A renal failure related to the feline world

Un fracaso renal relacionado con el mundo felino

Dear Editor,

Acute kidney injury (AKI) is one of the most common reasons for consultation.[1] Although the most common aetiology is prerenal, there are geographical, cultural and economic factors that can vary the most probable cause and the form of clinical presentation (the spectrum of which can also be very broad).[2] The hospital incidence is variable, reaching close to 20%.[3] AKI leads to an increased risk of death and a variable percentage of these patients do not recover their baseline renal function.[3]

We present the case of an 80-year-old woman, independent in activities of daily living (IADL) and living in a rural area. She was diagnosed with high blood pressure, anticoagulated paroxysmal atrial fibrillation, chronic kidney disease stage 3a A1 of unknown ethiology without nephrology follow-up (baseline creatinine of 1.08 mg/dl, urea 68 mg/dl and glomerular filtration rate [GFR] 49 ml/min/1.76 m^2) and aortic stenosis operated on with a bioprosthetic valve three years earlier. Her long-term treatment included bisoprolol, lorazepam, furosemide, olmesartan/hydrochlorothiazide, statin and Adiro (acetylsalicylic acid).

She went to the Emergency Room with a four-month history of a constitutional syndrome with asthenia, weight loss of 10 kg and hyporexia. Her vital signs were normal. Additional tests revealed microcytic anaemia with haemoglobin of 8.7 and mild lymphopenia. Deteriorated renal function was observed, with a creatinine level of 2.37 mg/dl, urea 85 mg/dl and with active sediment and negative urine culture. The patient was admitted, requesting: computed tomography (CT) of chest/abdomen; endoscopic studies of gastrointestinal system; and complete blood count with proteins.

Gastroscopy, colonoscopy and CT did not yield significant findings, reasonably ruling out cancer as the cause of her constitutional syndrome. In the analysis of proteins, a biclonal IgG-kappa lambda peak was found with negative immunofixation in urine, so a bone marrow biopsy was performed, with no abnormal findings. In parallel, the patient's renal function was progressively deteriorating, with a peak creatinine level of 8.64. mg/dl (previous 5.99 - >6.2 - >7.46 mg/dl) and with persistence of non-nephrotic proteinuria (protein/creatinine ratio 2,032 mg/g) and microhaematuria, with a progressive tendency to hypertension and oliguria leading to heart failure. This was in the context of complete nephritic syndrome with the need for urgent haemodialysis and, at that point, further immunological studies were requested.

During this whole process, an autoimmunity study was requested with antinuclear antibodies (ANA), antineutrophil cytoplasmic antibodies (ANCA) and anti-glomerular basement membrane antibodies (anti-GBM). They were negative, showing a decrease in C3 0.73 g/l (0.90–1.80) with an increase in rheumatoid factor 67.5 U/l (0–14). In light of the above and with no improvement in kidney function in the context of established nephritic syndrome without a clear cause, a kidney biopsy was performed (Fig. 1). The biopsy report referred to a mesangiocapillary glomerulonephritis (GN) with extracapillary proliferation (cellular crescents), with very positive IgM immunofluorescence with negative C3.

Given these findings, an infectious process with an atypical presentation was suspected as the cause of the condition, and taking into account the rural area of residence of the patient, the diagnostic series was expanded with zoonosis serologies (*Coxiella*, *Bartonella henselae* (BH), *Leptospira* and *Borrelia*). In the end, positive serology for BH was obtained (IgM and IgG titre 1/256). A blood polymerase chain reaction (PCR) was requested for *Bartonella* twice, which was negative in both cases. The possibility of endocarditis was raised without a conclusive diagnosis after transoesophageal echocardiogram and positron emission tomography (PET), but it was finally decided to treat the condition as such, with a regimen of doxycycline 100 mg and rifampicin 300 mg every 12 h for at least two weeks.

After starting antibiotic therapy, the patient's clinical condition and blood test results progressed favourably, with improvement in kidney function, and the haemodialysis sessions could be discontinued. Additionally, her levels of rheumatoid factor and C3 returned to normal (Table 1). Given the persistence of positive IgM for BH, doxycycline was continued for six weeks. Three months later, the IgM was negative and the IgG was positive, with a titre 1/512 for BH with creatinine of 2.52 mg/dl and urea 111 mg/dl.

This article has described a nephritic syndrome caused by a mesangiocapillary GN with cellular crescents secondary to an active BH infection, with no similar cases found in the literature.

The clinical spectrum of BH infection is broad, ranging from a latent and non-specific condition affecting the general state to cat-scratch disease and endocarditis with negative blood cultures.[4,5] It can even trigger immunological phenomena, such as mesangiocapillary glomerulonephritis, which over several months can lead to extracapillary proliferation. Immunological manifestations are usually related to the manifestation of the infection.[6–9]

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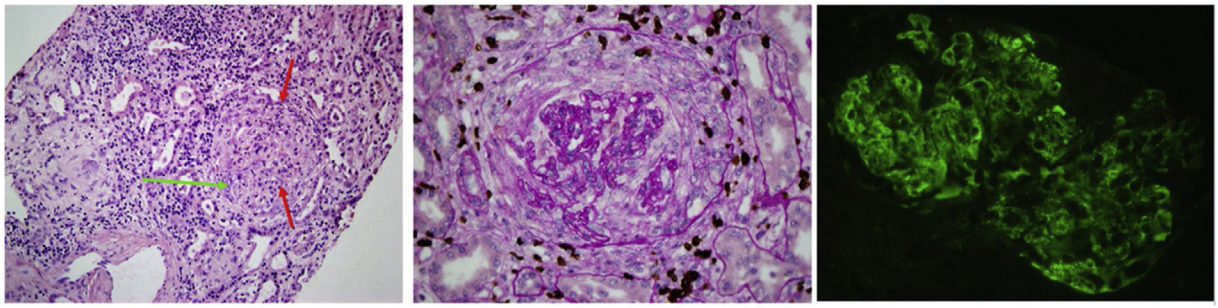

Fig. 1 – Haematoxylin-eosin and Periodic Acid-Schiff (PAS) positive sample from a renal cast seen under an optical microscope. In the image on the left, two renal glomeruli can be seen, showing a large extracellular growth containing numerous nuclei (cellular crescent) (red arrows), reducing the renal tuft (green arrow). The central image shows a glomerulus with a cellular crescent. The image on the left shows positive IgM immunofluorescence.

