



## CASE REPORT

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### Chronic Renal Failure Associated with Polycystic Kidney Disease in a Persian Cat

**Kenan Çağrı TÜMER**  
**Mustafa İSSİ**

Firat Üniversitesi,  
Veteriner Fakültesi,  
İç Hastalıkları Anabilim Dalı,  
Elazığ, TÜRKİYE

Feline polycystic kidney disease is a hereditary disease that effect especially Persian and Persian related cats. In this case report, it is aimed that presenting of the clinical, hematological, biochemical and ultrasonographic findings of chronic renal insufficiency associated with polycystic kidney disease in a Persian cat. A 3 years old, 1.8 kg male Persian cat was presented to Firat University Animal Hospital with a history of 2 week duration polyuria, polydipsia, severe weight loss and vomiting. Chronic renal failure associated with polycystic kidney disease was diagnosed. The supportive treatment was initiated but the cat died after 2 weeks. The necropsy was not performed because of cat's owner didn't allow. In conclusion, polycystic kidney disease should be considered in Persian cats that is brought with complaints of vomiting, weight loss, polyuria and polydipsia and it is thought that it may be useful to determine the prevalence of polycystic kidney disease in Persian and other egzotic cats in Turkey.

**Key words:** Cat, polyuria, polydipsia, polycystic kidney disease, weight loss

#### Bir İran Kedisinde Polikistik Böbrek Hastalığı ile İlişkili Kronik Renal Yetersizlik

Feline polikistik böbrek hastalığı, özellikle İran kedileri ve İran kedileriyle ilişkili diğer ırklarda görülen kalıtsal bir hastalıktır. Bu olgu sunumuyla; İran ırkı bir kedide polikistik böbrek hastalığına bağlı olarak şekillenen kronik böbrek yetersizliğinin klinik, hematolojik, biyokimyasal ve ultrasonografik bulgularının paylaşılması amaçlanmıştır. Bu olgunun materyalini 2 haftadır devam eden poliüri-polidipsi, aralıklı kusma, şiddetli kilo kaybı ve iştahsızlık şikayeti ile Firat Üniversitesi Hayvan Hastanesi İç Hastalıkları Kliniği'ne getirilen 3 yaşında, erkek, 1.8 kg ağırlığında bir İran kedisi oluşturdu. PKD ile ilişkili kronik renal yetersizlik tanısı koyulan kediye destekleyici tedaviye başlandı fakat kedi 2 hafta sonra öldü. Hayvan sahibinin izni olmadığı için nekropsi gerçekleştirilemedi. Sonuç olarak kilo kaybı poliüri ve polidipsi şikayeti ile getirilen İran kedilerinde polikistik böbrek hastalığının ayırıcı tanı tablosu içerisinde düşünülmesinin ve ülkemizde İran kedilerinde ve diğer egzotik kedilerde hastalığın prevalansının ortaya koyulması için yapılacak çalışmaların faydalı olabileceği düşünülmektedir.

**Anahtar Kelimeler:** Kedi, poliüri, polidipsi, polikistik böbrek hastalığı, kilo kaybı

#### Introduction

Feline polycystic kidney disease (PKD) is a hereditary disease that affect especially Persian and Persian related cats (1). It is thought that this disease is associated with a mutation in the PKD1 gene that encodes Polycystin-1 which are the glycoprotein of the renal tubulus and responsible for cell proliferation and cell differentiation (2, 3).

PKD is characterized by the formation of cysts especially in the kidney and less frequently in the liver and pancreas (4, 5). Affected cats can survive for a long time without any clinical symptom (6). Because the cysts enlarge by time, they cause pressure on the kidney parenchyma and irreversible function loss occurs in the kidneys (7). Clinical signs of this disease are nonspecific and usually it is associated with chronic renal failure (4). The most common symptoms of disease are weight loss, depression, vomiting, polyuria-polydipsia, anemia and abdominal enlargement (8).

