressive anemia associated with advanced CKD stages, further outlining the systemic impact of PKD. These findings emphasize the progressive nature of CKD in cats with PKD, despite diligent care provided by pet owners and veterinarians. Also, the importance of routine sonographic evaluation along with hematological and biochemical assessments is highlighted in our study to enhance early detection and improve outcomes in feline PKD. Future longitudinal studies with expanded cohorts are essential to validate these findings and refine diagnostic criteria for PKD-associated CKD.

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Introduction

Chronic kidney disease (CKD) is the most frequently diagnosed renal disorder in cats, characterized by a structural or functional abnormality in one or both kidneys persisting for more than three months.[1] The International Renal Interest Society staging system is widely used to classify the severity of CKD in dogs and cats. This system primarily relies on fasting blood creatinine concentrations in a well-hydrated and stable patient to assign a stage ranging from one to four. In stage 1 (non-azotemic CKD) creatinine levels are below 1.60 mg dL^{-1} and clinical signs are often absent. In Stage 2, creatinine levels range from 1.60 to 2.80 mg dL^{-1} and early clinical signs such as polyuria and polydipsia may appear. Stage 3 (moderate CKD) is characterized by creatinine levels between 2.90 and 5.00 mg dL^{-1} with clinical signs potentially progressing to anorexia, vomiting and weight loss. Stage 4, known as end-stage kidney disease is identified by creatinine levels exceeding 5.00 mg dL^{-1} and a poor quality of life for the cat.[2]

Azotemia is characterized by elevated concentrations of serum creatinine and urea, making it a key diagnostic marker. Creatinine serves as a more reliable indicator of glomerular filtration rate because it is minimally influenced by extra renal factors such as diet or hepatic function.[3]

Polycystic kidney disease (PKD) is an inherited disorder that causes the formation of fluid-filled cysts in the kidneys and, in some cases, other organs such as the liver and pancreas. Diagnostic approaches, including ultrasound imaging and genetic testing, are currently the most reliable tools for diagnosing and monitoring PKD.[4] Studies on human patients with PKD have demonstrated significant correlations between imaging factors, such as kidney length, volume, and cyst size, and the progression of the disease. These findings suggest that similar markers could be applicable in feline populations.[5]

Ultrasound is a non-invasive, widely used method to assess kidney size, function, and abnormalities. This technique is favored for its accessibility, safety, and effectiveness in clinical settings. While ultrasound is not fully accurate for determining precise kidney and cyst

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volumes, its consistent measurement accuracy makes it reliable for comparative analysis.[6] Therefore, the current study focused on relative changes in kidney and cyst volume rather than their exact sizes.

Cats provide a suitable model for studying PKD progression due to similarities in disease manifestation and the feasibility of applying imaging techniques commonly used in human medicine.[7] By advancing our understanding of the relationship between clinical and imaging markers in feline PKD, this research aims to contribute to both veterinary and comparative medical knowledge. This study investigates the progression of CKD in cats diagnosed with PKD, focusing on the predictive value of blood biomarkers for CKD and their association with imaging parameters, including kidney length, volume, and cyst characteristics.

Methods and Materials

Study design. This research is a prospective study that was conducted on 198 owned cats at several veterinary clinics and teaching veterinary hospitals of Zabol University and Shahid Bahonar University of Kerman.

Selection criteria. The cats included on this study had a diagnosis of PKD confirmed through veterinary referrals for routine checkups, the presence of clinical symptoms, or a documented history of PKD in their medical records. Inclusion was not dependent on the cats' sex, breed, or age. To ensure the integrity and feasibility of the study, several exclusion criteria were implemented. Cats diagnosed with end-stage renal disease (Stage 4) with a limited life expectancy were not included. Furthermore, cats which owners were unable to commit to scheduled follow-up appointments or did not provide informed consent for their cat's participation were also excluded. Diagnostic ultrasound studies (UMT-160; Mindray, Shenzhen, China) were performed on referred cats after obtaining written consent from their owners to include in the study. Once PKD was confirmed through ultrasound imaging, blood and urine samples were collected for analysis of biochemical parameters, complete blood count, and urinalysis (on the 1st day of reception). During the initial 6 months of the study, a total of 12 cats with PKD were identified and included in the analysis.

