Journal of Veterinary Science and Public Health

J. Vet. Sc. Public Health, v. 7, n. 2, p. 152-161, 2020

RENOMEGALY SECONDARY TO POLYCYSTIC KIDNEY DISEASE AND ASSOCIATED WITH NEUROLOGICAL SIGNS IN A CAT – CASE REPORT

RENOMEGALIA SECUNDÁRIA A DOENÇA RENAL POLICÍSTICA E ASSOCIADA A ALTERAÇÕES NEUROLÓGICAS EM UMA GATA – RELATO DE CASO

Geovanna Santana Benedito[1]; Ariel de Aguiar[2]; Ana Paula Frederico Rodrigues Loureiro Bracarense [3]; Lucas Alécio Gomes[4]*.

[1] Mestranda do Programa de Pós-graduação em Ciência Animal da Universidade Estadual de Londrina (UEL);
[2] Residente em Patologia Animal no Hospital Veterinário da Universidade Estadual de Londrina (UEL);
[3] Professor Associado do Departamento de Medicina Veterinária Preventiva da Universidade Estadual de Londrina (UEL);
[4]Professor Adjunto do Departamento de Clínicas Veterinárias da Universidade Estadual de Londrina (UEL).
*Autor para correspondência: Rodovia Celso Garcia Cid, Pr 445, km 380, Campus Universitário, Universidade Estadual de Londrina, Caixa Postal 10.011, CEP:86057-970, Londrina – PR; lucasalecio@gmail.com
DOI: 10.4025/revcivet.v7i2.52684

RESUMO: A renomegalia pode ser resultante de algumas doenças, entra elas a doença renal policística (DRP), que é uma afecção genética hereditária com caráter autossômico dominante, e se caracteriza pelo aparecimento de cistos no parênquima renal. É uma das doenças hereditárias mais comuns em gatos, sendo de maior ocorrência na raça persa. Os sinais clínicos derivam do comprometimento da função renal. O diagnóstico é realizado por meio de ultrassonografia abdominal, testes genéticos ou achados *pós mortem*. Não existe tratamento específico e a terapia consiste no controle dos sinais clínicos. O presente trabalho tem como objetivo relatar um caso de DRP em uma gata sem raça definida, atendida em um hospital veterinário escola. O tutor referia que o animal apresentava dificuldade para caminhar, vocalização e tremores. No exame físico detectou-se hipotermia, desidratação, taquicardia, hipertensão, mucosas congestas e aumento do volume abdominal. No exame neurológico observou-se alteração do estado mental (depressão), com opstótono e movimentos motores de "pedalagem" involuntários. Nos exames hemotológicos evidenciou-se azotemia e na radiografia abdominal observou-se acentuado aumento dos rins. O animal não respondeu ao tratamento e evoluiu para óbito. Na autópsia foi observado renomegalia bilateral apresentando substituição do parênquima renal por múltiplos cistos. Microscopicamente foi observado que córtex e medula renal estavam expandidos por cistos revestidos por células epiteliais cuboides a achatadas e lúmen contendo material homogêneo eosinofílico. Os achados macroscópicos e histopatológicos permitiram o diagnóstico de DRP. Sendo assim, conclui-se que em casos de aumento de volume abdominal em gatos, renomegalia e DRP devem constar nos diagnósticos diferenciais.

Palavras-chave: Rim. Hipertensão. Histopatologia. Distensão abdominal. Sinais neurológicos.

ABSTRACT: The polycystic kidney disease (PKD) is an inherited autosomal dominant genetic disorder, which is characterized by cysts in the renal parenchyma. It is one of the most common hereditary diseases in cats, being more representative in the Persian. Clinical signs derive from

impaired renal function. The diagnosis is performed by abdominal ultrasound, genetic testing or post mortem findings. There is no specific treatment and therapy consists in controlling clinical signs. The aim of the present study is to report a case of PKD in a mixed breed cat that attended at the veterinary hospital school. During the assessment, it was reported that the animal had difficulty walking, vocalization and tremors. The physical examination detected hypothermia, dehydration, tachycardia, hypertension, congested mucous membranes and abdominal distention. The neurological examination revealed altered mental status (depression), opstotone and involuntary pedaling motor movements. Complementary examinations detected azotemia, and abdominal radiography showed a marked increase in renal size bilaterally. The patient did not respond to the treatment and progressed to death. An autopsy revealed bilateral renomegaly, with multiple cysts. Microscopically, it was observed that the renal cortex and medulla were expanded by cysts covered by cuboidal to flattened epithelial cells and a lumen containing homogeneous eosinophilic material. The macroscopic and histopathological findings allowed the diagnosis of PKD. Therefore, it was possible to conclude that in cases that we have abdominal distention associated to neurological signs in cats, renomegaly and PKD should be included in the differential diagnoses.

Key words: Kidney. Hypertension. Histopatology. Abdominal distension. Neurological signs.