PKD can be diagnosed as noninvasively with high confidence by ultrasonography (7, 9). The sensitivity of ultrasound examination in the diagnosis of PKD was reported as 75% and 91%, in 16 weeks and 36 weeks cats respectively (10). The cysts can be seen as anechoic and distinct border, round or oval structures in the ultrasonographic examination of kidney (11).

In this case report, it is aimed that presenting of the clinical, hematological, biochemical and ultrasonographic finding of chronic renal injury associated with PKD is reported in a Persian cat.

#### Case History

A 3 years old, 1.8 kg weight, male Persian cat was presented to Firat University Animal Hospital with a history of 2 week duration polyuria, polydipsia, severe weight loss and vomiting.

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#### Yazışma Adresi Correspondence

**Kenan Çağrı TÜMER**  
Firat Üniversitesi,  
Veteriner Fakültesi,  
İç Hastalıkları Anabilim Dalı,  
Elazığ - TÜRKİYE

**kctumer@firat.edu.tr**

Mild depression, severe weight loss, moderate dehydration, low body temperature (36.5 °C), elevated heart rate (164 beat/min), normal respiratory rate (32 breath/min), halitosis, ulcerative areas on the tongue and gingivitis was detected in the clinical examination. Left kidney was enlarged and irregular structure was detected at palpation.

The blood was taken from vena cephalica antebrachii for hematological, blood gas and serum biochemical analysis. Hematological examination of blood was performed with a fully automated blood analyzer (Prokan PE-6800 Vet, China). Blood gas analysis were performed with venous blood gas analysing system (Abl-80, Radiometer Medical ApS, Denmark). Biochemical analysis were performed in Firat University Hospital Central Laboratory with automated biochemical analysing system (Advia Centaur, Siemens, Germany). Hematological, blood gas and electrolyte analysis revealed leukocytosis, normocytic normochromic anemia, acidemia, hypernatremia and high anion gap (Table 1). Serum biochemical analysis revealed hypoproteinemia, hypoalbuminemia, hyperphosphatemia, severe uremia and severe creatinaemia (Table 2).

Sterile urinary catheter (Buster Cat Catheters, Kruuse, Denmark) was administrated to collecting urine sample. Urine specific gravity was determined with brix refractometer (ATAGO, Japan). Decreased urine specific gravity (specific gravity: 1010) and proteinuria was detected in urinalysis with urinary test strip (Krulab Test Strip for Urine, Kruuse, Denmark).

The cat underwent abdominal ultrasonographic examination with micro convex transducer (GE 8C-RS, LOGIQ Book XP Vet, General Electric). Anechoic, round and distinct border structures were detected in the both kidneys (Figure 1).

The final diagnosis of cat was stage 4 chronic renal failure associated with PKD. The stage of chronic renal failure was evaluated as stage 4 according to International Renal Interst Society (IRIS) classification system.

The treatment was initiated with 250 mL 0.9% NaCl solution and vitamin complex (3 mL/day p.o, Supravit, Bayer). Additionally, dietary supplements for supporting renal function (1 g/5 kg twice a day, Ipakitine, Novakim, Turkey) and prescription diet that contains low phosphorus and sodium was recommended. But the cat died after two weeks. The necropsy was not performed because of cat's owner didn't allow.

## Discussion

PKD is a progressive hereditary disease that result from a mutation on the autosomal dominant gene and effects especially Persian and Persian related cats (3, 12).

The prevalence of PKD is 41.8% in France, 41% in Italy, 45% in Australia, 36.3% Slovenia and 49.2% in United Kingdom (7, 9, 13-15). There is no report associated with prevalence of PKD in the cat in Turkey.

