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Reporte de caso: enfermedad renal de curso prolongado en felino "*felis catus*" geriátrico de 12 años

Case report: long-term renal disease in a 12-year-old geriatric feline "felis catus"

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Palabras claves: Gato
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Resumen

Introducción. La Enfermedad Renal Crónica (ERC) es una patología común dentro de la clínica diaria especialmente diagnosticada en pacientes adultos mayores. Existen casos donde el diagnóstico se establece de forma ocasional, ya que los pacientes no presentan sintomatología definida y esto se convierte en un hallazgo incidental. La ERC se desarrolla progresivamente afectando además del funcionamiento renal otros sistemas complejos dentro del organismo del paciente provocando crisis azotémicas entre otras complicaciones. Aquí se describe el caso de un felino macho de 12 años diagnosticado en 2016 con enfermedad renal IRIS CKD STAGE 2 el cual se mantuvo controlado dentro de los parámetros normales durante 7 años, hasta que presentó una crisis azotémica elevando la estadificación a IRIS CKD STAGE 4. Objetivo. Describir el manejo realizado en un paciente felino con enfermedad renal de curso prolongado, tratado en la clínica veterinaria Mora desde 08 de agosto de 2016 al 11 de mayo de 2023. Metodología. El presente trabajo de investigación descriptiva, del tipo estudio de caso. Retribuye a un caso clínico de un paciente felino geriátrico con una nefropatía de desarrollo prolongado. Conclusión. En el caso descrito, el diagnóstico temprano de la enfermedad es importante para el pronóstico y el tratamiento de esta. Área de la ciencia: Medicina Veterinaria

Keywords:
Cat
Kidney
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Urea
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CKD
IRIS

Abstract

Introduction. Chronic Kidney Disease (CKD) is a common pathology in daily clinical practice, especially diagnosed in older adult patients. There are cases where the diagnosis is established occasionally, since the patients do not present defined symptoms, and this becomes an incidental finding. CKD develops progressively, affecting, in addition to kidney function, other complex systems within the patient's body, causing azotemic crises among other complications. Here we describe the case of a 12-year-old male feline diagnosed in 2016 with IRIS CKD STAGE 2 kidney disease, which was controlled within normal parameters for 7 years, until he presented an azotemic crisis, raising the staging to IRIS CKD STAGE 4 .objective. Describes the management conducted in a feline patient with long-term kidney disease, treated at the Mora veterinary clinic from 08/08/16-05/11/23.Methodology.The present descriptive research work, of the case study type. It refers to a

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clinical case of a geriatric feline patient with a long-term nephropathy.Conclusion.In the case described, early diagnosis of the disease is important for its prognosis and treatment.

Introduction

Chronic kidney disease is defined as the structural and/or functional alteration of one or both kidneys for 3 months or more. The estimated prevalence in cats is 1% - 3%" (Mesa & Cortes, 2019, p. 645).

Chronic kidney disease (CKD) can be caused by diseases/disorders affecting any part of the nephron, including the glomerulus, tubule, vascular supply, and surrounding interstitium. Most definitions of CKD require the presence of the lesion for at least 3-4 months to allow time for compensatory hypertrophy to influence renal function. Early detection of CKD facilitates appropriate intervention that could preserve renal function or at least slow its progressive decline (International Renal Interest Society[[IRIS], 2019].

The structural changes associated with Chronic Kidney Disease are irreversible and the progression of the disease, although slow, can lead to terminal illness; a decrease in the number of functional units is expected during the course of this disease (Finch & Heine, 2018).

Kidney function adapts depending on how many non-functioning nephrons there are in the kidney. The more nephrons stop working, the healthy ones increase in size to compensate for the work of those that are atrophied, their glomerular filtration function increases and at the same time the glomerular capillary pressure increases significantly, causing compensatory glomerular hyperfiltration in the nephrons (Triana, 2022).

Unfortunately, the diagnosis of CKD in dogs and cats in clinical practice is identified quite late in the disease process, usually once the patient already presents clinical signs. This limits the potential benefit of treatment which, in many cases, could delay progression rather than result in recovery of renal function, and makes it difficult to identify the underlying etiology (Syme, 2019).

