Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

sible to explain diagnoses, although in some cases they may lead to confusion. The interpretation of genetic studies is by no means simple and requires training. Clinicians will have to become increasingly more familiar with the specific nomenclature of these studies and with searching for variants in the international databases set up for sharing knowledge about these variants. Genetics cannot be separated from clinical practice and from the study of families, because this could lead to an unsuitable appraisal of the results, as exemplified by this clinical case. The limitations of genetic studies and what may be expected of them should be explained in consultations. Being a carrier of one or several genetic variants in a specific gene is not synonymous with disease. Moreover, ADPKD is characterised by major intrafamilial variability, whereby, and even if the genetic variant is shared, a disease may evolve very differently between different individuals due to mechanisms as yet unknown.[2,3] However, despite these limitations, until now, genetic studies play a major clinical role in the management of ADPKD and are essential in being able to make an early diagnosis of the disease. There is still a certain degree of controversy as to what the most adequate time for screening the children of affected patients is, since there is still no curative treatment, although neither is there any doubt that patients who carry hereditary diseases are entitled to a proper genetic assessment.[4,5] The autosomal dominant polycystic kidney disease (GEEPAD) study group has demonstrated that in 60% of cases ADPKD is diagnosed at a mean age of 34 years and after the birth of the first child.[6]

The only way to make an early diagnosis of ADPKD is to classify all families properly and perform genetic studies. Building family trees, conducting sequential genetic and segregation studies, when necessary, must also be addressed by nephrologists. Changing from an individual to a familial approach is essential to the proper management of this and other hereditary kidney diseases.

This clinical case highlights the need to combine clinical experience and genetics. This challenge will enjoy increasingly greater presence in daily healthcare work and is a change that we must adapt to.

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SARS CoV-2 seropositivity in haemodialysis patients

Seropositividad frente al SARS-CoV-2 en pacientes en hemodiálisis

Dear Editor,

We present a series of 52 patients with chronic kidney disease (CKD) on haemodialysis who presented SARS-CoV-2 infection diagnosed by nasopharyngeal swab polymerase chain reaction (PCR). The inclusion period was from 8 March 2020 until 21 February 2021, in which antibody testing for SARS-CoV-2 was performed during follow-up.

Mean age was 74.7 (±13.7) years, 80.7% were over 65; male sex predominated (73%), 7.6% were active smokers and 28.8% were former smokers. Of the main comorbidities, arterial hypertension (HTN) accounted for 96.1%, diabetes (DM) 69.2%, previous pulmonary disease 32.6%, coronary disease 25%, heart failure 23% and active cancer 3.8%. The most com-

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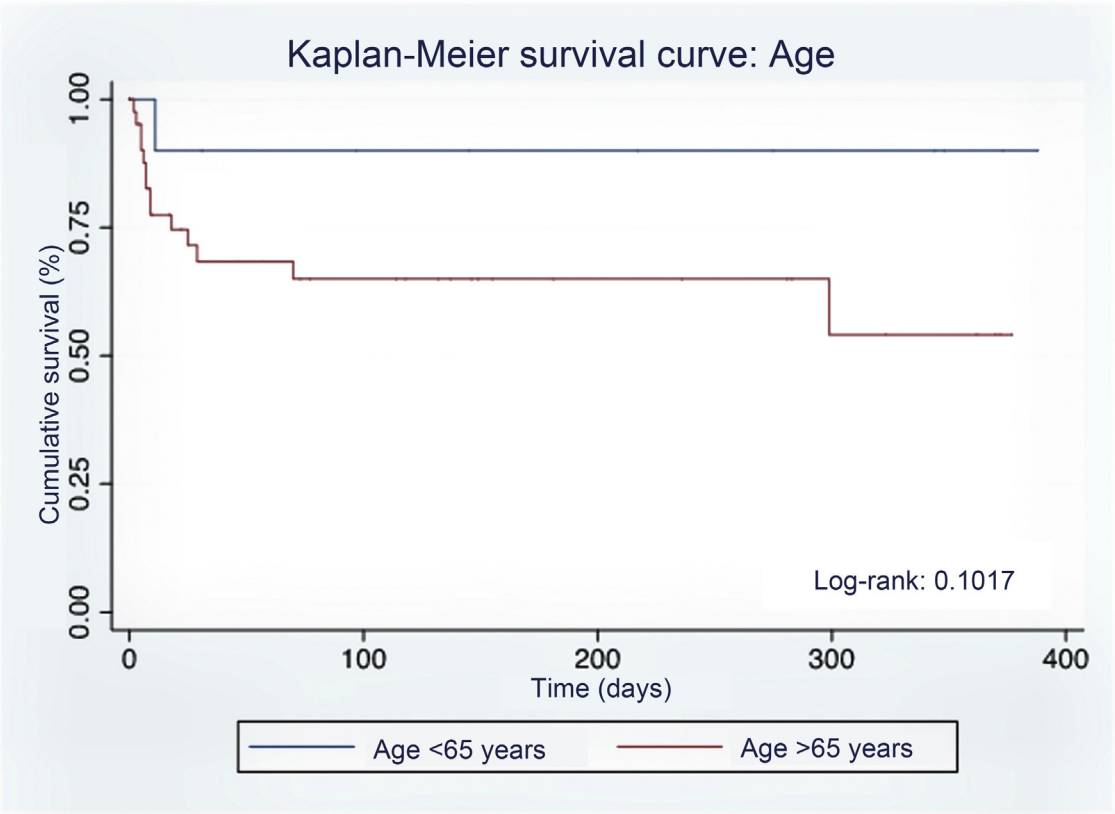

