patients admitted due to COVID-19. Nefrologia. 2021;41:34–40, http://dx.doi.org/10.1016/j.nefro.2020.08.005.
2. Rebaza-Mateo A, Yarasca-Moreno F, Tataje-Lavanda L. Apreciación del fracaso renal agudo en pacientes con por COVID-19. Nefrología. 2021, http://dx.doi.org/10.1016/j.nefro.2021.06.002.
3. Ministerio de Sanidad de España. Estudio ENE-COVID19: primera ronda. Estudio nacional de sero-epidemiología de la infección por SARS-CoV-2 en España [Acceses on May 2020]. Available in: https://www.mscbs.gob.es/ciudadanos/ene-covid/home.htm.
4. Portolés J, Marques M, López Sánchez PM, de Valdenebro M, Múñez E, Serrano ML, et al. Chronic kidney disease and acute kidney injury in the coronavirus disease 2019 Spanish Outbreak. Nephrol Dial Transplant. 2020;35:1353–61, http://dx.doi.org/10.1093/ndt/gfaa189.
5. Meijers B, Hilbrands LB. The clinical characteristics of coronavirus-associated nephropathy. Nephrol Dial Transplant. 2020;35:1279–81, http://dx.doi.org/10.1093/ndt/gfaa197.

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Appreciation of acute kidney failure in patients with COVID-19 infection

Apreciación del fracaso renal agudo en pacientes con COVID-19

Dear Editor,

We read with great interest the article published by Tarragón-Blanca et al, whose aim was to describe the different presentations of acute kidney injury (AKI) requiring intervention by the nephrologist, its clinical course and possible strategies for early detection and nephroprotection. The authors concluded that hypovolaemia and dehydration are the most common causes of AKI in patients with COVID-19, as well as a poorer respiratory, analytical and renal prognosis. They also recommended monitoring of renal markers, in addition to personalised management of blood volume, as these may be decisive in preventing AKI[1].

In the study, an updated clinical correlation was rightly made in the description of patients with COVID-19 infection in relation to the WHO classification and CURB-65, and the staging of acute kidney injury according to the KDIGO guidelines.

The therapeutic strategy for AKI continues to be both conventional renal replacement therapy and continuous veno-venous haemodiafiltration, positive reinforcement on our part for differentiating the clinical course of the patient associated with COVID-19 infection. They even compare the discharge of patients with AKI on admission and in-hospital AKI[2].

One of the weaknesses of the study is the small sample size, as type II error can occur, leading to false negatives being obtained, which could exclude variables that need to be taken into account. Elsewhere, in the description of the renal failure, a previous nephropathy could be considered associated with its aetiology, and the clinical course could be compared in in-hospital AKI associated with COVID-19 infection[3].

In addition, attention should be given to the differences between the increase in laboratory standards such as creatinine, urea, haematuria and proteinuria from previous episodes of nephropathy related to its aetiology, and in-hospital renal failure[3,4].

Hospital outcomes could include the duration of acute kidney injury and its relationship with the patient's hospital stay, whether they had AKI on admission or in-hospital[5].

We congratulate the authors for their published work, as it provides valuable information on the association of acute kidney injury with COVID-19, while also taking the follow-up by nephrologists into account. In addition, it broadens the panorama beyond mere parenchymal involvement by SARS-CoV-2, while being the first study to analyse a Spanish cohort.

Conflicts of interest

The authors have no conflicts of interest to declare with regard to the writing of this letter.