Table 1 – Progression of immunological parameters and renal function.

	17 Aug	19 Aug	18 Sep	10 Nov	25 Nov
C3 g/l	0.91	0.73	0.45	0.93	0.83
C4 g/l	0.12	0.11	0.13	0.2	0.2
Rheumatoid factor kU/l	67.5	91	55.7	11.5	9.9
Diuresis ml	2,000	2,300	800	1,500	1,500
Creatinine mg/dl	3.8	4.83	5.27	3.8	2.52

The exceptional aspect of our case is that mesangiocapillary glomerulonephritis was the only certain clinical manifestation of BH infection. With this case we wanted to stress the importance of the medical history, including patients' personal information, such as place of residence, as all of this needs to be taken into account for the differential diagnosis and approach to acute kidney injury.

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Conflicts of interest

The authors declare that they have no conflicts of interest.

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Adriana M. Cavada-Bustamante[a,c,*], Clara Sanz García[a], Natalia Menéndez García[a], María Gago Fraile[a], Blanca Vivanco Allende[b], Joaquín Bande Fernández[a]

[a] *Nefrología, Hospital Universitario Central de Asturias (HUCA), Oviedo, Spain*
[b] *Anatomía Patología, HUCA, Oviedo, Spain*
[c] *Nefrología, Hospital Universitario de Navarra, Pamplona*

* Corresponding author.
E-mail address: adriana.cavada.bustamante@navarra.es (A.M. Cavada-Bustamante).

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"Latent tuberculosis vs active tuberculosis in dialysis patients: lessons from an epidemiological study in orense"

"Tuberculosis latente vs tuberculosis activa en pacientes en diálisis: enseñanzas de un estudio epidemiológico en orense"

Dear Editor,

Galicia has one of the highest incidences of tuberculosis (TB) of any of the autonomous communities of Spain, which is even higher in patients on renal replacement therapy (RRT).[1,2]

In 2019, a plan was designed for the prevention and control of TB in Spain,[3] which stresses the importance of reinforcing the identification of latent tuberculosis infection (LTI) in certain groups of patients, including patients on RRT and, fundamentally, those on the kidney transplant waiting list.

We carried out a study of the prevalence of LTI in patients on RRT, both on haemodialysis (HD) and peritoneal dialysis (PD), by performing the tuberculin test (TT) and the Interferon Gamma Release Assay (IGRA) tuberculosis test, if the TT negative. If the TT or IGRA were positive, patients were evaluated by the tuberculosis unit (TBU) which, after ruling out active TB, with the tests that they considered appropriait was de decided whether or not to start chemoprophylaxis.

We evaluated 209 patients, seven of which were excluded due to having previously suffered from active TB. Of the remaining 202, 70.29% were on HD.

In patients on PD, 18.3% had TT^{+} compared to 12.7% on HD.

Patients who were TT^{-} were given an IGRA test, which was positive in 18.8% of patients on HD and 11.8% in patients on PD.

Patients with LTI were classified into two groups: those TT^{+}; and those TT^{-} and $IGRA^{+}$. The second group were older patients, with a longer time on dialysis and with a higher Charlson index (CI).

A total of 53 patients had LTI. Eleven cases (20.75%) did not receive treatment, either due to excessive associated comorbidity or patient refusal. Thirty-seven cases (69.81%) completed the prophylaxis treatment according to the TBU protocol, without notable side effects, and at the end, treatment had to be abandoned in five cases (9.43%) because of side effects.

The demographic characteristics and results are shown in Table 1.

During follow-up, three patients developed TB, all extrapulmonary; six months after completing prophylaxis, one of these patients on the PD programme developed peritonitis with a negative culture and was diagnosed with miliary TB. Another patient on HD who was TT^{-} and $IGRA^{+}$, interpreted as LTI, developed a pericardial effusion with haemodynamic compromise a few weeks after starting prophylaxis. Although there was no microbiological diagnosis, after antituberculosis treatment, which he completed successfully, produced a significant clinical and radiographic improvement.

A patient on HD who stopped chemoprophylaxis after developing a severe rash had to be admitted to hospital with pericardial effusion and constitutional syndrome. The clinical, laboratory and radiological data were consistent with TB, although it could not be confirmed histologically. In accordance with the TBU, he was started on antituberculosis treatment, with clear clinical improvement.

In patients on RRT, the combination of TT and IGRA is required to diagnose LTI, particularly in older adult patients who have been on dialysis for longer and have greater comorbidity, because in this group TT performs poorly.[4–6]

Prior to starting prophylaxis, active disease must be ruled out, particularly in patients on the kidney transplant waiting list.[7] It is important to remember that in patients on dialysis, TB is generally extrapulmonary,[8] therefore a simple chest X-ray or sputum cultures may not be sufficient to make the diagnosis. A high degree of clinical suspicion is essential to come up with a diagnosticsince histological diagnosis is not always possible, as was the case in our patients. The post-treatment clinical and radiological improvement they experienced supported the diagnosis of active TB.

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