Occasionally, azotemia is found as an incidental finding on a routine blood test, likely indicating the presence of CKD. The presence and intensity of the hallmark clinical signs depend on the stage of the disease but also vary between individual patients; hallmark clinical signs of CKD include polyuria and polydipsia, decreased appetite, weight loss, low body condition score, and pale mucous membranes (Segev, 2022). "For patients who develop chronic kidney disease, the underlying etiology may sometimes never be

identified because the histopathologic response to injury in the kidney is the same, with tubular interstitial nephritis and fibrosis usually being identifiable" (Jepson & Syme, 2018, p. 1).

Diagnosis and etiology

It is understood that the loss of excretory function causes retention of nitrogenous substances such as BUN and creatinine, which are eliminated by glomerular filtration, which leads to changes in water, acid-base and electrolyte balance (Forrester & Lees, 1996, p. 954). Most patients with CKD are diagnosed after an increase in creatinine, however, azotemia does not appear until the filtration rate decreases by 75%. The predominant etiology for this type of pathology is mostly attributed to tubulointerstitial nephritis and, with a lower prevalence, pathologies such as glomerulopathies, lymphoma and amyloidosis (Mesa & Cortes, 2019, p. 645).

Creatinine is a product of the non-enzymatic breakdown of phosphocreatine in muscle and daily production is largely determined by the individual's muscle mass. It is not metabolized and is almost entirely excreted by the kidneys through glomerular filtration. Normal creatinine concentrations are 0.8 to 1.8 mg/dl in cats (DiBartola, 2007, p. 1718). Creatinine is the most widely used laboratory parameter to establish the various stages of Chronic Kidney Disease (Guamán, 2022, p. 63).

Creatinine concentration, either in serum or plasma, is the most commonly used indicator of renal function for the early diagnosis of CKD. The usefulness of creatinine for diagnosis is improved if it is considered together with the concentration of blood urea nitrogen (BUN) (Finch & Heine, 2018, p. 219).

An increase in BUN and Creatinine above normal implies that at least 75% of the nephrons are not functioning properly, the coefficient may increase as a result of the increased reabsorption of urea in the body (DiBartola, 2007, p. 1718). Urea is synthesized in the liver from ammonium, part of it is absorbed in the intestine and is excreted mainly by the kidney. In the kidney, it is freely filtered through the glomeruli and passively reabsorbed in the tubules (Guamán, 2022, p. 59).

Staging and prognosis

The IRIS proposed CKD Staging System is a tool that allows veterinarians to communicate about patients without relying on reference intervals established by any particular laboratory, and is widely used in clinical and research settings to promote standardization and facilitate the diagnosis of this disease (Finch & Heine, 2018, p. 223).

The long-term prognosis, with a median survival time of 105 days in dogs and only 66 days in cats. In dogs only 57% and 13% survived to 6 and 12 months, respectively, and in cats 81% and 8% survived to 6 and 12 months, respectively (Segev, 2022).

Survival time is determined by the quality of established medical care, commitment of the guardian, severity and duration of clinical signs in uremic crises, speed of progression and age of the patient. We have as average life time in cats with IRIS 2 of 1151 days; IRIS 3 of 679 days, IRIS 4 of 35 days (Mesa & Cortes, 2019, p. 663).

"Affected felines often have prolonged periods of stable renal function followed by acute episodes of azotemia; the natural course of CKD appears to be variable among feline patients" (Elliot et al., 2003).

According to Bulmer & Sisson (2007): "initial treatment for CKD is based on maintaining water balance, with water provided ad libitum to the patient under treatment" (p. 956).

Different factors are involved in the progression of CKD, an assessment of these factors is important to understand the rationale for the therapeutic interventions that are recommended for the treatment of this type of patients where the main goal is to reduce the progression of the disease (Jepson & Syme, 2018, p. 437).

It should be emphasized that the IRIS staging system is only applicable to dogs and cats with stable CKD. Staging is not appropriate in patients with abnormal renal function in whom the blood creatinine or SDMA concentration changes dramatically over a short period of time. The same would be true if the diagnosis of renal disease is based on proteinuria or loss of the ability to concentrate urine. These changes must be persistent and non-renal prerenal or perirenal causes have been ruled out (IRIS, 2023).

The blood creatinine and SDMA concentrations used to define IRIS CKD stages 1 through 4 were reached through discussion and consensus, based on the clinical experience of the Board members and data derived from longitudinal studies. As noted above, these and other elements of the staging may be modified in the future as more knowledge is gained. Many of the current creatinine reference ranges for healthy dogs and cats are broad enough to include patients with mild to moderate kidney disease (IRIS, 2023).