INTRODUCTION

Polycystic kidney disease (PKD) is an inherited genetic disorder with autosomal dominant character, characterized by the appearance of cysts in the renal parenchyma and, less frequently, in the hepatic and pancreatic parenchyma (BILLER, et al., 1996). It is one of the most common hereditary diseases in cats, most prevalent in Persian and related breeds. There is no sexual predilection as demonstrated by Domanjko-Petri et al. (2008) and Barrs et al. (2001). The prevalence of PKD in Persian cats varies between 30 and 46 %, according to studies carried out in several countries (BARRS, et al., 2001; BARTHEZ et al., 2003; BILLER et al., 1996; CANNON et al., 2001; SATO, et al., 2019). For cats of other breeds, the prevalence is 4.2 %, according to Domanjko-Petri et al. (2008). A Brazilian study shows a prevalence of 31.6 % in Persian cats and 1.1 % in cats without a defined breed, all of them longhaired (ONDANI et al., 2009). In humans, the disease presents itself in a similar way and hereditary kidney disease is more common, causing complications similar to those of cats (CORNEC-LE GALL et al., 2019). PKD is caused by a mutation in exon 29, resulting from a stop codon at position 3284, in the polycystin-1 gene (LYONS et al., 2004). The autosomal dominant characteristic is related to three types of forms (PP, Pp and pp). PP (homozygous dominant) refers to animals that do not survive for presenting a severe and lethal form of the disease that causes intrauterine death or rapid renal failure. Pp animals, which present heterozygosis, represent the cats that develop renal cysts. Finally, pp animals are cats with genotyping and negative phenotyping (SCALON and MAZZOTTI, 2016).

The formation and growth of the cysts progress slowly causing compression of the healthy renal parenchyma and the renal function gradually deteriorates. Thus, the animals range from those asymptomatic and incidentally diagnosed to those that manifest signs presented in chronic kidney disease (CKD). The main signs presented are polyuria, polydipsia, anorexia, weight loss and emesis (DOMANJKO-PETRI et al., 2008). In general, the signs of renal failure in the animal appear between 3 and 10 years of age (EATON et al., 1997), or later, around 12 years of age, according to a study by Domanjko-Petri et al. (2008). Other clinical signs are systemic arterial hypertension, renomegaly and pain. Systemic hypertension can also lead to lesions in target organs (eyes, kidneys, heart and brain). O'Neill et al. (2013) reported cases of

brain/multifocal neurological changes in cats and dogs, and three of them were associated with hypertensive kidney disease, called hypertensive encephalopathy, similar to that described in humans. Haematological and biochemical tests may show aregenerative anaemia, azotemia, hyperphosphatemia and metabolic acidosis. Urinalysis may show isosthenuric urine and proteinuria (MEUTEN, 2015).

The content of the cysts is usually a serous fluid formed by fibrin but, when infected, it may be purulent or reddish in colour if there is intra-renal bleeding. In the histopathological evaluation, cystic structures surrounded by a simple layer of cuboidal epithelial cells are observed, presenting few villi and minimal or no lesion in the surrounding tissue (SCALON and MAZZOTTI, 2016).

Abdominal ultrasonography is a very accurate diagnostic method when performed by an experienced ultrasonographer with a high resolution transducer (DOMANJKO-PETRI et al., 2008). Ultrasonographically, renal cysts appear as round, anechoic or hypoechoic cavities. The largest number of cysts are located in the cortex or between the cortex and the renal marrow (BONAZZI et al., 2009). The diagnosis of the disease can also be accomplished through genetic testing (Polymerase Chain Reaction - PCR). This method has superior sensitivity for the diagnosis of young animals, because the cysts in younger cats may be so small that they are not visualized by ultrasound (VUCICEVIC et al., 2016). Abdominal radiography can also be performed but, depending on the age of the animal, renomegaly may not yet be evident. Radiographic changes due to the formation of renal cysts are only observed if there is an increase or distortion of the renal capsule. Renomegaly that is evident in radiography has other possible differential diagnoses, such as hydronephrosis, renal lymphoma, feline infectious peritonitis, feline perinephric pseudocysts, primary or metastatic renal neoplasia, haematoma or subcapsular abscess (SEILER, 2019).

Guerra et al. (2018) determined the amount of kidney cysts necessary to confirm the diagnosis of PKD in Persian cats according to age groups. In animals aged 15 months or less, the presence of at least one kidney cyst was sufficient to establish the diagnosis. In cats aged 16 to 32 months, two or more cysts in one or both kidneys were necessary; in animals aged 33 to 49 months, at least three cysts in one or both kidneys; and in cats aged 50 to 66 months, four or more cysts in one or both kidneys were necessary to confirm the diagnosis of the disease.

A recent study carried out by researchers in human medicine on cats with PKD (YU et al., 2019) demonstrated that serial measurements of total kidney volume, total cystic and fractionated volume, performed through tomography and magnetic resonance imaging, can determine the progression of PKD and the efficiency of therapies. According to the authors, cats are efficient biological models for such estimates.

