

Pakistan Veterinary Journal

ISSN: 0253-8318 (PRINT), 2074-7764 (ONLINE) Accessible at: www.pvj.com.pk

## CASE REPORT

# Clinical and Histopathological Findings of Renal Dysplasia in a Miniature Poodle Dog

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#### ARTICLE HISTORY (14-261) A B S T R A C T

Received: May 25, 2014 Revised: March 09, 2015 Accepted: March 11, 2015 Key words: Dog Renal biopsy Renal dysplasia

An 18-month-old, spayed female, miniature Poodle dog was presented for preoperative evaluation of severe azotemia prior to surgical correction of bilateral medial patellar luxation. On physical examination, the dog appeared emaciated and less than 5% dehydrated. Results of hematological analyses indicated leukopenia, azotemia, and hypercalcemia. Additionally, urinalysis revealed proteinuria and low specific gravity compatible with chronic kidney disease (CKD). Ultrasonography showed bilaterally small irregular kidneys. To make a definitive diagnosis, renal biopsy was performed under the guidance of ultrasound. Histopathology revealed a high percentage of fetal glomeruli, which was consistent with congenital renal dysplasia. This case describes clinical and histopathological features of congenital renal dysplasia diagnosed by percutaneous renal biopsy.

©2015 PVJ. All rights reserved **To Cite This Article:** Cho JH, SG Kim, MH Kang, CM Lee, WJ Kim, DW Song and HM Park, 2015. Clinical and histopathological findings of renal dysplasia in a Miniature Poodle dog. Pak Vet J, 35(3): 394-396.

# INTRODUCTION

Familial renal disease, including renal dysplasia, primary glomerulopathies, polycystic kidney disease, amyloidosis and glomerulonephritis are common causes of renal failure in juvenile dogs (Greco, 2001). It is well known that most renal diseases are progressive and ultimately fatal. However, the severity of disease and the rate of progression vary among dogs and cats. The onset of clinical signs usually occurs between four weeks and five years of age, with a mean age of two years (Greco, 2001). Renal dysplasia is one of the most common congenital or inherited renal anomalies in dogs, and has been reported in several canine breeds, including Lhasa Apso, Shih Tzu, Standard Poodle, Chow Chow, Great Dane, Samoyed, Alaskan Malamute, Golden Retriever, and Boxer (Hoppe et al., 1990). It is believed that renal dysplasia may result either from congenital defects (abnormal differentiation of renal tissue) or from neonatal injury during nephrogenesis in the first few weeks of life. To our knowledge, this case first describes renal dysplasia in a miniature Poodle regarding clinical and histopathological features and findings diagnosed by percutaneous renal biopsy.

## CASE PRESENTATION

**History and clinical examination:** An 18-month-old, spayed female miniature Poodle presented with a bilateral

medial patellar luxation. On physical examination, the dog appeared underweight and less than 5% dehydrated, and had a history of displaying a poor appetite accompanied by polyuria for several months. Hematologic examinations, serum biochemical analyses, and urinalysis revealed leukopenia, azotemia, and hypercalcemia. Urinalysis showed isosthenuria, and proteinuria (Table 1), but no abnormalities in the urine sediment. Abdominal ultrasound revealed bilaterally small irregular kidneys with pyelectasis. Further, the renal cortex was hyperechoic and had extremely poor corticomedullary definition (Fig. 1A and B).

**Differentials and diagnosis:** To differentiate between renal dysplasia, glomerulopathies, and amyloidosis, a renal biopsy was performed for definitive diagnosis. For histopathology, a percutaneous ultrasound-guided renal biopsy was collected from the left kidney with an 18gaugebiopsy needle (Pro-Mag<sup>TM</sup> biopsy needle, Angiotech, Surgical specialties corporation, Vancouver, Canada). The biopsy was performed according to a routine procedure under general anesthesia using 2% isoflurane (Terrell®, Hana Pharm, Korea)to achieve complete immobilization.

The renal tissue was stained with hematoxylin and eosin (H & E), periodic acid-Schiff (PAS), and Congo red stains. Overall, the proportion of abnormal glomeruli was significantly increased, with approximately 50% of the glomeruli appearing abnormal in the specimen.



Fig. 1: Kidney ultrasound images from a miniature Poodle with renal dysplasia. Both right (A) and left (B) kidneys were small, asymmetrical, hyperechoic and irregular in shape. The renal cortex and medulla was diffusely hyperechoic and there is poor corticomedullary definition. Bilateral pelvic dilation was also observed (arrowheads). Renal length was 2.1cm (A) and 2.4cm (B), respectively.



Fig. 2: Histology of a left kidney biopsy sample: A) Notice several immature glomeruli with dilated Bowman's capsule (black arrows), B) Retracted glomerular tufts with cystic dilation of Bowman's capsule are marked in higher magnification. Scattered infiltrates of mononuclear cells and interstitial fibrosis are also observed. C & D) PAS staining delineates basement membrane (a white arrowhead). A & B and C & D: H & E and PAS staining.

