Indian J. Vet. Pathol., 49(3) : 238-241, 2025: DOI: https://doi.org/10.56093/675qc250

SHORT COMMUNICATION

Polycystic kidney disease in a Labrador dog

S. Sivaraj*, A. Arulmozhi, M. Sasikala, K. Gopal, P. Balachandran, P. Srinivasan and D.C. Monisha
Department of Veterinary Pathology, Veterinary College and Research Institute, Namakkal, TANUVAS

Address for Correspondence
S. Sivaraj, PG Scholar, Department of Veterinary Pathology, Veterinary College and Research Institute, Namakkal, TANUVAS, E-mail: sivarajvetpatho@gmail.com

Received: 28.3.2025; Accepted: 29.5.2025

ABSTRACT

An eleven-month labrador dog was brought to Veterinary clinical complex, Veterinary College and Research Institute, Namakkal with a history of anorexia, frequent vomition and progressive weight loss. Hematology and serum biochemical analysis showed severe anaemia, leukopaenia, azotemia, hyperphosphatemia and hypocalcemia. The case was tentatively diagnosed as Polycystic kidney disease based on clinical signs, haematobiochemical parameters and ultrasonography. In spite of the palliative treatment, the animal died within a week and the same was referred to Department of Veterinary Pathology for necropsy. The carcass was severely emaciated and ulcers were noticed in the oral cavity. Kidneys showed numerous, whitish, irregular and varying-sized fluid-filled cysts studded over the entire renal parenchyma. Histopathological examination of the kidney revealed large amount of fibrous tissue in the interstitium with irregularly dilated cysts and some cysts with homogeneous, acidophilic material in the lumen. On Masson's trichrome staining and Van Gieson's staining, the fibrous stroma appeared blue and bright red in colour respectively. The liver showed portal fibrosis with bile duct hyperplasia. This communication deals with the pathology of congenital polycystic kidney disease in a dog.

Keywords: Anaemia, hyperphosphatemia, Labrador dog, masson's trichrome stain, polycystic kidney, Van Gieson's stain

Polycystic kidney disease (PKD) is a rare genetic disorder that occurs either as an autosomal dominant polycystic kidney disease (ADPKD) in adults or autosomal recessive polycystic kidney disease (ARPKD) in young ones[1]. The PKD is characterized by the formation of multiple cysts in the cortex and medullary region with primary lesions in the renal tubules. These cysts continue to grow and deteriorate the normal renal function leading to chronic kidney disease like symptoms and lesions[2]. The animal often do not develop any clinical signs until the disease has progressed significantly. At the advanced stage, the animals shows symptoms like polydipsia, polyuria, lethargy, poor appetite and progressive weight loss. Microscopically, PKD is characterized by extensive renal fibrosis and dilated cysts throughout the renal parenchyma with limited functional renal tissues. Eventually, these changes associated with PKD compromise the renal function and can progress to chronic kidney disease and end stage renal failure in affected dogs. Histopathological examination of the kidney remains the preferred method for the definitive diagnosis of the condition despite the ultrasonography, which helps in the early diagnosis of the condition. Though there are several literatures on occurrence of PKD in cats, there are only few reports on the occurrence and pathology of PKD in dogs. Understanding the pathology of PKD is essential for the accurate diagnosis of the condition, monitoring the disease progression which provides insights into the breeding programs especially in the predisposing breeds. In this regard the present communication deals with the pathology and various pathological manifestations of renal lesions in a dog diagnosed with polycystic kidney disease.

An eleven-month-old female Labrador dog weighing about 10 kg was presented to the Veterinary Clinical Complex, Veterinary College and Research Institute, Namakkal with a history of anorexia, frequent vomition, polyuria, polydipsia and progressive weight loss. The animal was subjected to routine clinical examination including abdominal ultrasonography. Whole blood was collected for hematology and serum biochemistry. However, the dog died after a week and the carcass was presented to the Department of Veterinary Pathology, VCRI, Namakkal for postmortem examination. A detailed necropsy was carried out and gross lesions were recorded. The organs showing lesions were collected in 10% formalin for histopathological examination. The sections were cut at 4µ thickness and were subjected to routine H&E staining and special staining *viz.*, Masson's trichrome and Van Gieson's stainings.

How to cite this article : Sivaraj, S., Arulmozhi, A., Sasikala, M., Gopal, K., Balachandran, P., Srinivasan, P. and Monisha, D.C. 2025. Polycystic kidney disease in a Labrador dog. Indian J. Vet. Pathol., 49(3) : 238-241.