In human medicine, measurement data of total renal volume are used to predict complications related to the disease, such as azotemia, pain and hypertension, since the increase in renal volume has been correlated with the decrease in the glomerular filtration rate. Thus, people with higher renal volume are evaluated more routinely than people with lower renal volume (BAE and GRANTHAM, 2010).

There is no specific treatment for PKD (SCALON and MAZZOTTI, 2016) and the ones performed are only to control the symptoms and consequences of chronic kidney disease. They consist of hypertension control, proteinuria, hydroelectrolytic disorders, nutritional management and hormonal therapies (POLZIN, 2013). In human medicine, there are studies on specific therapies for PKD with excessive water intake, sodium and protein restricted diets, and there are also studies with drug therapies (CORNEC-LE GALL et al., 2019).

Based on the aforementioned, the aim of this study is to share information by describing the clinical and laboratory aspects of an elderly female cat, without a defined race, that presented abdominal distension, azotemia and neurological signs, associated with polycystic

renal disease. This way, this report aims to contribute to the clinical reasoning of veterinarians attending cases with similar characteristics, and to the refinement of possible differential diagnoses.

DEVELOPMENT

The subject of this study, a castrated female cat, with no defined race, approximately ten years old, 3.2 kg, shorthaired, was assisted in the emergency sector of a veterinary school hospital. In the anamnesis the owner of the animal reported that the patient had had vocalization, anorexia, difficulty walking and tremors since the morning of that day, seemed to be in pain and was less responsive to the environment. He also reported the patient presented symptoms of normophagia, normoquezia, normodipsia and increased frequency of urination, but he could not report the volume of urine.

At physical examination, the animal was hypothermic (35.8°C), tachycardic (264 beats per minute), with congenital mucous membranes, degree of dehydration of 7 to 8 %, and normal nutritional status. Abdominal palpation showed an increase in volume of firm consistency in a bilateral retroperitoneal region of approximately 10 to 15 centimetres in diameter. Systolic blood pressure was 240 mmHg (non-invasive method, portable vascular Doppler). Other physical parameters were normal. In the neurological examination, there was a decrease in the level of consciousness and mydriasis not responsive to direct and consensual pupillary reflexes to light. In the four limbs, there was absence of proprioceptive positioning, absence of hopping ability, and presence of muscle flexion, with normal patellar reflexes. The patient also presented opisthotonous, involuntary motor pedalling movements, and intermittent stiffness of thoracic limbs. In view of the neurological changes, the syndrome was classified as multifocal. The results of the CBC (complete blood count) test that was requested were normal: RBC 7.55 x 10^6/μL, hemoglobin 8.8 g/dL and hematocrit 28.6 %. In biochemical tests, increased urea (202 mg/dL), creatinine (4.3 mg/dL) and glucose (225 mg/dL) were observed. In turn, the results for alanine aminotransferase, gamma glutamyl transferase, total proteins and albumin were in accordance with the reference values.

Abdominal radiography was performed showing an increase in the kidneys size, which occupied the entire hypogastric and mesogastric region, displacing the colon medially and ventrally (Figures 1 and 2).

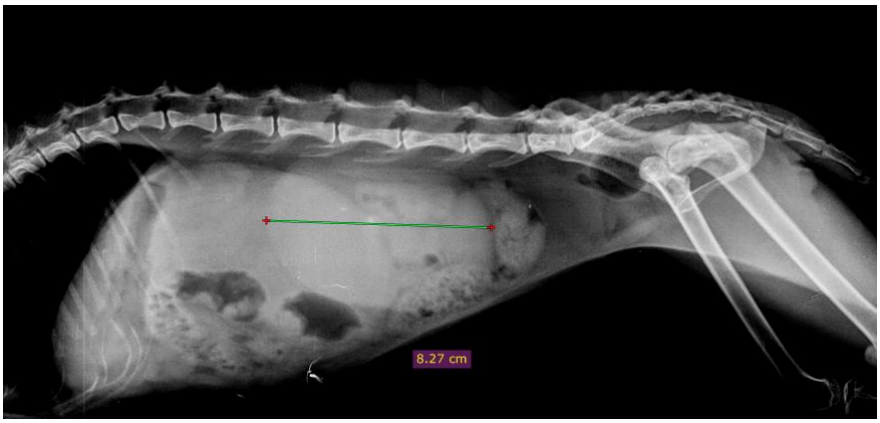

Figure 1: Abdominal radiographic image in right lateral projection of a ten-year-old female cat, with polycystic renal disease, demonstrating significant renomegaly, with the right kidney measuring approximately 8.27 cm (green line between the red dots).