Microscopically, immature or shrunken glomeruli with a dilated Bowman's capsular space and shrunken renal tuft were observed, and scattered infiltrates of mononuclear cells were seen between glomeruli. Secondary degenerative and inflammatory lesions including interstitial fibrosis, cystic glomerular atrophy, and interstitial nephritis were also noted (Fig. 2A-D). The results of the Congo red staining to rule out renal amyloidosis revealed.

Table I: Profiles	of	complete	blood	counts	and	serum	biochemistry
from a miniature	Poc	odle dog wi	ith rena	al dyspla	sia		

Parameter (unit)	Value	Reference range	
Complete blood count			
White blood cells $(x10^{9}/L)$	5.55	6~17	
Red blood cells (x10 <sup>12</sup> /L)	6.6	5.5~8.5	
Hematocrit (%)	43.59	37~55	
Hemoglobin (g/dL)	12.5	12~18	
Electrolytes			
Sodium (mmol/L)	148	4 ~ 52	
Potassium (mmol/L)	3.8	3.8~5.0	
Chloride (mmol/L)	109	102~117	
Serum chemistry			
Blood urea nitrogen (mg/dL)	114	8~26	
Creatinine (mg/dL)	4.3	0.5~1.3	
Calcium (mg/dL)	16.9	8.8~11	
Ionized calcium (mmol/L)	1.45	1.17-1.40	
Phosphorus (mg/dL)	5.3	3~6.2	
Urinalysis			
Urine specific gravity	1.010	1.015-1.045	
Urine protein <sup>*</sup>	+	0 to 4+	

<sup>\*</sup>I + Urine protein indicates mild proteinuria with less than 30mg/dL.

#### DISCUSSION

Renal dysplasia is generally associated with clinical manifestations that are to those in other forms of renal failure in dogs and cats (Seiler *et al.*, 2010). The clinical diagnosis is made based on the presence of azotemia and low urine specific gravity at the early ages. In some cases, proteinuria is observed, but not a common finding (Vaden *et al.*, 2013).

As described earlier, two different clinical types of renal dysplasia have been reported in Shih Tzu dogs (Hoppe *et al.*, 1990). One type was in good health but laboratory tests revealed mild abnormalities that indicated renal disease. In this type of renal dysplasia, renal failure progressively developed and was associated with increasing age. Consequently, the dogs may survive for several years following diagnosis. The second type of renal dysplasia occurred most often in young dogs associated with severe renal failure, and dogs that presented with the clinical signs usually did not respond to supportive therapy, and died within a few days. The dog reported here is considered to have the former type of renal dysplasia, based on the observations of mild hematological abnormalities and clinical manifestations.

Although clinical findings, clinicopathology, and ultrasonographic findings often indicate renal disease, definitive diagnosis of renal dysplasia is dependent on the histopathological evaluations by biopsy or post mortem examination (Hoppe et al., 1990). A renal biopsy is necessary for the definitive diagnosis in numerous renal diseases. In fact, it was reported that a wedge biopsy has higher quality and therefore is appropriate for focal cortical lesions compared to a needle biopsy. However, a needle biopsy is less invasive, and frequently used in veterinary medicine despite limitations related to the quality of the specimen, which may vary depending on the experience of the operator (Vaden et al., 2005). Additionally, the results of needle biopsies are usually satisfactory, especially when expected lesions are diffusely distributed in the renal cortex, as was the dog in the current report (Lees et al., 2011). The previous report indicated that complications associated with renal biopsy are infrequent, but are more likely to occur in severe

azotemic patients, and thus caution should be taken during anesthesia or sedation (Vaden *et al.*, 2005).

Definitive diagnosis of renal dysplasia requires the presence of characterized microscopic features that include asynchronous differentiation of nephrons which refers to fetal or immature glomeruli, persistent mesenchyme and metanephric ducts, atypical tubular epithelium, and dysontogenetic metaplasia (Greco, 2001). Further, observation of at least one of these primary lesions is required in order to make a diagnosis of renal dysplasia. In the present case, we could find immature glomeruli among five primary features of dysplasia, which made a definitive diagnosis of renal dysplasia possible. According to the reports described previously, among 45 renal dysplasia dogs, forty (89%) had fetal glomeruli, and four (9%) had only one lesions of fetal glomeruli (Morita et al., 2005). Although the dog in the current case displayed only one primary dysplastic feature, the percentage of fetal glomeruli was elevated, which could reflect the severity of the disease (Seiler et al., 2010). In advanced stages, it appears that secondary degenerative and inflammatory lesions such as interstitial fibrosis or nephritis become apparent, which is consistent with those in this case (Hoppe et al., 1990).Additionally, to differentiate renal amyloidosis, renal biopsy sample with Congo red staining was negative. Therefore, the dog was diagnosed as renal dysplasia based on the microscopic evidence.

In conclusion, this case first describes of the clinical findings and histopathological features of renal dysplasia in a miniature Poodle dog through percutaneous renal biopsy using ultrasound guidance.

**Authors' contribution:** CJH and PHM designed this work. CJH, KSG, LCM and KWJ contributed with the clinical diagnosis, treatments and assessments. SDW participated in tissue processing and performing the histopathology analysis. The manuscript was prepared by CJH under the supervision of PHM. KMH and HMP contributed to coordinating and reviewing the whole process. All authors revised the manuscript and approved the final version.

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