**Table 1.** Haematology and venous blood gas analysis result

| Parameter                                      | Results | Reference Value (4) |
|------------------------------------------------|---------|---------------------|
| White Blood Cell ( $\times 10^3/\mu\text{L}$ ) | 20.8    | 5-15                |
| Red Blood Cell ( $\times 10^6/\mu\text{L}$ )   | 5.28    | 6-10                |
| Hemoglobin (g/dL)                              | 8.5     | 8-15                |
| Hematokrit (%)                                 | 25      | 30-45               |
| MCV (fL)                                       | 48.8    | 39-55               |
| MCHC (g/dL)                                    | 33      | 30-36               |
| pH                                             | 7.17    | 7.24-7.4            |
| pCO <sub>2</sub> (mmHg)                        | 24.4    | 29-45               |
| pO <sub>2</sub> (mmHg)                         | 38      | 35-40               |
| Sodium (mmol/L)                                | 166     | 146-156             |
| Potassium (mmol/L)                             | 4.70    | 3.7-6.1             |
| Ionized Calcium (mmol/L)                       | 1.05    | 0.9-1.3             |
| HCO <sub>3</sub> <sup>-</sup> (mmol/L)         | 8.5     | 17-24               |
| AnionGap (mmol/L)                              | 33.1    | 7-17                |

**Table 2.** Biochemical analysis results

| Parameter                        | Results | Reference Value (4) |
|----------------------------------|---------|---------------------|
| Alanine Aminotransferase (U/L)   | 21      | 25 – 97             |
| Aspartate Aminotransferase (U/L) | 37      | 7 – 38              |
| Alkaline Phosphatase (U/L)       | 6       | 14 – 71             |
| Total Bilirubin (mg/dL)          | 0.1     | 0 – 0.2             |
| Total Protein (g/dL)             | 4.9     | 6 – 7.9             |
| Albumin (g/dL)                   | 2.3     | 2.8 – 3.9           |
| Phosphorus (mg/dL)               | 16.5    | 3.2 – 6.2           |
| Glukoz (mg/dL)                   | 120     | 73 – 134            |
| Total Calcium (mg/dL)            | 5.21    | 9.4 – 11.4          |
| Blood Urea Nitrogen (mg/dL)      | 489     | 19 – 34             |
| Creatinin (mg/dL)                | 5.61    | 0.8 – 1.8           |



**Figure 1.** Anechoic, round and distinct border cysts in left kidney

Although the cysts usually present in born, clinical signs are usually observed between 3 and 10 years of age (4). The clinical signs of PKD usually related with chronic renal failure (7). The weight loss, polyuri and polydipsia, anemia, uremic stomatitis, and abdominal enlargement are frequently encountered clinical signs (4, 8). In this case, the presence of vomiting, severe weight loss, anemia, polyuria, polydipsia and age of cat were compatible with literatures.

PKD can be diagnosed noninvasively with ultrasongraphy (7, 9). The presence of anechoic cystic structures have been identified within kidneys via ultrasongraphic examination in different study (1, 6, 9). Our ultrasongraphic findings are compatible with literatures.

It has been reported that hypertension may occur and use of angiotensin converting enzymes inhibitors (ACEI) and calcium channel blockers in the management and control of hypertension in cats with chronic renal injury (16, 17). Because of the systolic blood pressure was not measured in this case, any treatment was not given for hypertension.

It is stated that hypokalemia is more common in stage 2 and stage 3 chronic renal failure but hyperkalemia is more common in stage 4 chronic renal insufficiency because decreased glomerular filtration rate and potassium retention (17, 18). Na et al. (19) reported normokalemia in a woman with chronic renal failure and primary aldosteronism. They thought that normokalemia occurred due to the balance between hypokalemia associated with primary aldosteronism and hyperkalemia associated with chronic renal failure. In our case we also detected normokalemia.

Metabolic acidosis usually occurs in chronic renal insufficiency, because the kidneys can not excrete hydrogen ions and cannot reabsorb bicarbonate (17, 18). Also, decreased tissue perfusion due to dehydration and elevated lactic acid concentration can contribute to exacerbation of metabolic acidosis (18). In this case, the blood gas analysis revealed severe metabolic acidosis.

In conclusion, PKD should be considered in Persian cats that is brought with complaints of vomiting, weight loss, polyuria and polydipsia and it is thought that it may be useful to determine the prevalence of PKD in cats in Turkey.

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