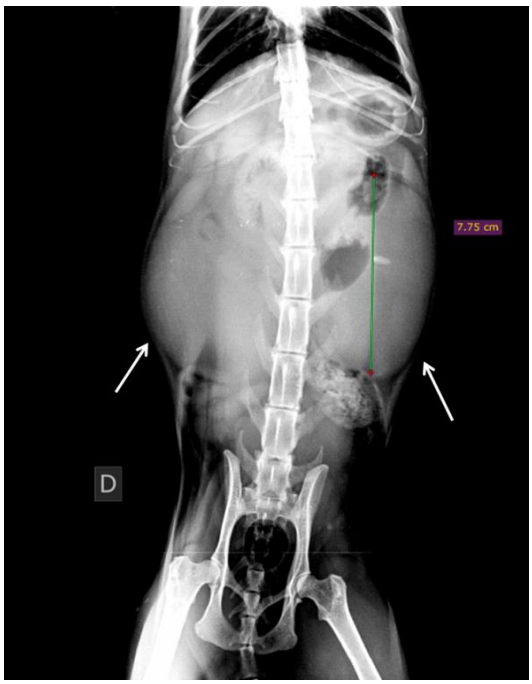

Figure 2: Abdominal radiographic image in ventral-dorsal projection of a ten-year-old female cat, with polycystic renal disease, showing bilateral renomegaly (arrows), with the left kidney measuring approximately 7.75 cm (green line between the red dots).

The animal was interned and as therapy the following medicines were administered parenteral fluid therapy with lactate ringer solution plus 3 ml of 19.1 % potassium chloride intravenously, sodium omeprazole (1 mg/kg, intravenously, every 24 hours), tramadol hydrochloride (3 mg/kg intravenously, slowly, every 8 hours), amlodipine tablets (0.39 mg/kg, orally, every 24 hours), diazepam (0.25 mg/kg, intravenously, every 8 hours), disodium phosphate of dexamethasone (0.25 mg/kg, intravenously, as a single dose), and 20% mannitol solution (1 g/kg, intravenously, for 20 minutes, as a single dose). The animal was heated and decubitus exchange took place every 6 hours. The treatment was ineffective and the animal evolved to death a few hours after hospitalization. The owner authorized the autopsy and the following alterations were identified: bilateral renomegaly with most of the renal parenchyma replaced by multiple cysts. Their sizes ranged from 1.0 to 3.0 cm in diameter, contained clear and serous fluid, with scarce renal parenchyma, limited to the subcapsular cortical region (Figure 3). Edema and pulmonary congestion were also evidenced.