In the present case, clinical examination of the dog revealed that the animal was lethargic with stunted growth, anaemic, dehydrated with intermittent vomition. Hematology revealed severe anaemia and dehydration (Table 1), while serum biochemistry revealed higher levels of serum creatinine and BUN along with

hyperphosphatemia and hypocalcemia (Table 2). Abdominal ultrasonography revealed the presence of multiple thin-walled anechoic cysts in both the kidneys. Based on the clinical signs, clinical pathology and ultrasonography, the condition was diagnosed as renal disease and the dog died within a week.

On necropsy, the carcass appeared emaciated with pale mucous membranes and ulcers in the oral cavity. The jaw bones were soft and pliable. Internal examination of the carcass revealed pale viscera; rounded up heart with left ventricular hypertrophy; lungs were leathery and revealed atelectatic changes; Liver showed mild hepatomegaly with rounded borders; Gastric and intestinal mucosa were haemorrhagic and showed area of ulcerative changes.

The kidneys were distorted in shape, shrunken and pale with adherent capsule. There were numerous, whitish, irregular and varying-sized fluid-filled cysts studded over the entire renal parenchyma (Fig. 1). The sagittal section of both the kidneys revealed poorly demarcated cortex and medulla (Fig. 2). The urinary bladder exhibited severe thickening of mucosa.

Table 1. Hemogram of the dog affected with Polycystic kidney disease.

Parameters	Results	Units	Reference range
Creatinine	19.5	mg/dl	0.5 - 1.8
BUN	185.6	mg/dl	8 - 28
Total protein	5.6	g/dl	5.4 - 7.1
Albumin	2.5	g/dl	2.3 - 3.3
Calcium	6.6	mg/dl	9.0 - 11.7
Phosphorus	18.9	mg/dl	2.6 - 5.3

Table 2. Serum biochemistry of the dog affected with Polycystic kidney disease.

Parameters	Results	Units	Reference range
Hb	3.6	g/dl	12.0 - 19.0
PCV	11	%	37 - 57
RBC	1.71	$x10^6/\mu L$	5.0 - 9.0
WBC	7.44	$x10^3/\mu L$	5 - 15

Histopathological examination of kidneys showed a large amounts of fibrous connective tissue containing many circular to ovoid irregular cysts with thin walls (Fig. 3). Most of the cysts were empty and few were filled with an eosinophilic homogenous proteinaceaous substance. The cysts were lined with a single layer of flattened squamous to low cuboidal epithelial cells. In addition, there were dilated tubules with atrophied glomeruli, immature tubules and proliferative arteriole in the dysplastic area (Fig. 4). Dilated renal tubules were surrounded by extensive collagenous fibrous tissues which were well demonstrated as blue (Fig. 5) and bright red colour fibers (Fig. 6) in Masson's trichrome and Van Gieson's stain respectively.

Histopathological examination of lungs revealed peribronchiolar fibrosis and desquamation of bronchiolar epithelium into the lumen. Most of the alveoli were

