

# Perirenal Pseudocyst in Consequence of Disorders of Several Interdependent Organ Systems in a Cat

## Bir Kedide Birbirlerine Bağlı Çeşitli Organ Sistemlerinin Bozulması Sonucunda Gelişen Perirenal Psöydokist Olgusu

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### Abstract

Perirenal Pseudocyst formation is a relatively uncommon disease in cats. This paper presents a rare case of pseudocyst formation in cat because of multiple interdependent systems pathology. A 17-years-old Siberian cat suffering from hyperthyroidism for four years was presented to the Veterinary Teaching Hospital of Istanbul University with a history of anorexia, respiratory distress, and abdominal enlargement due to fluid accumulation. Hypertrophic cardiomyopathy and unilateral perirenal pseudocyst were diagnosed by echocardiography and abdominal ultrasonography, respectively. Abdominal fluid drained percutaneously was aseptic exudate with no signs of any type of neoplasia. Abdominal effusion developed after one month, and right sided nephrectomy was performed. Interstitial nephritis was diagnosed by histopathological examination. It was concluded that multiple system involvement in the form of hypertrophic cardiomyopathy, hyperthyroidism, and interstitial nephritis coupled with increasing age contributed toward formation of this perirenal pseudocyst in the cat.

**Keywords:** Cat, perirenal pseudocyst, hyperthyroidism, hypertrophic cardiomyopathy

## Öz

Perirenal psöydokist oluşumu kedilerde nadir görülür. Bu makalede bir kedide birbiriyle bağlantılı multisistemik bozukluklar sonucu ortaya çıkan perirenal psöydokist olgusu sunuldu. Anoreksi, solunum problemi, ve abdominal boşlukta sıvı birikimi şikayeti ile 17 yaşlı Sibirya kedisi İstanbul Üniversitesi Veteriner Fakültesi Araştırma hastanesine getirildi. Ekokardiyografik ve abdominal ultrasonografik incelemeler sonucunda hipertrofik kardiyomiyopati ve unilateral perirenal psöydokist varlığı tespit edildi. Direne edilen abdominal sıvı histopatolo-

## Introduction

Perirenal fluid in dogs and cats can be caused by urine leakage, haemorrhage, abscess, cyst, neoplasia, lymphoma and feline infectious peritonitis (Ochoa et al., 1999; Beck et al., 2000; Hol-

Address for Correspondence: Utku BAKIREL • E-mail: utkubak@istanbul.edu.tr Received Date: 08 December 2016 • Accepted Date: 13 April 2017 • DOI: 10.5152/actavet.2018.008 © Copyright 2018 by Official Acta Veterinaria Eurasia. Available online at www.dergipark.gov.tr/iuvfd jik incelemesi neticesinde neoplazi bulgusu içermeyen aseptik eksudat olduğu tespit edildi. 1 ay sonra abdominal efüzyon nüksetti ve sağ taraflı nefrektomi uygulandı. Histopatolojik muayene neticesinde interstisyel nefritis teşhis edildi. Hipertrofik kardiyomiyopati, hipertiroidizm ve interstisyel nefrit şeklinde çoklu sistem tutulumunun artan yaşla birlikte perirenal psödokist oluşumuna katkıda bulunduğu sonucuna varıldı.

Anahtar kelimeler: Kedi, perirenal psöydokist, hipertiroidizm, hipertrofik kardiyomiyopati

loway and O'brien, 2007). Pseudocysts are either unilateral or bilateral fluid filled sacs without epithelial lining (Chastain and Grier, 1975, McCord et al., 2008). Perirenal pseudocyst (PrPc) has been frequently reported in cats (Lemire and Read, 1998; Rishniw et al., 1998; Hill and Odesnik, 2000; McCord et al., 2008; Raf-



fan et al., 2008). Perirenal fluid is often accompanied by chronic renal failure. However the aetiology still remains unclear (Ochoa et al., 1999; Beck et al., 2000; McCord et al., 2008). There is relatively little research on the role of other disorders in the formation of perirenal fluid accumulation. It was aimed to draw attention to the presence of hypertrophic cardiomyopathy, hyperthyroidism and interstitial nephritis which might all have an effect on the formation of perirenal pseudocyst in the cat.