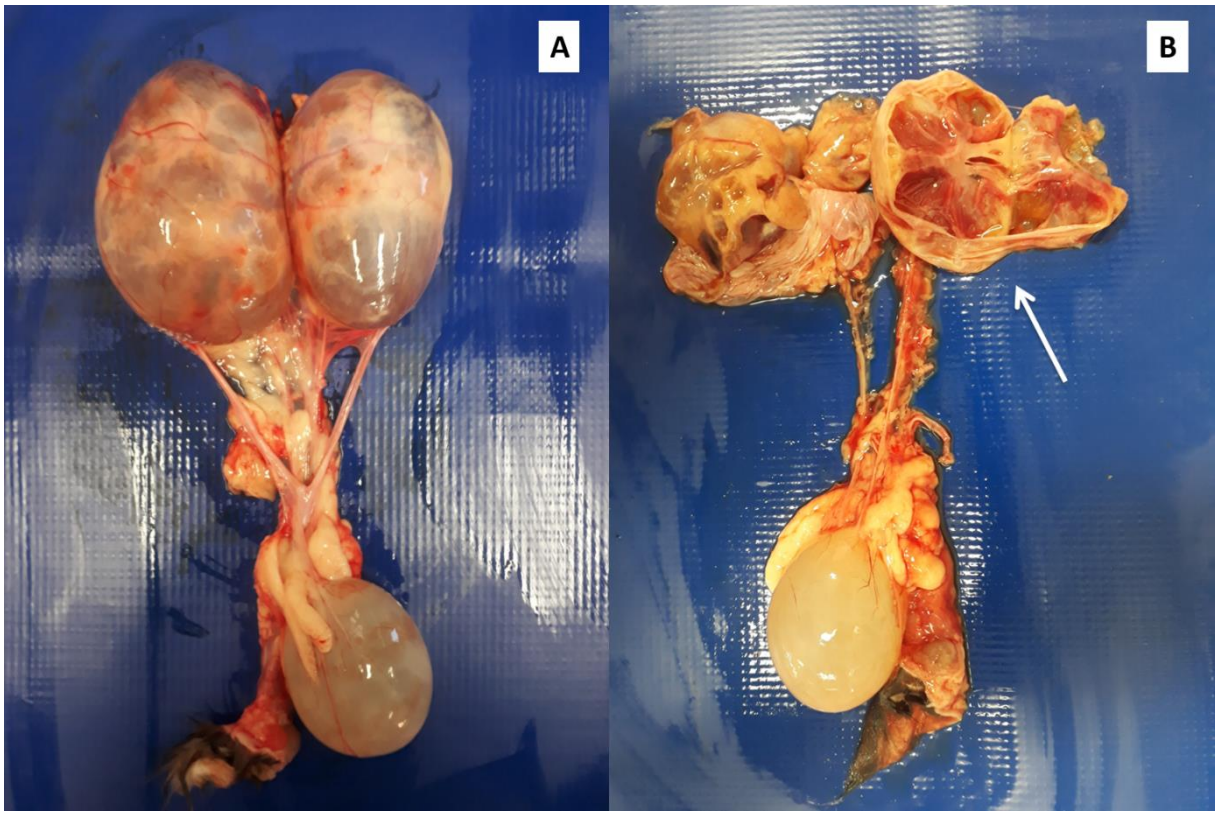

Figure 3. Macroscopic appearance of the urinary system of a ten-year-old female cat, with polycystic kidney disease during an autopsy procedure, showing (A) kidneys with prominent increase in volume and (B) longitudinal renal section (arrow).

Microscopically it was observed that approximately 60 % of the cortex and renal marrow were expanded by cysts coated by flattened cube epithelial cells (Figure 4A), and lumen containing eosinophilic homogeneous material (Figure 4B). This resulted in compression and atrophy of the adjacent tubules and ducts. Multifocal cytoplasmic vacuolization and necrosis were observed in the tubular epithelium (Figure 4C). The glomeruli had an accentuated diffuse thickening of the basal membrane with deposition of segmental eosinophilic amorphous material, being separated by protrusions of the basal membrane matrix. There was also a homogeneous eosinophilic substance in the glomerular and tubular lumen, multifocal, moderate to sharp (Figure 4D). Throughout the fragment there was moderate expansion of the interstice by fibrous conjunctive tissue; an accentuated amount of lymphocytes and plasmocytes (Figure 4D) and rare deposits of basophilic granular mineral. The macroscopic and histopathological findings allowed the diagnosis of polycystic renal disease.