All treatments for chronic kidney disease (CKD) must be tailored to the individual patient. The following recommendations are useful starting points for most animals at each stage. Serial follow-up of these patients is ideal and treatment should be tailored according to treatment response (IRIS, 2023).

In general, in the early stages of CKD (stages 1 and 2), there are few clinical symptoms, extrarenal signs of the disease, and the therapeutic emphasis is on slowing progression.

Beginning in stage 3, extrarenal signs become more common and severe. The importance of administering treatments that address the clinical signs of CKD and improve the cat's quality of life assumes greater importance and outweighs the importance of treatments designed to slow progression in stage 4 (IRIS, 2023).

Treatment

Once the diagnosis of CKD has been established, the diagnostic and therapeutic objectives include: Identifying the factors that affect the cat's quality of life, selecting the treatments (pharmacological or nutritional) that should improve its quality of life, identifying the factors that increase the risk of progression of kidney disease, selecting the treatments (pharmacological or nutritional) that could reduce the risk of progression of kidney disease, monitoring the response to treatment and ensuring that it is appropriate for each particular case (Mimg-Ge,&Mimg-Ge, 2023, pp. 3-4).

The treatment is palliative and varies according to the patient's stage. Its purpose is to reduce the workload of the kidneys, relieve clinical symptoms and consequences due to uremic intoxication, reduce acid-base disturbances, so that the progression of the disease is slower (Rodriguez, 2016).

It is rare to achieve specific treatment of the underlying cause of CKD, since the cause that causes it is usually unknown, therefore, thetreatment isIt usually focuses on reducing the progression and treating the consequences of the disease (Taylor, 2007, p. 563).

Regarding the treatment for stage 2 CKD, the following are listed as common: Discontinue all potentially nephrotoxic medications, identify and treat any prerenal or postrenal abnormalities, measure blood pressure and urine protein creatinine ratio (UP/C), consider feeding a clinical renal diet. Manage dehydration according to the condition they present and provide water ad libitum (IRIS, 2023).

Treatment of dehydration usually with Ringer Lactate using a maintenance solution or if necessary, acute resuscitation therapy in the event of an azotemic crisis with fluid therapy 15-20 ml/kg every 20 minutes until the crisis normalizes (Mesa & Cortes, 2019, p. 638).

A patient with previously stable CKD who suffers a uremic crisis may arrive at the consultation in an acute manner with signs of “acute on chronic” disease or also known as decompensated renal disease, caused mainly by loss of fluid volume either exogenously or by lack of intake, concomitant urinary tract diseases, dental diseases, hypertension (Taylor, 2007, p. 562).

Compared to maintenance diets, diets for this disease should be lower in protein, phosphorus and sodium, and increase the buffering capacity of the diet, soluble fiber, B vitamins and antioxidants, potassium and fatty acids (Restrepo, 2021).

Factors to consider in prescription diets are recommended to have a decrease in protein, but at the same time this is highly digestible, decreased amounts of phosphate and sodium; increases in B vitamins and caloric density, greater intake of potassium, antioxidants and polyunsaturated fatty acids (Taylor, 2007, p. 564).

Methodology

This descriptive research work, of the case study type, pays attention to a clinical case of a geriatric feline patient with a long-term nephropathy.

The following materials were used in the case report:

- Patient's medical history
- Laboratory tests
- Abdominal Ultrasound
- Literature

Clinical case description

A 12-year-old, neutered, mixed-breed male feline, weighing 4.25 kilograms (kg), came to the clinic on August 8, 2016 due to decreased appetite, weight loss (approximately 1 kilogram kg), and hair loss. The owners report that the patient has previously had episodes of bloody diarrhea and on this occasion with sporadic vomiting. The general physical examination shows slightly congested gums at the base of the teeth, slight eye discharge, no nasal discharge, slightly dirty ears, abdominal palpation without alterations, synchronous femoral pulse. Positive swallowing reflex (DR); Negative cough reflex (DR); Normal cardiac and pulmonary auscultation; Heart rate (HR) 186 beats per minute (bpm); Respiratory rate (RR) 34 breaths per minute (rpm) Capillary refill time (Tllc) 3 seconds; Temperature (T) 39.1 °C; Skin elasticity or turgor of 2.5 seconds; percentage of dehydration of 6%. Based on the signs, differential diagnoses were established; among which were kidney disease, gastritis and parasitosis, which were later ruled out or confirmed with laboratory tests.

