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Cardiovascular changes in Persian cats with polycystic kidney disease: a study of cardiac troponin I, echocardiography and blood pressure

Niloufar Saberi[1], Ehsan Khaksar[2], Mohammad Molazem[3] and Seeyamak Mashhady Rafiee[1]

Abstract

Objectives Cardiovascular complications are well known in humans with autosomal dominant polycystic kidney disease (PKD), but limited data exist for cats. This study aimed to assess echocardiographic changes, cardiac troponin I (cTnI) levels and systolic blood pressure (SBP) in Persian cats with PKD to detect early cardiac abnormalities.

Methods In total, 52 Persian and mixed-Persian cats were enrolled, with 26 cats in the control group and 26 diagnosed with PKD via ultrasound due to the unavailability of genetic testing. Although genetic testing is the gold standard for definitive diagnosis, this study utilised high-sensitivity ultrasound as an alternative diagnostic tool. This method aligns with existing literature supporting its effectiveness in detecting PKD, particularly in regions where genetic testing is not accessible. Echocardiographic examinations employed M-mode and two-dimensional echocardiography to measure the diastolic thickness of the interventricular septum and the left ventricular free wall. Doppler ultrasonography was used to measure SBP and cTnI serum levels were determined using a Monobind-ELISA kit.

Results Median SBP and cTnI levels in PKD cats were 155 mmHg and 85.80 ng/l, respectively, which was significantly higher than the control group ($P \leqslant 0.001$). Interventricular septum in systole, as well as diastolic thickness of the interventricular septum and the left ventricular free wall, was significantly elevated in PKD cats compared with controls ($P \leqslant 0.001$). No significant differences were observed in other echocardiographic parameters.

Conclusions and relevance Asymptomatic PKD-affected Persian cats exhibited elevated SBP and cardiac structural changes; however, the clinical significance of these findings remains uncertain due to a lack of long-term follow-up. While early cardiac changes may be present, further research is necessary to establish their clinical relevance and guide appropriate management strategies. Monitoring PKD cats is advised, but a direct clinical impact is not confirmed at this stage.

Plain language summary

This study focuses on how polycystic kidney disease (PKD) affects the heart health of Persian cats. PKD is common in these cats, and while it is known to cause heart issues in humans, less is understood about its impact on cats. This research aimed to detect early heart problems in Persian cats with PKD by examining their heart structure, a heart protein called cardiac troponin I (cTnI) and blood pressure.

Keywords: Polycystic kidney disease; early markers; cardiac troponin I; echocardiographic changes; systolic blood pressure

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Introduction

Polycystic kidney disease (PKD) is an inherited disorder prevalent in Persian and mixed-Persian cats due to a mutation in the *PKD1* gene. This condition leads to the formation of fluid-filled cysts in the kidneys, which grow and replace normal tissue, potentially causing renal failure and posing a serious health risk to affected cats.[1–4]

Cardiorenal syndrome (CRS) in cats affects both the cardiovascular system and kidneys, with dysfunction in one organ impacting the other.[5] CRS involves autonomic reflex changes, fluid imbalance and neurohumoral alterations.[6] Cardiovascular abnormalities in humans with autosomal dominant polycystic kidney disease (ADPKD) are well documented, including hypertension driven by activation of the renin–angiotensin–aldosterone system (RAAS). While similar mechanisms may exist in cats, the application of human ADPKD findings to feline PKD should be interpreted with caution because the pathophysiology may differ between species.[7–9]

Systemic hypertension in cats can lead to a range of adverse clinical effects, including damage to cardiac, vascular and renovascular tissues; however, the extent of irreversible damage in cats with hypertension remains uncertain and requires further study.[5] In hypertensive humans with secondary left ventricular hypertrophy (LVH) due to hypertension, moderate rises in cardiac troponin I (cTnI) concentrations have been observed, indicating significant cardiac involvement; however, whether these findings relate to humans or cats with PKD and hypertension is unknown and requires further investigation.[10]