## Case

A 17-year old neutered male Siberian cat which had been suffering from hyperthyroidism for four years was presented to the Veterinary Teaching Hospital of Istanbul University. During the physical examination of the cat, systolic murmurs (grade IV/ VI) were auscultated from the left side of the thorax and the hyperkinetic pulse was palpated from the femoral artery. The mean systolic and diastolic blood pressure were measured as 180 and 100 mmHg, respectively. The blood cell count and blood chemistry analysis were done. The ultrasonographic examinations of the thyroid lobe and echocardiographic evaluation were performed. Hyperthyroidism and non-obstructive hypertrophic cardiomyopathy were diagnosed. Appropriate treatment including furosemide (2 mg/kg, q24h SC), spironolactone (1 mg/kg, q12h PO), ampicillin sulbactame (20 mg/kg, q12h IM) and infusion therapy, along with atenolol (6.25 mg, g12h, PO and methimazole (5 mg, g12h PO) was prescribed. The cat recovered after the treatment. Five months later, he was referred to Istanbul University Veterinary Faculty Clinic, once again with a history of hydrops ascites and inappatence, from which he recovered through initial treatment. During the physical examination, a remarkable sign of a mass was felt by palpation of the abdomen. The total blood count and serum biochemistry were analysed and abdominal ultrasonographic examination was applied.

The total blood counts were analysed using Mindray BC-2800 Vet (Shenzen, China) haematology analyser. At the initial examination, the results of total blood counts were found to be within the normal range. The biochemical profile was measured using Tokyo Boeki Prestige 24i (Tokyo, Japan) chemistry analyser. The laboratory results revealed elevations of blood urea nitrogen (BUN: 56 mg/dL) and creatinine (crea: 3.8 mg/dL) levels whereas the other biochemical parameters were unremarkable (Table). The serum thyroid profile was measured using radioimmunoassay Immulite 2000 analyzer (Siemens, Germany). The serum thyroid stimulating hormone (TSH) level was less than 0.03 ng/mL and apparent elevations in the triidothyronine (T<sub>2</sub>: 511 ng/dL), thyroxine (T<sub>4</sub> >15  $\mu$ g/dL) and free thyroxine (free  $T_{4}$  >7.0 µg/dL) concentrations were detected. Snap tests (feline Leukaemia virus (FeLV), feline immunodeficiency virus (FIV) and feline coronavirus (FeCoVtest) tests (IDEXX Laboratories, USA) were performed following manufacturer's instructions. All the results were found to be negative.

Increases in the thickness of the interventricular septum, IVSs: 1.1 cm (0.74±0.13) and IVSd: 0.6 cm (0.46±0.6) and the left ventricular posterior wall, LVPWs: 1.04 cm (0.75±0.11) and LVPWd: 0.88 cm (0.43±0.07), and decreases in the left ventricle inter diameters, LVIDs: 0.63 cm (0.88±1.8) and LVIDd: 1,2cm (1.59±0.23) were determined. The left atrial internal dimensions LA: 1.89 cm (1.21±0.18) and aorta were determined to be normal in size by means of an echocardiographic examination (Figure 1a). According to the echocardiographic examination, non-obstructive hypertrophic cardiomyopathy was diagnosed (Moise et al. 1986, Chetboul et al, 2006). By means of ultrasonography, enlargement in the left lobe of the thyroid gland was determined (20.4 mm and 8.66 mm), and no parenchymal morphological changes were observed in either kidney nor in the liver (Zwingenberger A and Wisner E, 2008). Dietary modifications, including reduced protein, phosphorus and sodium, were offered for the management of renal failure (stage 3).