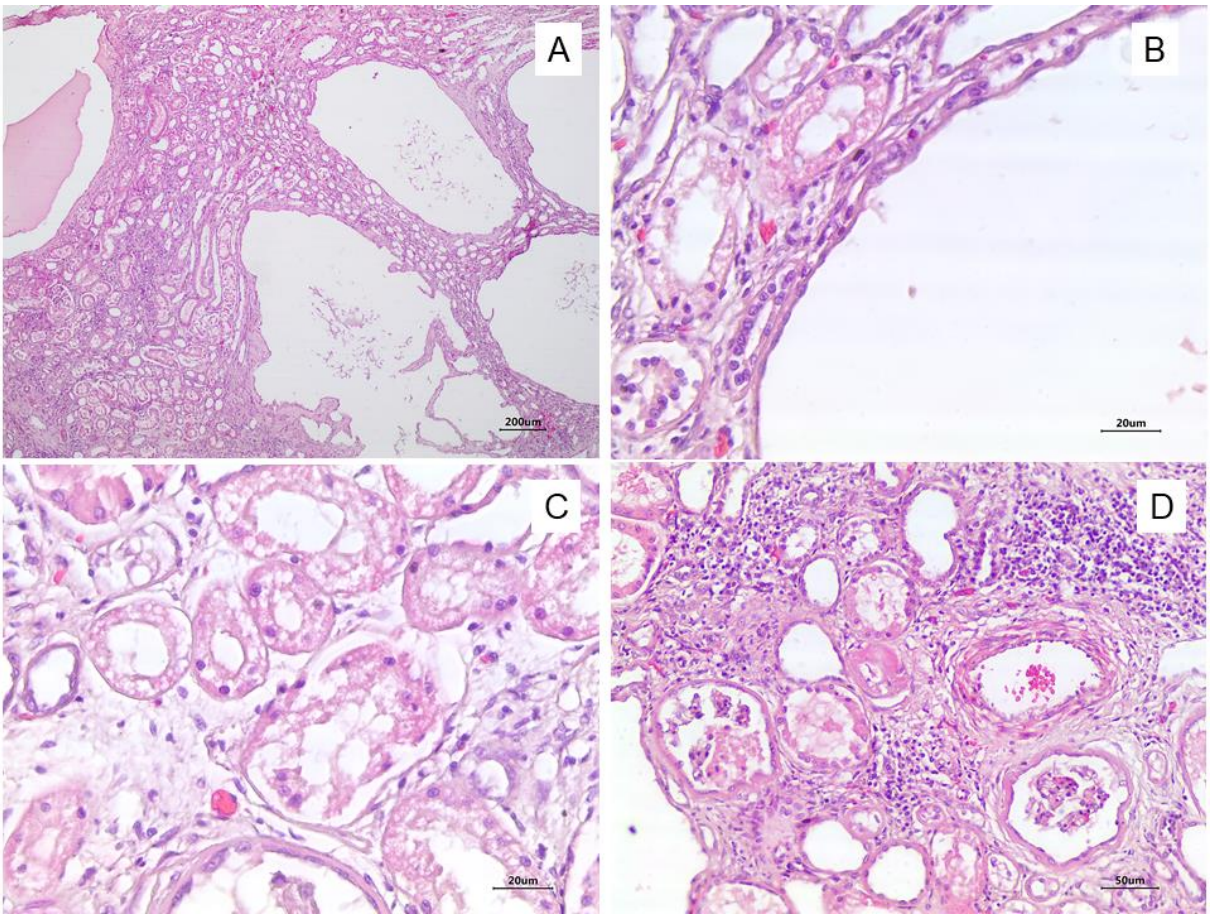

Figure 4. Feline kidney photomicrograph. (A) Multiple renal cysts with lumen containing eosinophilic material or without content. HE. Bar 200 µm. (B) Flattened tubular epithelium-coated cyst. HE. 20 µm bar. (C) Intracytoplasmic vacuolization of the tubular epithelium and tubular necrosis. HE. Bar 30 µm. (D) Thickening of the glomerular capsule, accompanied by glomerular atrophy. Multifocal lymphocytic infiltrate. HE, Bar 50 µm.

DISCUSSION

Polycystic kidney disease is a genetic hereditary condition with a higher prevalence in Persian cats and related breeds. Domanjko-Petri et al. (2008) describe a 4.2 % prevalence of the disease in animals of other breeds. A Brazilian study shows a prevalence of 1.1 % in cats without a defined breed, all of them longhaired (ONDANI et al., 2009). Given this lower occurrence, the value of case reports in animals of non-predisposed breeds stands out, emphasizing the importance of having PKD in the list of differential diagnosis in cases of renomegaly.

In this report, the female cat showed late clinical signs of the disease, at the age of ten years, consistent with other studies (EATON et al., 1997; (DOMANJKO-PETRI et al., 2008). In addition, there were no classical signs of CKD such as polydipsia, emesis or weight loss. The owner reported increased frequency of urination, but could not inform about the volume of urine and it was not possible to differentiate pollakiuria from polyuria.

The patient had systemic arterial hypertension. In the macroscopic analysis of the encephalon, no alterations were found, thus, the hypothesis of hypertensive encephalopathy is suggested as a possible cause of neurological alterations. This justified the treatment with amlodipine due to the suspicion of hypertensive encephalopathy. Proprioceptive deficits can be justified by the depressed state of consciousness in which the patient was. Volta et al. (2010)

also describe a case of PKD with concomitant hypertension, initially treated with enalapril maleate. This drug, however, only controlled systemic blood pressure when associated with amlodipine. O'Neill et al. (2013) report four cases of hypertensive encephalopathy, two of them in cats that had no threat reflex, mydriasis that did not respond to direct and consensual pupillary reflexes to light, as in the case of our study. The authors, however, attributed such alterations to retinal displacement. Such conclusion was not possible in our study since the fundoscopy examination was not carried out. One of these animals was in an abnormal (irresponsive) state of consciousness and another one was in a stupor, with opisthotonus, decerebrated posture and absent postural reactions in the four limbs. These neurological signs resemble those presented by the female cat in our study. In the four cases reported, the animals were treated with enalapril, amlodipine, and three of them with prednisone. As a result, an improvement of neurological signs was observed after normalization of systemic arterial pressure.

Stocco et al. (2016) and Agopian et al. (2016), in a study on the renal morphometry of domestic cats with macroscopically unchanged kidneys, concluded that the kidneys of this species do not exceed 4.0 cm and 4.5 cm in length. This shows that renomegaly in the patient of our study was really accentuated; with kidneys measuring 8.27 and 7.75 cm in length.

Nivy et al. (2015) describe bacterial infection of kidney cysts as a potential complicating factor of the disease. In this study, there was no evidence of bacterial infection, as the patient did not have fever and/or leucocytosis. However, it is important to perform a culture and antibiogram of the urine and cyst material (if possible) in cases of PKD. In this report, these tests were not performed, what characterizes a limitation of the study.

Abdominal radiography is not classified as a method for diagnosing PKD since bilateral renomegaly has other possible differential diagnoses. However, in the absence of other diagnostic methods, it can be used as a screening method, as we did in this report. The definitive diagnosis can only be concluded by autopsy and histopathological examination.

The histological findings in this study, such as renal tubular ectasia, and multiple renal cysts coated with cuboidal epithelium, corroborate those evidenced by Helps et al. (2007). This same study concluded that tissues fixed in wax and formaldehyde can be successfully used for real-time PCR test to detect the PKD gene. This may be useful in retrospective studies or when the patient has died and there is a need to confirm the diagnosis of PKD. The genetic test was not carried out in our study but it does not reduce the importance of this report due to its refinement of clinical and diagnostic data in cats with renomegaly. Bosje et al. (1998) also describe histopathological changes, similar to those found in our study, such as kidney cysts coated with a single layer of cuboid epithelial cells, filled with clear or haemorrhagic fluid, and kidney cysts causing compression of surrounding tissues.