Additional tests were performed such as Blood Chemistry (ABAXIS VETSCAN VS2), 2014, model No. 15100, Union City, California, United States of America and the result was ALP 7 BUN 27 CRE 2.8. There was a moderate increase in creatinine showing moderate renal azotemia, which according to the IRIS staging corresponds to Chronic Kidney Disease STAGE 2. A complete blood count was performed where lymphocytosis, leukocytosis, neutropenia and eosinophilia were observed, and hypochromic microcytic anemia and platelet aggregates were evident. The patient was hospitalized and supportive fluid therapy was implemented (08/08/2016). 24 hours after establishing the therapy, a new sample was taken for specific creatinine control, obtaining a result of 2.8.

On 08/14/2016, a control study was carried out to monitor the initial values, which yielded CRE 2.87, thus confirming the diagnosis previously established in the previous days. From this moment on, the use of Royal Canin Renal for cats as exclusive food was implemented as a home treatment.

During the following months, routine checks were carried out where the BUN and CRE values remained within the same stage until 2023, when she presented with an azotemic crisis again.

On 02/28/2023 the patient comes to the consultation due to weakness and loss of appetite. A general physical examination shows pale pink mucous membranes, capillary refill time greater than 2.5 seconds, increased swallowing reflex, abdominal palpation with pain in the epigastrium and mesogastrium, rectal temperature of 38 °, with body condition 2/5, dehydration percentage of 8%, skin turgor greater than 3 seconds. It is decided to hospitalize the patient for management of hypovolemic shock and follow-up tests are performed for his renal function where we obtain as blood chemistry results BUN 274 CRE 20.8 AMY 1769 K 7.1 TP 9.7 complete blood count, leukocytosis, lymphopenia, and neutrophilia. We establish a diagnosis of renal azotemia as part of a decompensated STAGE 4 (IRIS) chronic kidney disease.

The patient was hospitalized for 4 days in the intensive care unit with resuscitation fluid treatment at a high flow of crystalloids at a rate of 10-15 ml per kg of live weight, and the tests were repeated daily, obtaining an evident decrease in CRE and BUN, recategorizing the patient as IRIS STAGE 2, returning to the phase prior to decompensation.

On 06/23/2023, the patient comes to the consultation because he has lost 12.5% of his live weight, so control tests are performed starting with an ultrasound where a right kidney with nephroliths in the renal parenchyma is evident, a 3.9 mm x 4.1 mm stone in the right ureter. Right kidney hydronephrosis. Hypotrophied left kidney.

In the urinalysis performed, the cytochemical results were elevated nitrite, leukosuria, hematuria, amorphous oxalate crystals and struvite crystals. SDMA was processed, obtaining a result greater than 100ug/dl BUN 40, CRE 2.5 complete blood count: leukocytosis and neutrophilia. MAP 162/100

On 07/28/2023, he comes again for an azotemic crisis, complementary tests were carried out, resulting in: BUN 80, CRE 4.8, K 5.9; Complete blood count: leukocytosis, lymphopenia, and neutrophilia. The patient is hospitalized for treatment of azotemia and treatment with resuscitation fluids is started. The next day, control tests were performed: BUN 106, CRE 7.2, K 6.6; Complete blood count: neutrophilia and anemia. Laboratory tests are performed again 24 hours later, Blood Chemistry: ALB 1.6, T BIL 1.1, BUN 121, CRE 8.8, Na 140, TP 4.4. Complete blood count: regenerative hypochromic

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normocytic anemia, where it is evident that the patient no longer responds to therapy, MAP 160/97 is monitored. The patient is admitted to multi-organ - multi-system failure and the owners decide to euthanize.

Table 1

Initial Blood Chemistry

Analytes	Reference Value	8/8/2016
ALB	2.2-4-4	3.6
ALP	10-90.	7
ALT	20-100	40
AMY	300-1100	994
TBIL	0.1-0.6	0.3
BUN	10-30.	27
AC	8.0-11.8	10.3
FOS	3.4-8.5	5.3
CRE	0.3-2.1	2.8
GLU	70-150	90
NA+	142-164	145

Table 1

Initial Blood Chemistry(continuation)

Analytes	Reference Value	8/8/2016
K+	3.7-5.8	4.0
TP	5.4-8.2	6.8
BGLOB	1.5-5.7	3.2

ALB: albumin, ALP: alkaline phosphatase, ALT: alanine aminotransferase, AMY: amylase, TBIL: bilirubin, BUN: blood urea nitrogen, CA: calcium, FOS: phosphorus, CRE: creatinine, GLU: glucose, NA: sodium, K+: potassium, PT: prothrombin time, GLOB: globulin