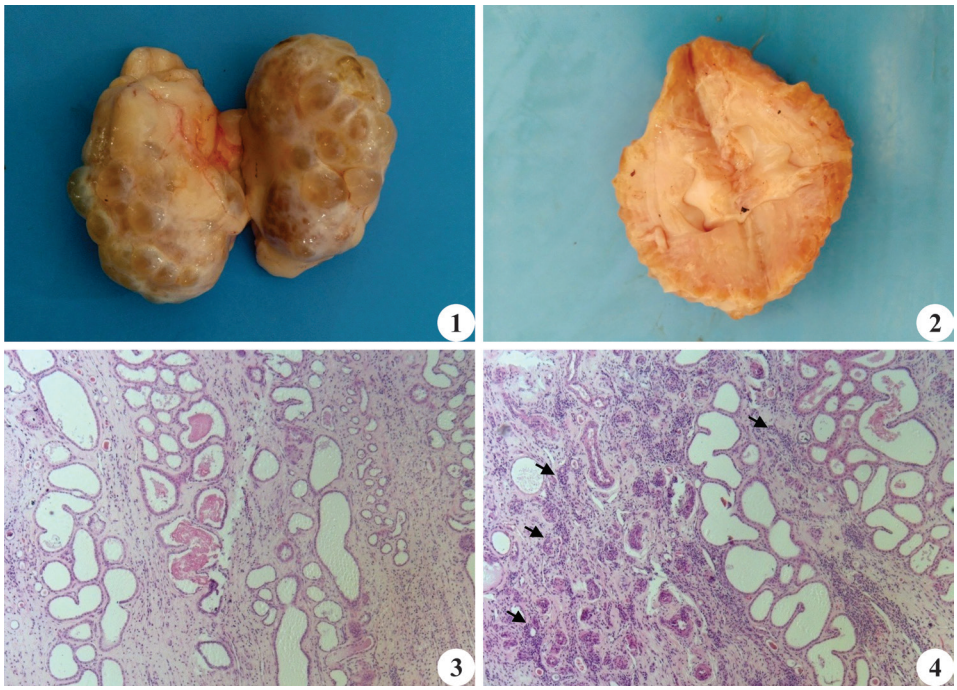

Fig. 1. Kidneys showing numerous, irregular and varying sized fluid-filled cysts; **Fig. 2.** Irregular left kidney showing reduced cortex; **Fig. 3.** Large amounts of fibrous connective tissue in the interstitium surrounding the irregular dilated cysts containing acidophilic material (H&E x40); **Fig. 4.** Dilated tubules with the immature glomeruli (arrows) with inapparent capillary lumina and tubules (H&E x40).

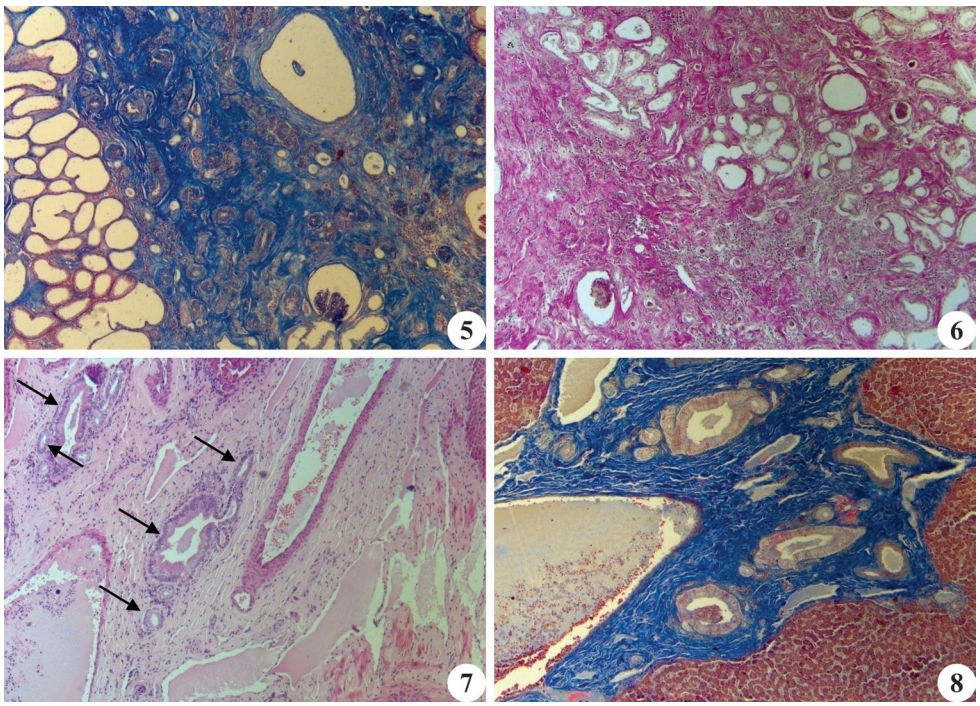

Fig. 5. Kidney showing extensive fibrous tissue proliferation in the interstitium (blue) with dilated renal tubules and atrophied glomeruli (Masson's trichrome stain x40); **Fig. 6.** Kidney showing abundant fibrous stroma (bright red) with dilated tubules replacing the normal renal parenchyma (Van Gieson's stain x40); **Fig. 7.** Liver showing replacement of hepatic tissue with fibrous tissue and bile duct proliferation (arrows) (H&E x40); **Fig. 8.** Liver showing severe periportal fibrosis (blue) and bile duct proliferation (Masson's trichrome stain x40).

collapsed and a few compensatory emphysematous areas were observed. However, there was no significant lesions noticed in the cardiac myofiber.