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REFERENCES

1. Tarragón B, Valdenebro M, Serrano ML, Maroto A, Llópez-Carratalá MR, Ramos A, et al. Fracaso renal agudo en pacientes hospitalizados por COVID-19. Nefrología. [Online]. 2021;41:34–40, http://dx.doi.org/10.1016/j.nefro.2020.08.005.
2. Claure-Del Granado R, Casas-Aparicio G, Rosa-Diez G, Rizo-Topete L, Ponce D. Renal Replacement Therapy for Acute Kidney Injury in COVID-19 Patients in Latin America. Kidney Blood Press Ress. [Online]. 2020;45:775–83, http://dx.doi.org/10.1159/000511914.
3. Menon T, Sharma R, Kataria S, Sardar S, Adhikari R, Tousif S, et al. The Association of Acute Kidney Injury With Disease Severity and Mortality in COVID-19: A Systematic Review and Meta-Analysis. Cureus. [Online]. 2021;13:e13894, http://dx.doi.org/10.7759/cureus.13894.
4. Mohamed MMB, Velez JCQ. Proteinuria in COVID-19. Clin Kidney J. [Online]. 2021;14 Supplement_1:i40–7, http://dx.doi.org/10.1093/ckj/sfab036.
5. Cheng Y, Zhang N, Luo R, Zhang M, Wang Z, Dong L, et al. Risk Factors and Outcomes of Acute Kidney Injury in Critically Ill Patients with Coronavirus Disease 2019. Kidney Dis. [Online]. 2020;14 Supplement_1:1–9, http://dx.doi.org/10.1159/000512270.

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A case of acute interstitial nephritis following the Pfizer–BioNTech COVID-19 vaccine

Un caso de nefritis tubulointersticial aguda después de la vacunación con Pfizer-BioNTech COVID-19

Dear Editor,

We still do not fully understand the mechanisms behind renal involvement in SARS-CoV-2 infection, whether direct injury from the virus or resulting from depletion and/or release of cytokines as a severe complication of the respiratory condition.[1,2] After vaccination was rolled out, the world population began to see a ray of hope. However, this has been obscured by reports of adverse events creating a potential barrier to large-scale vaccination efforts. We present the case of a patient with acute tubulointerstitial nephritis (ATIN) on top of diabetic nephropathy, in which no related agent was identified, except for the Pfizer-BioNTech Comirnaty© COVID-19 mRNA vaccine (BNT162b2).

This was a 78-year-old male with hypertension, hyperuricaemia, dyslipidaemia, diabetes mellitus with good metabolic control and chronic kidney disease (CKD) stage 3a-b/A3. In November 2018, he had a serum creatinine (Cr) level of 1.7 mg/dl, estimated glomerular filtration rate by CKD-EPI of 39 ml/min/1.73 m^2 and albumin/creatinine ratio (ACR) of 1400 mg/g, with no repeat or subsequent nephrology follow-up. He had no history of COVID-19 infection. He was on long-term treatment with statins, ACE inhibitors, allopurinol, vildagliptin and metformin. He went to Emergency Room after a lab tests that revealed Cr of 5.38 mg/dl, urea of 156 mg/dl, anaemia with eosinophilia, and ACR of 3397 mg/g, with no nephrotic syndrome. He had urine sediment with microhaematuria and leukocyturia without eosinophils in the urine (Table 1). The patient reported mild hyporexia and asthenia since receiving the first dose of the COVID-19-BNT162b2 vaccine three weeks prior, but no fever or skin rash; 48 h before admission, he received the second dose of the vaccine but reported no additional symptoms. He showed mild signs of dehydration on physical examination. No abnormalities on chest X-ray and PCR for SARS-CoV-2 was negative. The ultrasound showed small kidneys (left 9.4 cm and right 10 cm) with slightly hyperechoic parenchyma; no signs of dilation of the collecting duct system. Renal biopsy (Fig. 1) showed a total of 40 glomeruli, 21 were globally sclerosed (52%), the rest presented lesions of nodular glomerulosclerosis. The interstitium showed foci of severe mononuclear inflammatory infiltrate with abundant eosinophils, with tubular damage, fibrosis and tubular atrophy. The arterioles showed no lesions and there were no immune deposits in the immunofluorescence. The findings were consistent with the pathology diagnosis of immunoallergic ATIN[3,4] that combined with the clinical characteristics and the recent administration of the COVID-19 vaccine, led us to define this case as ATIN potentially induced by the BNT162b2 vaccine. Treatment was started with three boluses of methylprednisolone 125 mg/day followed by

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