Limited research has focused on cardiac abnormalities in Persian cats with PKD. Reports suggest mild left ventricular dilatation, increased endocardial echogenicity, a slight increase in mean restrictive diastolic filling pattern and mitral valve regurgitation.[11] LVH has been identified in 50% of normotensive ADPKD cats, along with a slight increase in basal interventricular septal thickness at end-diastole near the left ventricular outflow tract and increased aortic artery flow velocity.[12]

This study aimed to evaluate echocardiographic changes, cTnI levels and systolic blood pressure (SBP) in Persian cats with PKD to identify whether significant cardiovascular changes could be detected in this population. The purpose of the study is to determine whether these parameters reflect subclinical cardiovascular effects associated with PKD.

Materials and methods

Client-owned Persian and mixed-Persian cats were recruited from local veterinary clinics, which regularly conducted health screenings. Recruitment was carried out over 6 months through referrals from veterinarians, who identified potential candidates either during routine check-ups or when PKD was suspected based on ultrasonographic findings.

Inclusion criteria required the cats to be of Persian or mixed-Persian breeds, verified through owner-provided documentation (eg, pedigree certificates) and a physical examination by veterinarians to assess distinct breed characteristics. Any cat with unclear breed characteristics or ambiguous lineage was excluded from the study to ensure consistency and transparency.

Before participation, informed consent was obtained from all owners. Cats were systematically evaluated and excluded if they displayed any clinical signs of illness, including anorexia, fever, vomiting, diarrhoea, dyspnoea, cough, seizures, cyanosis, exercise intolerance, oedema and ascites, or if any abnormal ultrasound findings other than PKD were detected (eg, masses, cystitis or nephroliths). Additionally, all cats underwent comprehensive blood tests (complete blood count, biochemistry and thyroxine levels) following 12 h of fasting to confirm their overall health status.

This thorough evaluation process ensured that only healthy, asymptomatic Persian and mixed-Persian cats were included in the study.

The study population consisted of 26 Persian cats diagnosed with PKD and 26 healthy Persian cats (non-PKD), which served as the control group. All cats were recruited through local veterinary clinics.

The control group consisted of 26 healthy Persian cats, which were recruited through local veterinary clinics. Selection criteria included cats with normal blood tests, no systemic diseases and no kidney cysts detected on

[1]Department of Clinical Science, Science and Research Branch, Islamic Azad University, Tehran, Iran
[2]Department of Clinical Sciences, Garmsar Branch, Islamic Azad University, Garmsar, Iran
[3]Department of Veterinary Surgery and Diagnostic Imaging, Faculty of Veterinary Medicine, Tehran University, Tehran, Iran

Corresponding author:
Ehsan Khaksar DVM, DVSc, PhD, Department of Clinical Sciences, Garmsar Branch, Islamic Azad University, Daneshjoo Street, Daneshgah Square, Garmsar, 358163116, Iran
Email: ehsan_khaksar@yahoo.com

ultrasound. These cats were identified during routine checkups at the clinics and confirmed to be in good health through comprehensive physical examinations and laboratory evaluations, including complete blood counts, biochemistry panels and ultrasonography to rule out any underlying renal or systemic conditions. Cats in this study received no medications. All relevant animal care guidelines were adhered to, and the study was approved by Azad University's Research Ethics Committee (approval no. IR.IAU.SRB.REC.1402.022).