After five months, a remarkable sign of the mass was apparent. The azotemia (BUN: 44 mg/dL, crea: 3.8 mg/dL) was stable. An abdominal ultrasonography showed a hyperechoic lesion to be visable in the liver parenchyma. The left kidney and other abdominal organs appeared to be normal. However, the abdominal ultrasonography showed a remarkable cystic image with an ovoid hyperechoic area inside, above the urine bladder (Figure 1b). The hyperechoic area inside the cystic structure was defined as the right kidney which was measured as 3x1.4 cm in size and surrounded by the cyst with dimensions of 11x9 cm. Consequently, a unilateral PrPc was diagnosed and percutaneous-ultrasound guided drainage was performed (Figure 1c). The laboratory analyses of the drained fluid (yellowish mostly clear) showed numerous non-degenerated neutrophils confirming inflammation. A urine sediment examination and bacterial culture were performed via cystocentesis under ultrasound guidance and mild leucocytosis was found in the urine sediment examination. In the bacterial culture of the urine, Staphylococcus aureus was cultivated and Amoxicillin was detected as a sensitive antibiotic.

After one month, the fluid reaccumulated (Figure 1d). The right kidney inside the cystic structure was too small and seemed to be deformed. So a decision for nephrectomy was made. Administration of 0.9% saline solutions (10 mL/kg/h IV) was started 2 hours before the surgery and cefazolin (20 mg/kg IV) was given. Atropine (0.04 mg/kg SC) and propofol (5 mg/kg IV) were given for premedication and the anesthesia was induced with isofluran in oxygen 30–50 %. The large cystic structure and the right kidney were removed with surgical procedure (Fossum TH, 2002). The cyst with semi-translucent capsule was filled with a yellowish fluid. By means of laboratory analyses it was detected that the cystic fluid (230 mL) was aseptic exudate.

Histopathological evaluation of the right kidney revealed chronic interstitial nephritis. There was a diffuse mononuclear cell infiltration in the cortex. The tubules were severely dilated

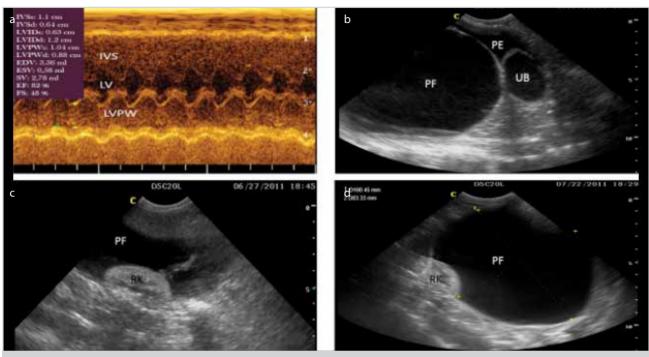


Figure 1. a-d. Echocardiographic and ultrasonographic imagines. (a) M-Mode echocardiographic image of left ventricle. (b) Abdominal ultrasonography of PrPc. (c) Imaging after percutaneous-drainage of perirenal fluid. (d) Fluid reaccumulation after one month d: diastolic; s: systolic; IVS: inter ventricular septum; LVID: left ventricular internal dimension; LVPW: left ventricular posterior wall; EDV: end-diastolic volume; ESV: end-diastolic volume; SV stoke volume; EF: ejection fraction; FS: fractional shorting; PF: perirenal fluid; PE: peritoneal effusion; UB: urine bladder; RK: right kidney

and separated by inflammation and fibrous connective tissue. The Bowman capsules were dilated as well and the glomerular tufts were atrophic (Figure 2a). The walls of the pseudocysts were composed of multi-layered bands of connective tissue. Portions of the connective tissue contained mild infiltrates of lymphocytes. No epithelium was noted (Figure 2b). The histopathological diagnosis was chronic interstitial nephritis and perirenal pseudocyst formation.