Table 2

Specific analysis of Creatinine 2016

Analyte	Reference value	9/8/2016	10/14/2016
CRE	0.3-1.4	2.8	2.87

Note:CRE: Creatinineto

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Table 3

Hemogram performed in 2016

Analytes	Reference values	8/8/2016
WBC	5.4-15.3	18.2
LYM	0.4-5.8	14.9
MON	0.1-1.4	0.1
NEW	2.8-12.8	1.3
EOS	0.0-0.4	1.7
BAS	0.0-0.2	0.1
RBC	5.20-8.06	7.48
HGB	12.4-19.1	11.5
HCT	29.8-57.5	34.7
MCV	62.7-72.0	46.4
MCHC	34.0-36.6	33.1
PLT	160-525	475

WBC: leukocytes, LYM: lymphocytes, MON: monocytes, NEU: neutrophils, EOS: eosinophils, BAS: basophils, RBC: erythrocytes, HGB: hemoglobin, HTC: hematocrit, MCV: mean corpuscular volume, MCHC: mean corpuscular hemoglobin concentration, PLT: platelets

Table 4

Blood chemistry performed in 2023

Analyte	**Reference Value**	**11/3/2023**	**4/21/2023**	**6/23/2023**	**28/7/2023**	**29/7/2023**	**30/7/2023**
ALB	**2.2-4-4**	**3.4**	**3.8**	**3.5**	**2.9**	**2.5**	**1.6**
ALP	**10-90.**	**30**	**11**	**21**	**15**	**10**	**15**
ALT	**20-100**	**42**	**43**	**27**	**32**	**33**	**35**
AMY	**300-1100**	**1166**	**1043**	**1292**	**1022**	**641**	**551**
TBIL	**0.1-0.6**	**0.2**	**0.1**	**0.2**	**0.3**	**0.4**	**1.1**
BUN	**10-30.**	**29**	**28**	**40**	**80**	**106**	**121**
AC	**8.0-11.8**	**10.4**	**11.7**	**12.6**	**10.7**	**9.1**	**9.5**
FOS	**3.4-8.5**	**5.2**	**4.2**	**6.1**	**4.3**	**3.9**	**7.0**
CRE	**0.3-2.1**	**2.4**	**2.4**	**2.5**	**4.8**	**7.2**	**8.8**
GLU	**70-150**	**87**	**93**	**197**	**135**	**137**	**102**
NA+	**142-164**	**149**	**149**	**154**	**151**	**146**	**140**
K+	**3.7-5.8**	**3.6**	**5.3**	**5.5**	**5.9**	**6.6**	**5.7**
TP	**5.4-8.2**	**7.3**	**7.5**	**7.4**	**7.2**	**5.5**	**4.4**
GLOB	**1.5-5.7**	**4.0**	**3.7**	**3.4**	**4.3**	**3.1**	**2.4**

ALB: albumin, ALP: alkaline phosphatase, ALT: alanine aminotransferase, AMY: amylase, TBIL: bilirubin, BUN: blood urea nitrogen, CA: calcium, FOS: phosphorus, CRE: creatinine, GLU: glucose, NA:sodium, K+: potassium, PT: prothrombin time, GLOBE: globulin.

Table 5

Hematic biometry (blood count) performed andn2023

Analytes	Reference value	28/2/2023	6/23/2023	28/7/2023	29/7/2023	30/7/2023
LEU	3.50-20.70	19.54	30.57	25.20	17.29	11.63
LIN	0.83-9.10	0.84	1.08	0.82	0.93	1.62
MON	0.09-1.21	1.01	1.97	1.20	0.28	0.61
NEW	1.63-13.37	17.62	27.45	23.07	15.96	9.29
EOS	0.02-0.49	0.05	0.06	0.10	0.11	0.11
BAS	0.00-0.20	0.02	0.01	0.01	0.00	0.00
ERI	7.70-12.80	11.60	9.74	8.24	6.86	5.64
Hb	10.0-17.0	17.5	13.5	11.0	9.1	7.3
HCT	33.70-55.40	48.51	42.48	35.88	29.96	24.50
VCM	35-52	42	44	44	44	43
CHCM	27.035.0	36	31.7	30.7	30.3	29.7
PLT	125-618	319	478	300	176	150