Serum samples were centrifuged and stored at –70°C. cTnI levels were measured using a Monobind-ELISA kit (Monobind).[13] This assay has been validated for use in feline subjects, ensuring its sensitivity and accuracy in detecting cardiac injury biomarkers in cats. The lower limit of detection for the assay was 1.5 ng/l, and all procedures followed the manufacturer's instructions to ensure consistency. The reference interval for troponin I in healthy cats ranges from <0.03 to 0.16 ng/ml (<30–160 ng/l) based on established guidelines.[14,15]

SBP in cats was measured using Doppler ultrasonography (Vet-Dop2; Vet Quip) after settling the cats in a quiet room for 5–20 mins to reduce anxiety. A 2.5 cm cuff was attached to the right forelimb and the probe was placed on the first palmar common digital artery. The mean of five consistent measurements was recorded for SBP.[16,17]

Echocardiographic examinations were conducted using a Mindray DP-50 Ultrasound System equipped with 7.5 and 12 MHz probes (Mindray). The equipment was calibrated according to the manufacturer's specifications before each session to ensure optimal performance. Cats were placed on their right side without sedation to minimise stress and movement artefacts during the procedure. Two-dimensional and M-mode echocardiography were used to evaluate cardiac structures, including measurements of the diastolic thickness of the interventricular septum and left ventricular free wall. Specific settings such as gain, depth and focus were adjusted to enhance image clarity for each cat. These steps were followed consistently to ensure reproducibility and diagnostic accuracy across all subjects. Myocardial hypertrophy was defined as diastolic wall thickness ⩾6 mm.[18] Additional echocardiographic parameters included the measurement of the aortic root (Ao) and left atrium (LA) diameters. Left atrial enlargement was identified when the LA:Ao ratio exceeded 1.5,[11,19] aligning with established veterinary guidelines. Doppler echocardiography was used to detect flow and valve disorders. Three measurements per parameter were taken, and median values were used.

Cats with creatinine levels below 1.6 mg/dl were classified as International Renal Interest Society (IRIS) stage 1, while those with creatinine levels between 1.6 and 2.8 mg/dl were assigned to IRIS stage 2. This classification follows the current IRIS guidelines, ensuring an accurate representation of chronic kidney disease (CKD) progression in these cats.[20]

Data analysis was conducted using SPSS, version 22 (IBM), presenting the mean ± SD, median and interquartile ranges (IQRs). The Kolmogorov–Smirnov test was applied to assess normal distribution. Group indices were compared using *t*-tests, Mann–Whitney tests, Pearson χ^2 and Fisher's exact tests. P <0.05 was considered statistically significant.

Results

Of the 52 Persian cats enrolled (50% PKD and 50% non-PKD), no significant sex differences were noted (P = 0.267). There was no significant difference between PKD and non-PKD cats in mean age (61.4 ± 25.5 months, range 24–144 vs 62.8 ± 22.1, respectively; range 12–108; P = 0.755) or mean weight (4.3 ± 0.9 kg, range 2.75–5.85 vs 4.4 ± 0.8, respectively; range 2.9–5.9; P = 0.359).

Significant differences were found in key biochemical parameters between groups, as expected (Table 1). PKD cats had higher levels of blood urea nitrogen (BUN) and creatinine (CR) compared with the control group. Of the 26 PKD cats, 19 were classified as IRIS stage 1 and seven as IRIS stage 2 based on current creatinine cut-offs. No significant differences were observed in other biochemical parameters between the two groups.[21]

Although all cTnI results fell within the reference interval (<30–160 ng/l),[22] there was a significant difference in concentrations between the PKD and non-PKD groups (P ⩽0.001). The median cTnI concentration was 85.8 ng/l (IQR = 8.4–103.8) in PKD cats, compared with 3.0 ng/l (IQR = 1.5–13.2) in non-PKD cats (Figure 1). This trend indicates that PKD cats may have higher baseline cTnI levels compared with non-PKD cats.