Initially, the renal failure progressed after the operation. Azotemia level (Table 1) and clinical signs such as inappetence, nausea, excessive urination became more severe. Treatment was started with IV infusion combined with lactated Ringer's solution in 5% dextrose (90 mL), 0.45% sodium chloride solution (80 ml), ampicillin sulbactame (10 mg/kg, q12h, IM), ranitidine (0.5 mg/kg, g12h, IM), metoclopramide (0.2 mg/kg, g12h, IM), erythropoietin (100 Units/kg 3 times weekly SC), aluminium hydroxide (30 mg/kg, q12h, PO) and force feeding with commercial food for kidney failure (Hills® K/D). After 3 days, saline solutions (200 mL, SC) were administered. The case was able to be followed for 4 months post-operatively. Although the owner of the cat did not always collaborate for proper treatment, intermittent treatment of kidney failure was able to be applied for long term. The cat lost 2.10 kg from the body weight (from 6 kg to 3.90 kg) during 4 months. There was a significant reduction in the degree of azotemia level at the 4<sup>th</sup> month of the

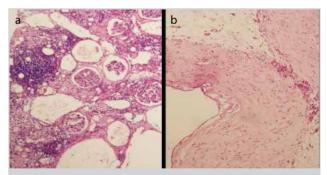


Figure 2. a, b. Histopathologic evaluation of renal cortex and pseudocyst. (a) Renal cortex shows a diffuse interstitial, predominantly mononuclear, inflammatory infiltrate. Tubules are dilated severely and separated by inflammation and fibrous connective tissue. The Bowman capsules are dilated as well and the glomerular tufts are atrophic. (b) The wall of the pseudocyst composed of multi-layered bands of connective tissue (No epithelium is noted)

treatment (Table). The cat survived five months after the surgery before being euthanized at the request of the owner.

#### Discussion

This case was presented to draw attention to the presence of disorders of several interdependent organ systems (e.g. Endo-

	Pro operation						Post operation				
Blood tests	Pre-operation						Post-operation				
	4у	Зу	2у	9m	7m*	2 m**	3d	10d	1m	3m	4 m
PCV (%)	57.6	54.3	58	38.4	37.8	31.1	20.7	24.9	28.1	29.4	23.4
Hb (g/dL)	17.8	16	16.9	12.6	12.7	10.1	6.9	8.4	9.4	9.9	7.6
WBC (10º/µL)	12.2	13.5	13.4	13.3	13.3	11.2	20.1	11.5	20.1	14	21.2
PLT (10 <sup>9</sup> /μL)	313	481	450	384	629	374	545	557	609	628	472
BUN (mg/dL)	27	51	39	47	56	44	86	89	76	166	130
Crea (mg/dL)	2.1	3.3	3.5	3.4	3.8	3.8	6.8	6.8	5.7	4.7	3.1
P (mg/dL)	5.9	5.9	5.2	3.9	5.3	5.7	6.7	8	6.6	13.7	14.9
T₄ (μg/dL)	6.2	4	10	4.8	>15	2	NA	NA	NA	2.6	6.2

Table 1. Long-term pre- and post-operative results of blood testing

PCV: packed cell volume; Hb: haemoglobin; WBC: white blood cell; PLT: platelet; y: years; m: month(s); NA: not analysed; \*at the time diagnosis of HCM; \*\*at the time of diagnosis of PrPc)

crine, urinary, and circulatory) which may have caused the formation of perirenal pseudocyst (PrPc) in the cat. Mostly chronic renal disease is implicated in the aetiopathogenesis of PrPc (Ochoa et al., 1999; Beck et al., 2000; Raffan et al., 2008). It was suggested that there might be a link between renal parencyhmal disease and pseudocyst formation. According to underlying parenchymal disease, subcapsular PrPcs are formed in cats by accumulation of transudate through the capsule and parenchyma of the kidney. Transudates can accumulate according to alterations of hydrostatic or oncotic pressure within vasculature or interstitium of an organ (Coles, 1986). Vascular drainage may be affected by interstitial fibrosis (DiBartola and Westropp, 1987). However, the relation is still unclear (Ochoa et al., 1999; Beck et al., 2000; McCord et al., 2008). Chronic interstitial nephritis was diagnosed in our case histopathologically. However, in a study carried out on the six cats and twelve dogs, interstitial nephritis was diagnosed only in one case as an underlying cause of PrPc (Holloway and O'brien, 2007).