Monitoring process for polycystic kidney disease (PKD) cases. Affected cats were evaluated at least twice: initially (on the 1st day of reception) and at the end of the study (18 months later) between 2021 and 2023. Each examination included tests for biochemical parameters, complete blood count, ultrasound imaging, urinalysis, and clinical signs, all of which were meticulously recorded. Ultrasound evaluations were conducted to document changes in cyst size, number, and kidney volume. Moreover, complete blood count, biochemical parameters and urine analysis were monitored again after 18 months in selected cats. In some cases, based on the veterinarian's prescription and clinical findings, additional evaluations (up to four) were conducted at regular intervals. However, due to limited data availability for some cases, conclusions were primarily drawn from the initial and final evaluations. During the ultrasound studies, morphological characteristics of cysts including their location (cortex or medulla), size, and number were meticulously recorded. Changes in kidney and cyst volume over time were determined using ultrasound. All patients were prepared by clipping the hair coat and applying acoustic coupling gel. The kidneys were scanned with the patient in dorsal and lateral recumbency. The left and right kidneys were imaged through the left and right ventral abdominal wall, just caudal to the ribs. A Micro Convex transducer (7.00 - 10.00 MHz) and color Doppler ultrasound machine (Mindray) was used. The treatments administered to affected cats were heterogeneous, including dietary modifications, fluid therapy, antihypertensive medications, and calcium channel blockers guided by referring veterinarians or our internist. Due to the frequent changes in therapeutic and dietary regimens observed in most cats throughout the 18-month study, the research team did not standardize or include these treatments in the analysis. Instead, our focus was on characterizing disease progression using laboratory and sonographic evaluations, independent of therapeutic interventions.

Measuring kidney volume. Kidney volume was estimated using measurements of length, width, and depth, assuming an ellipsoidal shape. The kidney volume was estimated using the standard ellipsoid formula:

$$V = (\pi \times L \times W \times D) / 6$$

where L, W, and D represent length, width, and depth, respectively. This method is consistent with veterinary ultrasonographic practices for renal volume estimation.[6]

Determining CKD staging. The International Renal Interest Society staging system was employed to classify CKD severity in PKD-affected cats.[1] The CKD stages were determined based on serum creatinine levels and clinical findings, with Stage 4 not being included.

Biochemical analysis. Blood samples collected in non-anticoagulant tubes were centrifuged at room temperature for 30 min at 503 g to separate the serum. Biochemical parameters, including creatinine, urea, phosphate, calcium, albumin, total protein, alanine aminotransferase, and gamma-glutamyl transferase, were measured using an automated biochemical analyzer and the following commercial kits were used: creatinine (Jaffé method; Pars Azmoon, Tehran, Iran),[8] urea/blood urea nitrogen (BUN, urease-glutamate dehydrogenase method; Zist Shimi, Tehran, Iran),[8] phosphate (acid phosphatase method; Pars Azmoon),[8] calcium (Arsenazo III method; Pars Azmoon),[9]

albumin (Bromocresol green method; Zist Shimi),[10] total protein (Biuret method; Pars Azmoon),[11] alanine amino-transferase (Kinase method; Pars Azmoon), and gamma-glutamyl transferase (Kinase method; Pars Azmoon).[12]

Hematology analysis. Hematology parameters, including hematocrit, red blood cell count, mean corpuscular volume, lymphocytes, and neutrophils, were measured using an automated hematology analyzer with specific kits for this device (Hema-21; Nsbiotec, Malaga, Spain). Samples were collected in tubes containing EDTA and analyzed within 2 hr of collection.

Urine analysis. Urine analysis was conducted to assess specific gravity, proteinuria, and the presence of abnormal cells, employing the following procedures. Specific gravity was determined through direct measurement using a handheld refractometer. Proteinuria was evaluated via urine dipsticks, with the results interpreted based on the provided colorimetric scale. For urine sediment microscopy, samples were first centrifuged at 1,500 revolutions *per* minute for 5 min to concentrate the cellular components. The resulting sediment was then examined under a light microscope at a magnification of 400 × to identify any abnormal cellular elements.

Statistical analysis. The dependent variables measured in this study included serum creatinine, BUN, albumin, phosphate, and calcium levels, hematological parameters, urinalysis results, and ultrasound characteristics such as renal cyst size and number. Changes in the mean values of these parameters from baseline to study completion were analyzed using paired t-tests. Comparisons between cats with PKD and healthy controls were performed using independent t-tests, with Welch's t-test applied for comparisons with unequal variances. All statistical analyses were conducted using IBM SPSS Software (version 29.0; IBM Corp., Armonk, USA).