Table 1 Biochemical parameters significantly different between non-PKD and PKD Persian cats (mean ± SD, min–max)

Parameters	Non-PKD cats (n = 26)		PKD cats (n = 26)		Reference interval	*P* value
	Mean ± SD	Min–max	Mean ± SD	Min–max		
BUN (mg/dl)	27.2 ± 4.8	17–32	32.7 ± 10.2	19–52	17–32	0.01
CR (mg/dl)	1.2 ± 0.2	0.9–1.55	1.6 ± 0.2	1.4–2.1	0.9–2.1	⩽0.001

Independent sample *t*-test
BUN = blood urea nitrogen; CR = creatinine; PKD = polycystic kidney disease

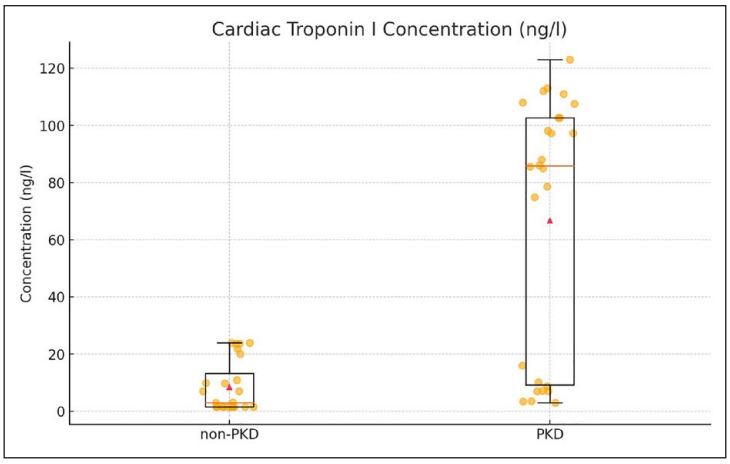

Figure 1 Distribution of cardiac troponin I (ng/l) in non-PKD and PKD groups. The box plots display the interquartile range, median and outliers for each group, while the scatter plot overlays individual data points to highlight the distribution within each group. The median is shown by the red line, and the mean is shown by the triangle. Scatter points are represented by dots to provide a clearer view of the individual data points within each group. PKD = polycystic kidney disease

Table 2 Systolic blood pressure of Persian cats enrolled in the study (median [interquartile range], min–max)

Parameters		Non-PKD cats (n = 26)		PKD cats (n = 26)		*P* value
		Median (IQR)	Min–max	Median (IQR)	Min–max	
SBP (mmHg)		130 (120–140)	120–140	155 (137.5–162.5)	120–170	≤0.001
Number of cats in different SBP categories*	Normotensive (SBP <140 mmHg)	19		6		
	Prehypertensive (SBP 140–159 mmg)	7		7		
	Hypertensive (SBP 160–179 mmHg)	0		13		
	Severely hypertensive (SBP ⩾180 mmHg)	0		0		

Mann–Whitney test
*The number of cats in different SBP categories is based on the ACVIM guidelines[24]
ACVIM = American College of Veterinary Internal Medicine; IQR = interquartile range; PKD = polycystic kidney disease; SBP = systolic blood pressure

Of the 26 PKD cats, 13 exhibited elevated SBP (⩾160 mm Hg), which may suggest hypertension; however, since only asymptomatic Persian cats were included in this study, none of these cats displayed any clinical signs typically associated with hypertension, such as ocular changes (eg, retinal haemorrhage, retinal detachment) or other hypertensive signs.[23] A significant difference in SBP was observed between groups ($P \leqslant 0.001$). Non-PKD cats had a median SBP of 130 mmHg, while PKD cats had a median SBP of 155 mmHg (Table 2).[24]

LVH was observed in 69% of PKD cats, with significantly higher values for interventricular septum in systole, as well as diastolic thickness of the interventricular septum and left ventricular free wall compared with non-PKD cats ($P \leqslant 0.001$). Left atrial enlargement was noted in 15% of PKD cats, but this did not significantly differ between groups ($P = 0.07$) (Table 3).

There were no statistical differences between groups in other echocardiographic parameters, including ejection fraction, fractional shortening and left ventricular internal diameter end-diastole and end-systole (Table 3). No abnormalities in valve leaflets were observed.