In liver, replacement of normal hepatic tissue with fibrous tissue proliferation and bile duct hyperplasia (Fig. 7) were noticed. The portal fibrosis was evidenced by blue coloured area in Masson's trichrome staining (Fig. 8). Fibrous tissue proliferation with ulcers were also noticed in the gastric and intestinal mucosa.

Stomach revealed loss of superficial epithelium and thickening of lamina propria with fibrous tissue proliferation. There were destruction of gastric glands and replacement with fibrous tissue noticed. There was severe destruction and desquamation of intestinal villi epithelium as well as crypts of Lieberkühn.

Polycystic kidney disease may occur due to heritable conditions[3] or certain chemicals[4] or defects in the tubular basement membrane[5]. Anaemia and decreased hemogram noticed in the present study was very well correlated with the renal lesions as erythropoietin production is hampered by the polycystic kidneys[6].

Progressively deterioting kidneys fails to excrete the metabolic end products such as urea, creatinine and phosphorus[7] which results in azotemia due to increased BUN and creatinine, hyperphosphatemia and subsequent hypocalcemia. The resultant alteration of the serum calcium and phosphorus level might have attributed to the soft, pliable and rubbery nature of the jaw bones which had been observed in the present case[8]. Similarly the excess accumulation of the nitrogenous waste substance *viz.*, urea and creatinine stimulates the chemoreceptor trigger zone, which irritates the intestinal mucosa resulting in frequent vomition and progressive weight loss noticed in the present case[9].

Microscopically, the large amounts of fibrous tissue with numerous dilated cysts observed can be well correlated with the inflammatory changes within the interstititum and subsequent fibrosis due to prolonged irritation, triggered by the chemical mediators such as cytokines, lymphokines and chemotactic factors[10].

To conclude, in polycystic kidney disease, a large area of the functional renal tissue is replaced by fibrous tissues and dilated tubular cysts resulting in end-stage renal failure mimicking the pathological changes occurring in the chronic renal failure of stage 4 leading to irreversible damage to the kidneys, resulting in anaemia due to the diminished production of erythropoietin and renal osteodystrophy with imbalanced serum calcium and phosphorus levels, finally culminating in the unfavourable prognosis.

Financial support & sponsorship: None

Conflicts of Interest: None

Use of Artificial Intelligence (AI)-Assisted Technology for manuscript preparation: The authors confirm that

there was no use of AI-assisted technology for assisting in the writing of the manuscript and no images were manipulated using AI.

REFERENCES

1. Wills JS, Barrett EL, Barr FJ, Bradley KJ, Helps CR, Cannon JM and Gruffydd Jones TJ. 2009. Evaluation of the repeatability of ultrasound scanning for detection of feline polycystic kidney disease. *JFMS* **11:** 993-996.
2. McKenna SC and Carpenter JL. 1980. Polycystic disease of the kidney and liver in the Cairn Terrier. *Vet Pathol* **17:** 436-442.
3. Zachary JF. 2022. Pathologic basis of veterinary disease. 7th edn, St. Louis, MO. pp. 724-725.
4. Evan AP, Gardner Jr KD and Bernstein J. 1979. Polypoid and papillary epithelial hyperplasia: A potential cause of ductal obstruction in adult polycystic disease. *Kidney Int* **16:** 743-750.
5. Welling LW and Grantham JJ. 1972. Physical properties of isolated perfused renal tubules and tubular basement membranes. *J Clin Invest* **51:** 1063-1075.
6. Polzin DJ. 2011. Chronic kidney disease in small animals. *Vet Clin North Am Small Anim Pract* **41:** 15-30.
7. Davis EM. 2015. Oral Manifestations of Chronic Kidney Disease and Renal Secondary Hyperparathyroidism: A Comparative Review. *J Vet Dent* **32:** 87-98.
8. Silver J and Naveh-Many T. 2013. FGF-23 and secondary hyperparathyroidism in chronic kidney disease. *Nat Rev Nephro* **9:** 641-649.
9. Polzin DJ. 2011. Chronic Kidney Disease. In: Bartges J and Polzin DJ (Eds). Nephrology and Urology of Small Animals. Hoboke: Wiley-Blackwell, pp. 433-471.
10. Cowley Jr BD, Gudapaty S, Kraybill AL, Barash BD, Harding MA, Calvet JP and Gattone II VH. 1993. Autosomal-dominant polycystic kidney disease in the rat. *Kidney Int* **43:** 522-534.