CONCLUSION

In cats with abdominal distension associated with neurological signs, the list of differential diagnoses should be composed of renomegaly and hypertensive encephalopathy, which will optimize the tests to be ordered for exclusion or confirmation of polycystic kidney disease.

REFERENCES

AGOPIAN, R.G.; GUIMARÃES, R.A.; FERNANDES, M.V.M.; SILVA, M.M.S.; RIGHETTI, C.R.D.P.; BOMBONATO, P.P.; LIBERTI, E.A. Estudo morfométrico de rins em felinos domésticos (Felis catus). **Pesquisa Veterinária Brasileira**, v.36, n.4, p.329-338, 2016. <DOI: 10.1590/S0100-736X2016000400013>.

BAE, K.T., GRANTHAM, J.J. Imaging for the prognosis of autosomal dominant polycystic kidney disease. **Nature Reviews Nephrology**, v.6, p.96-106, 2010. <DOI: 10.1038/nrneph.2009.214>.

BARRS, V.R.; GUNEW, M.; FOSTER, S.F.; BEATTY, J.A.; MALIK, R. Prevalence of autosomal dominant polycystic kidney disease in Persian cats and related-breeds in Sydney and Brisbane. **Australia Veterinary Journal**, v.79, p.257-259, 2001. <DOI: 10.1111/j.1751-0813.2001.tb11977.x>.

BARTHEZ, P.Y.; RIVIER, P.; BEGON, D. Prevalence of polycystic kidney disease in Persian and Persian related cats in France. **Journal of Feline Medicine and Surgery**, v.5, p.345-347, 2003. <DOI: 10.1016/S1098-612X(03)00052-4>.

BILLER, D.S.; DIBARTOLA, S.P.; EATON, K.A.; PFLUEGER, S.; WELLMAN, M.L. RADIN, M. J. Inheritance of polycystic kidney disease in Persian cats. **Journal of Heredity**, v.87, n.1, p.01-05, 1996. <DOI: 10.1093/oxfordjournals.jhered.a022945>.

BONAZZI, M.; VOLTA, A.; GNUDI, G.; COZZI, M.C.; STRILLACCI, M.G.; POLLI, M.; LONGERI, M.; MANFREDI, S.; BERTONI, G. Comparison between ultrasound and genetic testing for the early diagnosis of polycystic kidney disease in Persian and Exotic Shorthair cats. **Journal of Feline Medicine and Surgery**, v.11, p.430-434, 2009. <DOI: 10.1016/j.jfms.2008.10.003>.

BOSJE, J.T.; VAN DEN INGH, T.S.G.S.M.; VAN DER LINDE-SIPMAN, J.S. Polycystic kidney and liver disease in cats. **Veterinary Quarterly**, v.20, n.4, p.136-139, 1998. <DOI: 10.1080/01652176.1998.9694858>.

CANNON, M.J.; MACKAY, A.D.; BARR, F.J.; BRADLEY, K.J.; GRUFFYDD-JONES, T.J. Prevalence of polycystic kidney disease in Persian cats in the United Kingdom. **Veterinary Record**, v.149, p.409-411, 2001. <DOI: 10.1136/vr.149.14.409>.

CORNEC-LE GALL, E.; ALAM, A.; PERRONE, R.D. Autosomal dominant polycystic kidney disease. **Lancet**, v.393, p.919-935, 2019. <DOI: 10.1016/S0140-6736(18)32782-X>.

DOMANJKO-PETRI, A., CERNEC, D.; COTMAN, M. Polycystic kidney disease: a review and occurrence in Slovenia with comparison between ultrasound and genetic testing. **Journal of Feline Medicine and Surgery**, v.10, p.115-119, 2008. <DOI: 10.1016/j.jfms.2007.07.004>.

EATON, K.A.; BILLER, D.S.; DIBARTOLA, S.P.; RADIN, M.J.; WELLMAN, M.L. Autosomal dominant polycystic kidney disease in Persian and Persian-cross cats. **Veterinary Pathology**, v.34, p.117-126, 1997. <DOI: 10.1177/030098589703400204>.

GUERRA, J.M.; FREITAS, M.F.; DANIEL, A.G.; PELLEGRINO, A.; CARDOSO, N. C.; DE CASTRO, I.; ONUCHIC, L.F.; COGLIATI, B. Age-based ultrasonographic criteria for diagnosis of autosomal dominant polycystic kidney disease in Persian cats. **Journal of Feline Medicine and Surgery**, p.01-09, 2018. <DOI: 10.1177/1098612X18764591>.