Discussion

Although genetic testing for PKD is strongly recommended for Persian and Persian-derived breeds to prevent the transmission of PKD and exclude cats carrying the mutated gene, it is not widely available in countries such as Iran, Turkey and Brazil, despite the prevalence of Persian cats in these regions. This unavailability of genetic testing limits its use as a screening tool, and in regions such as Iran, where genetic testing is inaccessible, ultrasound is often used as a practical alternative. While ultrasound remains a reliable diagnostic tool, it is not

Table 3 Echocardiographic profile of Persian cats enrolled in the study (median [interquartile range], min–max)

Parameters	Non-PKD cats (n = 26)		PKD cats (n = 26)		Normal reference values	*P* value
	Median (IQR)	Min–max	Median (IQR)	Min–max		
FS (%)	42 (28–46.2)	25–89	47 (39–87)	34–89	28–52%	0.09
EF (%)	76.5 (51–78.7)	44–84	71.5 (59–74)	57–80	55–90%	0.20
IVSs (mm)	4 (3–4.5)	3–6	7 (7–10)	5–10	3–5 mm	**⩽0.001**
IVSd (mm)	4 (3–4)	3–5	5.50 (5–8)	4–8	3–5 mm	**⩽0.001**
LVIDs (mm)	7 (7–9)	6–12	8 (7–8.25)	7–9	6–9 mm	0.37
LVIDd (mm)	15 (12–16)	12–18	15 (13.7–16.2)	12–17	12–18 mm	0.55
LVFWs (mm)	6 (6–6.2)	5–7	6 (6–7)	5–8	3–6 mm	0.20
LVFWd (mm)	5 (3–5)	3–5	6 (6–7)	4–7	3–5 mm	**⩽0.001**
LA:Ao	1.2 (1.1–1.5)	1–1.50	1.5 (1–1.5)	1–1.60	1–1.5	0.07

Mann–Whitney test
P values in bold are statistically significant
EF = ejection fraction (%); FS = fractional shortening (%); IQR = interquartile range; IVSd = interventricular septum in diastole; IVSs = interventricular septum in systole; LA:Ao = left atrium:aortic root ratio; LVIDd = left ventricular internal diameter in diastole; LVIDs = left ventricular internal diameter in systole; LVFWd = left ventricular free wall in diastole; LVFWs = left ventricular free wall in systole; PKD = polycystic kidney disease

as accurate as genetic testing for definitively identifying carriers of the PKD mutation. This emphasises the urgent need for widespread screening, whether through genetic testing or ultrasound, to identify affected cats and ensure they are neutered to prevent breeding and the further spread of PKD. Such preventive measures are particularly important in regions with high PKD prevalence, where the disease continues to affect breeding populations.[25–28]

Many cats with IRIS stages 1 and 2 CKD may appear clinically healthy and go undiagnosed without specific investigations, which is a characteristic not unique to PKD but common in other forms of CKD. With appropriate screening, such cats can be identified earlier, potentially leading to opportunities for earlier intervention depending on the specific manifestations and complications of the disease.[21] In this study, despite appearing healthy, seven PKD cats were found to be in early stage 2 CKD, underscoring the silent progression of the disease and the importance of more frequent and comprehensive assessments. This study focused on assessing cardiac changes to explore the early detection of associated cardiovascular problems in PKD-affected cats.

Research on cats afflicted with PKD has shown elevated mean SBP compared with controls.[11] The growth of renal cysts likely reduces kidney blood flow, leading to renal hypoperfusion and the activation of the RAAS, which is often associated with hypertension.[11,29]

A case study on a PKD-affected cat also demonstrated that PKD can lead to hypertension and complications such as arterial thromboembolism,[30] highlighting the serious cardiovascular risks. In humans with ADPKD, 50–70% initially present with hypertension, even with normal kidney function.[10] Similarly, our study found that hypertension can occur in asymptomatic PKD cats, revealing hidden cardiovascular risks that may exist even when clinical signs are not visible.