WBC: leukocytes, LIN: lymphocytes, MON: monocytes, NEU: neutrophils, EOS: eosinophils, BAS: basophils, RBC: erythrocytes, Hb: hemoglobin, HTC: hematocrit, MCV: mean corpuscular volume, MCHC: mean corpuscular hemoglobin concentration, PLT: platelets

Table 6

Taking blood pressure

Date	PAM
6/23/2023	162/100-(123)
28/7/2023	149/81-(102)

Note:MAP: average pressure in the arteries during a cardiac cycle

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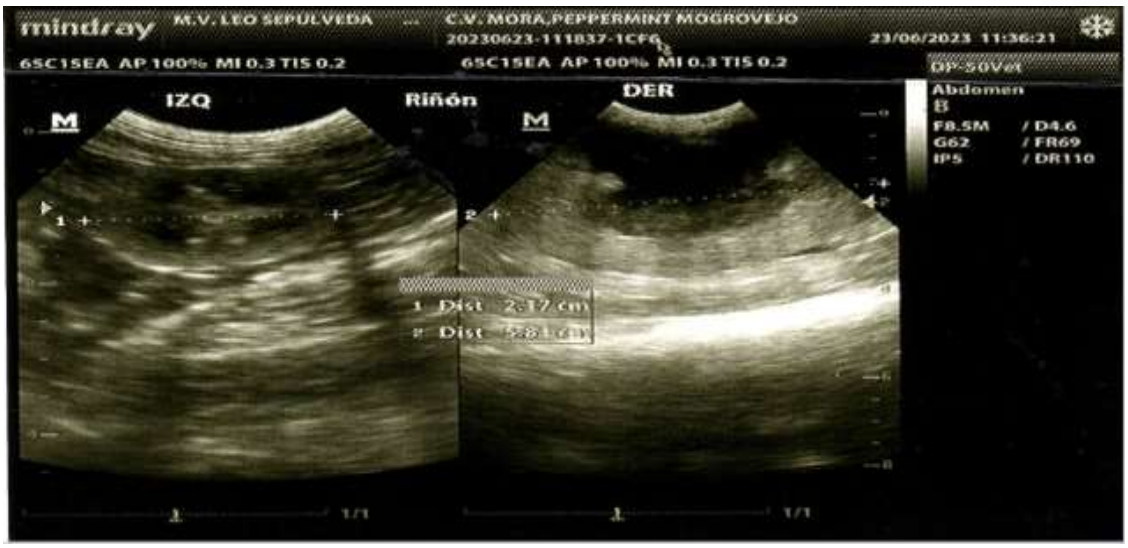
Figure 1

Image of left and right kidney



Note: Left kidney: small organ compared to the right kidney. poor definition of diverticula, with predominance of decreased echogenicity. Right kidney: asymmetry and moderate dilatation of the renal pelvis, dilated proximal ureter, with the presence of an echogenic structure of approximately 4 mm that produces a clean acoustic shadow.

Figure 2

Monitoring patient blood pressure using petMAP graphic

Results

Chronic kidney disease has a high prevalence in human medicine and in domestic felines, where approximately 10% of cats older than 10 years suffer from this pathology as reported by (Finch et al., 2016). Having an important difference in our case since the diagnosis of our patient was when he was 5 years old and remained until he was 12.

The favorable response to the azotemic crises that the patient presented during the prolonged development of his disease corresponds to the timely implementation of intravenous fluid treatment.

As recommended by Mesa & Cortes (2019), establishing treatment with acute resuscitation fluid therapy increased the probability of improvement in the patient, and this, by having a timely response, was crucial in returning to what was considered homeostasis in him.

Although this disease has a poor functional prognosis and short survival time according to what King et al. (2007) previously described, the average life expectancy did not exceed 319 days; in the case of our patient, it reached 7 years on average after diagnosis.

Conclusions

- In the described case, early diagnosis of the disease is important for its prognosis and treatment.
- Monitoring degenerative diseases is a factor to consider for the constant evaluation and recategorization of these diseases, thus avoiding the development of a crisis. Particularly in felines, changes in their daily behavior must be taken into account, since minimal alterations are a fundamental component for the diagnosis of a complication.
- The correct implementation and application of international protocols (IRIS) for the treatment of Chronic Kidney Disease, together with the continuous updating of clinicians, are in fact the fundamental pillar for success in the intensive care unit.

Conflict of interest

The authors certify that there are no conflicts of interest in this work.

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