Results

Population overview. Out of 198 cats referred for examination, 12 were diagnosed with PKD via ultrasonography (Fig. 1). The cohort consisted of Persian (n = 92), Himalayan (n = 12), Scottish (n = 8), and domestic shorthair (n = 86) cats, with PKD exclusively identified in Persian (n = 11) and domestic shorthair (n = 1) cats. At baseline, CKD stages were determined using the International Renal Interest Society criteria. Among the PKD-affected cats, 2 (16.66%) were classified as stage 3, 5 (41.66%) as stage 2, and 5 (41.66%) as stage 1. Bilateral kidney involvement was noted in 91.66% (n = 11) of affected cats. All cats participated in follow-up evaluations throughout the study period, except for one 6- year-old cat which entered the study on the first day of reception with stage 3 CKD. This cat progressed to end-stage renal failure by the ninth month, ultimately resulting in death due to end stage kidney disease.

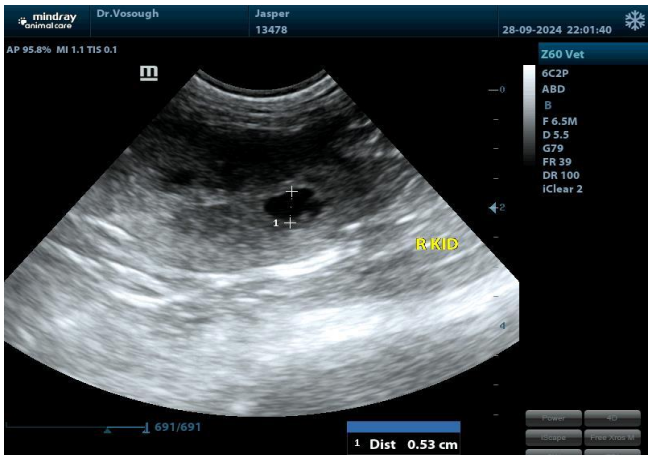

Fig. 1. Sagittal plan image of the right kidney in a domestic short hair cat. There is single cyst within the renal cortex. The cyst has anechoic contents, is well defined and the cyst wall blend with the renal cortex.

Ultrasound indicators. The comparison of cyst dimensions measured at the beginning and end of the study, based on periodic ultrasound assessments, revealed an average increase in cyst volume of 14.00% over the 18-month period. Kidney length measurements revealed a significant increase in PKD-affected cats over the study period. At baseline (day 0), the mean kidney length in PKD-affected cats was 4.20 ± 0.70 cm), compared to 3.90 ± 0.60 cm) in healthy controls. By the final day, kidney length in PKD-affected cats had increased by 7.00% (4.50 ± 0.6 cm). In one case, a cyst was exclusively detected in the right kidney, which remained cyst-free throughout the study. In seven cases, cysts were only found in the cortical region of the kidney, while in the remaining cases, cysts were present in both the cortical and medullary regions. An analysis of survival and lifespan within the study cohort revealed that one subject reached the endpoint of kidney survival, ultimately succumbing to kidney failure at month nine. To the authors' knowledge, another subject reached the endpoint of kidney survival—either through euthanasia or death resulting from kidney failure—three months after the study concluded.

Plasma biochemistry factors. The evaluation of biochemical factors in cases of PKD at baseline (first day of reception) revealed that 16.66% of subjects had concentrations of creatinine (> 1.60 mg dL^{-1}), phosphorus (4.50 mg dL^{-1}), and blood urea nitrogen (35.0 mg dL^{-1}) above standard reference values. Notably, elevated creatinine concentrations alone were observed in 58.33% of cases. Over the course of the study, an increase in plasma levels of creatinine, phosphorus, and BUN was detected, and the mean changes in these variables were statistically significant ($p < 0.05$; Table 1). This stability suggests that calcium metabolism may not be as directly impacted by PKD progression as other biochemical markers phosphorus or creatinine.