Hypertension is commonly associated with LVH in both humans and cats with CKD. However, while similar trends are observed in feline PKD, it is important to recognise that the mechanisms in cats may differ from humans.[11,31] For example, human studies on ADPKD show several cardiovascular manifestations, such as aneurysms, aortic root dilation and mitral valve defects, which have not been extensively documented in feline PKD.[12] Therefore, caution should be exercised when applying human findings to cats. In our study, LVH was identified as the most common cardiac complication in PKD Persian cats, underscoring the need for regular cardiovascular monitoring of these animals. Further research specific to feline PKD is necessary to better understand the significance of these cardiovascular changes and their long-term implications.[32–34]

cTnI is a precise and sensitive biomarker that indicates cardiac muscle cell damage, with elevated levels commonly observed in cats with heart-related disorders. Increased cTnI levels typically reflect ongoing myocardial damage, which is a hallmark of several myocardial diseases in cats. Although human studies have linked elevated cTnI levels to subclinical LVH and dysfunction in community-based studies involving detailed echocardiographic analysis, caution is needed when applying these findings to cats because the mechanisms in cats and humans may differ.[19,35]

In our study, although all cTnI levels in PKD cats were within the normal reference interval, a significant difference was observed between the PKD and non-PKD groups. This finding, although important, should be interpreted carefully because the observed differences suggest only mild abnormalities in PKD cats, particularly in those with mild azotemia. The limitations of our study, including the lack of long-term follow-up, mean that the clinical significance of these findings remains uncertain.

While these results suggest that PKD cats may be predisposed to subclinical myocardial changes, more detailed and larger studies are necessary to determine the long-term cardiovascular implications.

Although the combination of elevated cTnI levels, high SBP and LVH could suggest ongoing myocardial damage, the data from this study show only mild cardiac abnormalities. These findings emphasise the need for further research to evaluate the clinical relevance of these changes. At this stage, it would be premature to advocate for comprehensive cardiac assessments based on the data, and future studies should focus on exploring whether cTnI, SBP and LVH can serve as early indicators of cardiovascular health risks in PKD cats.

Conclusions

This study provides preliminary insights into potential cardiovascular changes in PKD Persian cats, revealing some evidence of cardiac and blood pressure changes. However, these findings do not establish a need for early detection or management but rather suggest that further research is needed to assess the clinical significance of these mild changes, especially since many values, such as cTnI levels, were within normal ranges.

The study's limitations, including the lack of genetic testing and long-term follow-up, prevent drawing definitive conclusions about the progression or management of these cardiovascular changes. While monitoring cardiovascular parameters in PKD cats may be useful, further studies are required to determine their true clinical relevance.

Given the hereditary nature of PKD, routine genetic or ultrasound screening is still recommended for breeding cats to reduce PKD transmission. More research is needed to explore preventive strategies and better understand the role of the RAAS in PKD-related cardiovascular health.

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Ethical approval The work described in this manuscript involved the use of non-experimental (owned or unowned) animals. Established internationally recognised high standards ('best practice') of veterinary clinical care for the individual patient were always followed and/or this work involved the use of cadavers. Ethical approval from a committee was therefore not specifically required for publication in *JFMS*. Although not required, where ethical approval was still obtained, it is stated in the manuscript.

Informed consent Informed consent (verbal or written) was obtained from the owner or legal custodian of all animal(s) described in this work (experimental or non-experimental animals, including cadavers, tissues and samples) for all procedure(s) undertaken (prospective and retrospective studies). For any animals or people individually identifiable within this publication, informed consent (verbal or written) was also obtained from the individuals involved for their use in the publication.

ORCID iD Niloufar Saberi https://orcid.org/0000-0001-9915-1500
Ehsan Khaksar https://orcid.org/0000-0001-7363-0300

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