Letter to the Editor

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. Although the most common aetiology is pre-renal, 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). The hospital incidence is variable, reaching close to 20%. AKI leads to an increased risk of death and a variable percentage of these patients do not recover their baseline renal function.

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 aetiology without nephology follow-up (baseline creatinine of 1.08 mg/dl, urea 68 mg/dl and glomerular filtration rate [GFR] 49 ml/min/1.73 m²) 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 acute 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 bicalon 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 conclusive diagnosis after transesophageal 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 con-

DOI of original article:

ARTICLE IN PRESS

Nefrología. 2024;xxx(xx):xxx-xxx

Revista de la Sociedad Española de Nefrología

www.revistanefrologia.com
It over an injury.

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.\(^5\)\(^6\) 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.\(^6\)\(^9\)

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.

**Funding**

No funding was received for this study.

**Conflicts of interest**

The authors declare that they have no conflicts of interest.

**Table 1 – Progression of immunological parameters and renal function.**

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</table>

**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.

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https://doi.org/10.1016/j.nefroe.2024.03.020