HELPS, C.; TASKER, S.; HARLEY, R. Correlation of the feline PKD1 genetic mutation with cases of PKD diagnosed by pathological examination. **Experimental and Molecular Pathology**, v.83 p.264-268, 2007. <DOI: 10.1016/j.yexmp.2007.04.002>.

LYONS, L.A.; BILLER, D.S.; ERDMAN, C.A.; LIPINSKI, M.J.; YOUNG, A.E.; ROE, B.A.; QIN, B.; GRAHN, R.A. Feline polycystic kidney disease mutation identified in PKD1. **Journal of the American Society of Nephrology**, v.15, p.2548-2555, 2004. <DOI: 10.1097/01.ASN.0000141776.38527.BB>.

MEUTEN, D. Avaliação e interpretação laboratorial do sistema urinário. In: THRALL, M.A.; WEISER, G.; ALLISON, R.W.; CAMPBELL, T.W. **Hematologia e**

Bioquímica Clínica Veterinária. 2.ed. São Paulo: Roca, 2015. Cap.23, p.689-806.

NIVY, R.; LYONS, L.A.; AROCH, I. SEGEV, G. Polycystic kidney disease in four British shorthair cats with successful treatment of bacterial cyst infection. **Journal of Small Animal Practice**, v.56, p.585-589, 2015. <DOI: 10.1111/jsap.12327>.

O`NEIL, J.; KENT, M.; GLASS, E.N.; PLATT, S.R. Clinicopathologic and MRI characteristics of presumptive hypertensive encephalopathy in two cats and two dogs. **Journal of the American Animal Hospital Association**, v.49, n.6, p.412-420, 2013. <DOI: 10.5326/JAAHA-MS-5942>.

ONDANI, A.C.; CARVALHO, M.B.; DE BRUM, A.M.; PEREIRA, M.L. Prevalência da doença renal policística em gatos domésticos da região de Jaboticabal. **Veterinária Notícias**, v.15, v.2, p.89-94, 2009.

POLZIN, D.J. Evidence-based step-wise approach to managing chronic kidney disease in dogs and cats. **Journal of Veterinary Emergency and Critical Care**, San Antonio, v.23, p.01-11, 2013. <DOI: 10.1111/vec.12034>.

SATO, R.; UCHIDA, N.; KAWANA, Y.; TOZUKA, M.; KOBAYASHI, S.; HANYU, N.; KONNO, Y.; IGUCHI, A.; YAMASAKI, Y.; KURAMOCHI, K.; YAMASAKI, M. Epidemiological evaluation of cats associated with feline polycystic kidney disease caused by the feline PKD1 genetic mutation in Japan. **The Journal of Veterinary Medical Science**, v.81, n.7, p.1006-1011, 2019. <DOI: 10.1292/jvms.18-0309>.

SCALON, M.C.; MAZZOTTI, G.A. Sistema Genitourinário – Rins policísticos felino. In: MAZZOTTI, G.A., DA ROZA, M.R. **Medicina Felina Essencial Guia Prático**. 1.ed. Curitiba: Equalis, 2016. p.229-231.

SEILER, G.S. Rins e Ureteres. In: THRALL, D.E. **Diagnóstico de Radiologia Veterinária**. 7.ed. Rio de Janeiro: Elsevier, 2019. Cap.41, p.823-846.

STOCCO, A.V.; SANTOS SOUSA, C.A.; GOMES, M.S.; SOUZA JÚNIOR, P.; ABIDU FIGUEIREDO, M. Is there a difference between the right and left kidney? A macroscopic approach in Brazilian Shorthair Cat. **Arquivo Brasileiro de Medicina Veterinária e Zootecnia**, v.68, n.5, p.1137-1144, 2016. <DOI: 10.1590/1678-4162-8339>.

VOLTA, A.; MANFREDI, S.; GNUDI, G.; GELATI, A.; BERTONI, G. Polycystic kidney disease in a Chartreux cat. **Journal of Feline Medicine and Surgery**, v.12, p.138-140, 2010. <DOI: 10.1016/j.jfms.2009.06.001>.

VUCICEVIC, M.; SLIJEPCEVIC, D.; DAVITKOV, D.; AVDALOVIC, V.; ALEKSIC-KOVACEVIC, S.; STEVANOVIC, J.; STANIMIROVIC, Z. First report of Polycystic kidney disease occurrence in Persian cats in Serbia. **Veterinária Italiana**, v.52, n.1, p.51-56, 2016. <DOI: 10.12834/VetIt.599.2885.2>.

YU, Y.; SHUMWAY, K.L.; MATHESON, J.S.; EDWARDS, M.E.; KLINE, T.L.; LYONS, L.A. Kidney and cystic volume imaging for disease presentation and progression in the cat autosomal dominant polycystic kidney disease large animal model. **BMC Nephrology**, v.20, n.259, p.2-11, 2019. <DOI: 10.1186/s12882-019-1448-1>.