The lack of significant alterations may also indicate compensatory mechanisms maintaining calcium homeostasis despite declining kidney function. When comparing the PKD-affected cases to the healthy control group (Table 1), distinct differences emerged. The control group was evaluated at two time points: baseline (1st day of reception) and the final day of the study. A statistical comparison between the baseline results of the control group and the day zero results of the PKD group revealed no significant differences in any of the measured biochemical factors. However, comparisons between the control group and the final study day revealed significant differences in mean levels of creatinine, BUN, phosphorus, gamma-glutamyl transferase enzyme, total protein, and even calcium ($p < 0.05$; Table 1).

Urinalysis. Urinalysis of the affected cases included an assessment of urine specific gravity and the presence of proteinuria. On the initial day of the study (day zero), the mean urine specific gravity was 1.020, and proteinuria was detected in four cases, accounting for 33.33% of the total. By the conclusion of the study, the mean specific gravity had increased to 1.025, with proteinuria persisting in the same four cases (33.33%).

Hematology. Hematologic parameters were compared between healthy cats and those with PKD+. Although some variables showed no significant differences at the beginning of the study (initial day), significant reductions in hematocrit and erythrocyte counts were observed in PKD+ cats by the final day. These findings suggest progressive anemia in affected individuals. Other indices, including mean corpuscular volume, mean corpuscular hemoglobin, mean corpuscular hemoglobin concentration, lymphocyte percentage, and neutrophil band counts, did not differ significantly across groups or time points. Notably, as CKD progressed, erythrocyte counts and hematocrit levels demonstrated a decreasing trend. Detailed values and statistical comparisons are provided in Table 2.

Clinical observations. During periodic veterinary examinations, clinical symptoms such as anorexia, weakness, vomiting, and diarrhea were documented in a subset of cats affected by PKD. Anorexia and weakness were the most frequently observed symptoms, noted in 33.33% (n = 4) of cases. Vomiting and diarrhea were less common, occurring sporadically in 16.66% (n = 2) of affected cats. Symptoms did not correlate uniformly with CKD stage or severity, and their occurrence varied widely among individuals. No significant associations were identified between clinical signs and biochemical or hematological parameters during the study period.

Table1. General data and comparison of biochemical factors of healthy group versus cats with polycystic kidney disease (PKD) at two times: Initial and the final day of the study. Data are presented as mean ± SD.

Variables	Healthy (Initial day)	PKD (Initial day)	PKD (Final day)	*p*-values		
				Healthy *vs.* PKD initial day	Healthy *vs.* PKD final day	PKD initial day *vs.* final day
Age (months)	44.00 ± 9.00	52.00 ± 7.00	70.00 ± 7.00	-	-	-
Weight (kg)	3.80 ± 1.20	3.80 ± 0.80	3.10 ± 1.20	-	-	-
Creatinine (mg dL^{-1})	1.48 ± 0.39	1.82 ± 0.75	2.81 ± 1.31	0.109	< 0.001	< 0.001
BUN (mg dL^{-1})	26.70 ± 4.49	29.70 ± 4.93	36.70 ± 9.30	0.093	< 0.001	0.013
ALP (U L^{-1})	62.90 ± 11.54	67.08 ± 15.29	59.62 ± 16.19	0.399	0.510	0.202
GGT (U L^{-1})	2.88 ± 0.64	3.64 ± 0.73	3.61 ± 0.80	0.357	0.003	0.153
Total Protein (g dL^{-1})	6.71 ± 0.44	7.10 ± 0.46	7.50 ± 0.66	0.025	< 0.001	0.053
Phosphate (mg dL^{-1})	3.46 ± 1.09	3.51 ± 1.23	4.40 ± 1.22	0.908	0.043	< 0.001
Calcium (mg dL^{-1})	9.61 ± 0.29	10.09 ± 1.33	10.75 ± 1.5	0.347	0.009	0.179
Albumin (g dL^{-1})	3.06 ± 0.46	3.40 ± 0.80	2.98 ± 0.48	0.418	0.615	0.551

BUN: Blood urea nitrogen; ALP: Alkaline phosphatase; GGT: Gamma-glutamyl transferase.

Table 2. Hematologic parameters comparison between healthy cats and cats with polycystic kidney disease (PKD) at two times: Initial and the final day of the study. Data are presented as mean ± SD.