Attention was also drawn to possible effect of hyperthyroidism and hypertension in the formation of PrPc. However, there is insufficient information on this matter. In a retrospective study on 13 cats with PrPc, the hyperthyroidism was only evaluated in 2 cats by elevation in T<sub>4</sub> concentration and palpation of thyroid nodules. No complete examination and testing for hyperthyroidism was performed in all the other cats (Ochoa et al., 1999). In our case, the hyperthyroidism was diagnosed by elevation of T<sub>2</sub> and T<sub>4</sub> levels and enlargement in the left lobe by ultrasonographic examination. Hyperthyroidism and chronic kidney disease are the possible causes of hypertension. Hypertension typically induces vascular changes, and it is tempting to speculate that pronounced vascular changes may prevent venous drainage. Hypertension leads to more arterial than venous damage (Ochoa et al., 1999). The systolic murmur (grade III/VI) was detected in the clinical examination and non-obstructive hypertrophic cardiomyopathy was detected by echocardiographic examination in this case. Hypertrophic cardiomyopathy could be the result of high blood pressure because of hyperthyroidism and renal failure. In a retrospective study on 7 cats with PrPc, systolic murmurs have been determined, despite the lack of hypertension in the 2 cats (Ochoa et al., 1999). In the other study, mild concentric hypertrophy of the left ventricle was detected in one cat with PrPc, but no information was given about thyroid function tests (Lemire and Read, 1998). In another study on 26 cats with PrPc, no underlying cardiomyopathy or hyperthyroidism was mentioned (Beck et al., 2000). At the initial examination of our case, mean systolic blood pressure was measured as 180 mmHg in our cat. This could be the reason for PrPc.

Treatment choices are either pseudocyst resection or nephrectomy, if there is no bilateral disease (Rishniw et al., 1998; Ochoa et al., 1999; Essman et al., 2000). Resection of the pseudocyst is usually an effective choice of treatment. But it does not help to prevent the progression of renal dysfunction (Beck et al., 2000; Essman et al., 2000). Surgical removal of PrPc without nephrectomy has longer survival times. As the right kidney inside the cystic structure was too small and seemed to be deformed, nephrectomy was decided for our cat. Although end-stage renal failure (stage 4) had occurred in the first month after the operation, the medical management (dehydration, systemic hypertension, metabolic acidosis, and plasma phosphate concentration) and the dietary regulation provided remission of renal failure (stage 3) within 4 months after the operation. The cat survived 5 months after which he was euthanized at the request of the owner. Beck et al., (2000) determined the mean age of survival rate to be 9 months after the surgery in cats with PrPc. The prognosis for cats with pseudocyst formation depends on the degree of renal dysfunction at the time of diagnosis (Beck et al., 2000; Morrow, 2005). In our cat, stage 3 renal failure was present at the time of diagnosis of PrPc. Even thoughstage 4 renal failure was determined just after the operation, the level of the azotemia was decreased to stage 3 due to proper treatment. Parallel to our findings, Ochoa et al.,

(1999) also observed the more severe azotemia in the nephrectomised cat with PrPc.

Abdominal ultrasound and fine-needle aspirate of the fluid may yield a definitive diagnosis for PrPc, and long-term treatment after nephrectomy may have a beneficial effect on azotemia. However, pet owners should be informed about treatment in the post-operative period and should collaborate with physicians in this regard. According to the previous studies, there is a general opinion that chronic renal failure might be the possible cause of PrPc. However, hypertrophic cardiomyopathy or cardiac problems were reported in a few cats with PrPc. Even though hypertrophic cardiomyopathy was diagnosed after the occurrence of hyperthyroidism and chronic renal failure, we believe that the existing disorders (hyperthyroidism, hypertrophic cardiomyopathy and chronic renal failure) developed in relation to one another with the effect of increasing age. Although it was not clearly stated, systemic hypertension was considered to be the factor that triggered the formation of PrPc, as the common finding of these three disorders were systemic hypertension ultimately. Yet, the aetiology is still unclear and further research is needed for the explanation of the underlying cause of PrPc.

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