Variables	Healthy (Initial day)	PKD (Initial day)	PKD (Final day)	*p*-values		
				Healthy *vs.* PKD initial day	Healthy *vs.* PKD final day	PKD initial day *vs.* final day
Hematocrit (%)	38.90 ± 4.80	38.90 ± 4.80	28.80 ± 4.70	0.824	< 0.01	0.003
Erythrocytes (× 10^6 *per* µL)	7.66 ± 1.23	7.33 ± 1.38	5.23 ± 1.71	0.487	< 0.01	0.05
MCV (fL)	40.70 ± 1.40	40.6 ± 1.40	40.20 ± 3.80	0.966	0.829	0.713
MCH (pg)	15.80 ± 0.90	15.60 ± 1.10	15.40 ± 1.30	0.680	0.420	0.680
MCHC (g dL^{-1})	31.20 ± 0.80	31.00 ± 0.90	30.08 ± 1.10	0.710	0.320	0.710
Lymphocytes (%)	42.30 ± 1.60	43.30 ± 1.60	43 ± 1.40	0.581	0.644	0.252
Band neutrophils (%)	7.40 ± 1.60	6.90 ± 1.70	6.90 ± 2.70	0.926	0.870	0.729
Segmented neutrophils (%)	52.30 ± 1.40	51.50 ± 1.40	50.24± 1.10	0.669	0.660	0.201

MCV: Mean corpuscular volume; MCH: Mean corpuscular hemoglobin; MCHC: Mean corpuscular hemoglobin concentration.

Discussion

This study evaluated the progression of PKD in cats, focusing on diagnostic tools, biochemical markers, and disease progression indicators over an 18-month period. The study was conducted on 198 cats of various breeds, including Persian, domestic shorthair, Scottish, and Himalayan. The findings revealed that CKD in affected cats progressed rapidly despite diligent follow-up care by pet owners and veterinarians. This progression was closely associated with the monitoring of renal cysts through ultrasonography.

A 14.00% increase in cyst size over 18 months was observed, while the number of detectable cysts remained constant. This contrasts with human studies, which typically report an increase in cyst numbers with age.[13] However, the findings do not conclusively establish whether the decline in renal function and CKD progression in feline PKD is driven solely by cyst enlargement or an increase in cyst numbers. Further investigation is required to differentiate between these potential mechanisms.

The significant correlation between biochemical parameters and cyst enlargement suggests that monitoring cyst growth could serve as a reliable indicator of PKD progression. However, validating this hypothesis necessitates longitudinal studies with longer observation periods. Interestingly, the findings of Wills *et al*. reported a 66.00% increase in cyst numbers over a 10-month period, potentially due to undetected small cysts during initial assessments or the younger age of the affected cats in their study. These discrepancies underline the need for standardized methodologies in feline PKD research.[14]

Comparisons with human PKD studies further emphasize the value of imaging techniques in disease monitoring. Ultrasound, a cost-effective and accessible method, was identified as an effective tool for tracking kidney enlargement and cyst growth, consistent with findings in human PKD research,[5,15] demonstrated that ultrasound provides accurate renal volume estimates in cats,[6] and Wills *et al*. confirmed its repeatability for detecting renal cysts in feline populations.[14] While magnetic resonance imaging has been shown to be highly effective in predicting disease progression and evaluating therapeutic responses, this study advocates for simpler techniques like ultrasound for long-term monitoring and treatment planning in feline PKD.[16]

Biochemical markers also played a crucial role in this study. Elevated plasma concentrations of creatinine and phosphorus were significantly correlated with ultrasound findings, including increased cyst size and kidney involvement. Statistical analysis confirmed significant differences in creatinine and phosphorus levels between PKD-affected cats and healthy controls. However, these results contrast with findings from Noori *et al*. which reported no significant correlation between these markers in Persian cats, likely due to the shorter disease duration and younger study population.[17]

The significant elevation in plasma creatinine and phosphorus levels reflects declining glomerular filtration rate due to cyst-induced renal parenchymal compression. This aligns with findings in human PKD studies, where cyst expansion correlates with reduced kidney function. Elevated phosphorus levels, observed in advanced CKD stages, further underscore the systemic impact of renal dysfunction.[18]

The anemia observed in advanced CKD appears to have multiple underlying causes. A primary factor is erythropoietin deficiency, as declining kidney function reduces production of this essential hormone that stimulates red blood cell production - a well-established phenomenon in human CKD[13] Additionally, the accumulation of uremic toxins may contribute by shortening red blood cell survival. Nutritional factors also likely play a role, since decreased appetite in late-stage CKD can lead to deficiencies in iron and vitamins essential for erythropoiesis. While this pattern of normocytic, normochromic anemia mirrors what is seen in human CKD patients, it contrasts with findings from a study,[17] that reported no significant anemia in Persian cats. This discrepancy may reflect differences in either the severity of kidney disease or the duration of observation across studies.

Blood urea nitrogen levels increased over the study period but did not reach statistical significance. This aligns with human studies, where elevated BUN is more indicative of advanced CKD stages.[19] The absence of significant BUN changes in this study may reflect the limited number of cases in advanced PKD stages and the extensive renal function loss observed in the affected population. Similarly, fluctuations in serum creatinine were noted, but these did not correspond to significant changes in CKD staging, which might explain the lack of clinical signs such as uremia.

The stability of plasma calcium levels, despite declining kidney function, suggests compensatory mechanisms such as increased intestinal absorption or reduced renal excretion. This contrasts with human PKD, where hypercalcemia is often observed due to vitamin D dysregulation.[20]

The lack of observed hypoalbuminemia or proteinuria in our study contrasts with previous findings,[21] that reported urinary proteomic alterations in PKD-affected cats. Several factors could explain this discrepancy. First, our cohort predominantly consisted of early-stage CKD cases (75.00% in stages 1 - 2), which may not yet exhibit these biochemical changes. Second, standard dipstick urinalysis may not be sensitive enough to detect mild proteinuria. More advanced diagnostic approaches, such as magnetic resonance imaging or novel urinary bio-markers like symmetric dimethylarginine, may be better

for identifying early renal damage. Notably, repeated urinalysis performed both at baseline and study completion consistently showed no detectable urinary albumin. These results suggest that serum and urinary albumin measurements may have limited value as markers of progression in feline PKD, although additional studies are needed to verify this conclusion.[17]

Advanced imaging techniques such as magnetic resonance imaging or urinary biomarkers like (symmetric dimethylarginine) may be more effective in detecting early renal damage. Urinalysis conducted at baseline and at the study's conclusion also detected no urinary albumin. These results indicate that serum and urinary albumin may not be reliable markers of progression in feline PKD. However, additional research is needed to confirm this.[21]

This study encountered significant limitations. The impact of cyst location (medulla, cortex, or cortex-medulla) on renal function could not be evaluated due to the limited number of cases with medullary cysts. Additionally, the aim of estimating the lifespan of cats with PKD was not achieved, primarily due to the short observation period and variability in disease severity and care practices among the study population. The variability in treatment regimens highlights a study limitation. However, the reliance on objective parameters (e.g., cyst growth, creatinine trends) minimized confounding effects, as these markers are less sensitive to short-term interventions compared to subjective clinical assessments. Due to the challenges of sampling and the limited size of the statistical population, some correlation analyses (such as the Spearman test) require further validation in larger studies. This research lays the groundwork for future investigations with broader statistical populations to extract findings with greater precision.

This study observed a statistically significant increase in plasma concentrations of creatinine, phosphorus, and BUN over time and at the conclusion of the study compared to baseline levels. In contrast, no significant changes were noted in plasma calcium levels, and variations in calcium concentrations over time were not significant. Periodic ultrasonographic evaluations revealed a 14.00% increase in cyst size from the beginning to the end of the study, with measurements performed in the sagittal plane to ensure accuracy in documenting cyst dimensions.

Additionally, there were statistically significant differences in erythrocyte count and hematocrit levels between the initial and final phases of the study.

Comparative analyses between the affected and control (healthy) groups at baseline and the end of study indicated no significant mean differences in any measured variables at baseline. However, significant changes were observed in creatinine, BUN, phosphorus, calcium, erythrocytes, and hematocrit levels in the affected group by the end of the study.

These findings underscore the progressive nature of CKD in cats with PKD, despite diligent care provided by pet owners and veterinarians. The study highlights the critical role of factors such as creatinine, phosphorus, BUN, erythrocytes, and hematocrit in estimating the progression of PKD. Routine monitoring of these parameters is essential for assessing disease progression and guiding the management of PKD.

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Conflict of interest

The authors declare that there are no conflicts of interest in disclosing this work.

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