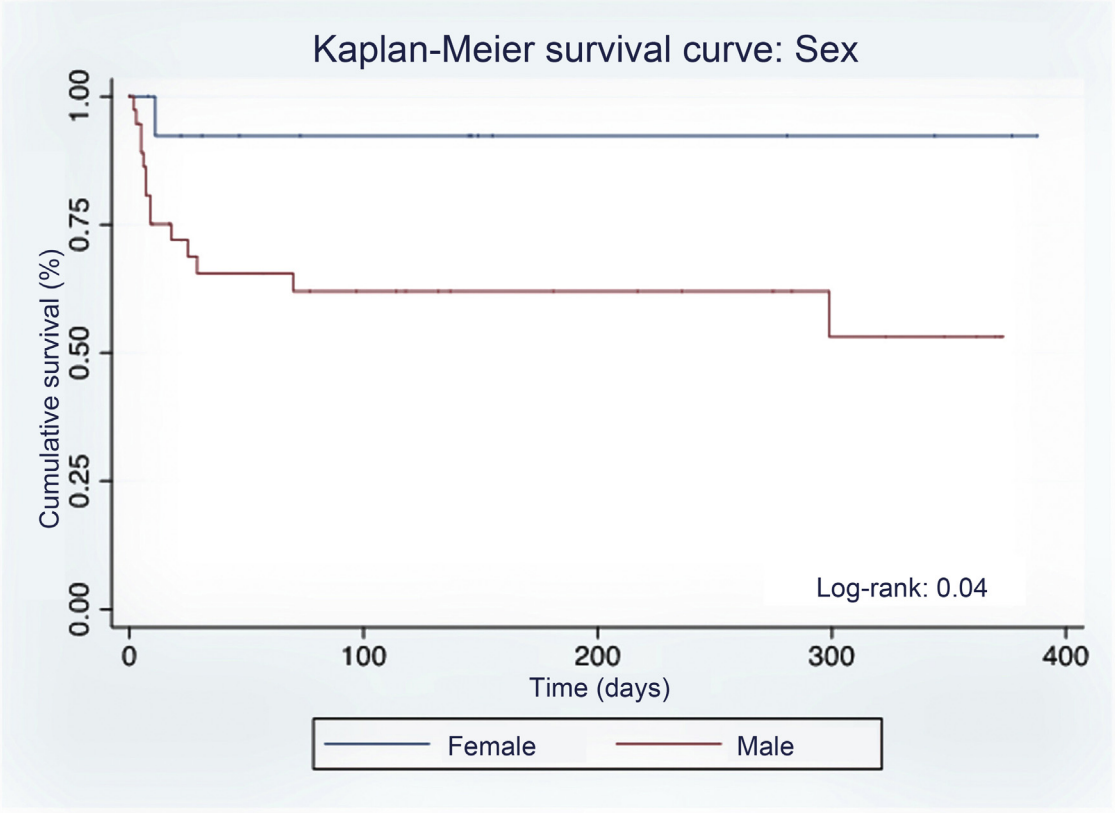

Figure 1 – Kaplan-Meier survival curve.

Table 1 – Characteristics of patients with SARS-CoV-2 infection.

Variables	Survivors (*n* = 36)	*Death* (*n* = 16)	*p*
Age, years	72.6 (15.6)	79.3 (7.6)	0.10
Age >65 years, %	63.4	36.5	0.10
Sex m/f, %	59.4/92.8	40.5/7.1	**0.02**
Arterial hypertension, %	67.3	32.6	0.32
Diabetes mellitus, %	72.2	27.7	0.39
Coronary heart disease, %	53.8	46.1	0.18
Heart failure, %	66.7	33.3	0.86
Chronic pulmonary disease, %	58.8	41.1	0.28
Smoking: active, former/no, %	47.3/84.6	52.6/15.3	**0.003**
Previous kidney transplantation, %	62.5	37.5	0.64
Lymphocytes, x10E9/L	0.89 (1.0)	2.0 (3.3)	0.08
C-reactive protein, mg/dL	8 (8.9)	12.7 (10.0)	0.14

M: male; F: female.

mon chronic kidney disease (CKD) aetiologies were diabetic and vascular, followed by undetermined aetiology. A 15.8% had received a kidney transplant and 3.8% were on the transplat waiting list.

The most frequent clinical symptom was fever (75%), followed by cough (42.3%), dyspnoea (40.3%) and GI symptoms (diarrhoea 9.6%, nausea 5.7%). Mean lymphocyte count at admission was 1.23 (±2.0) × 10E9/L and C-reactive protein was 9.3 (±9.2) mg/dL.

The treatment received was lopinavir/ritonavir (57.6%), azythromycin (34.6%), hydroxychloroquine (30.7%), tocilizumab (9.6%), with neither remdesivir nor high steroid doses (Fig. 1).

One of the 52 patients was managed as an outpatient. One patient was admitted to the Intensive Care Unit. Mortality was 31.3% (16 patients) due to SARS-CoV-2 pneumonia (31.3%), associated with being male and a smoker. There were no statistically significant differences in relation to the clinical and analytical variables (Table 1).

Of the survivors (36 patients, 68.6%), 19 patients (54.2%) were discharged home and 16 patients (45.6%) were transferred to a community health centre to continue their recovery. SARS-CoV-2 antibodies were determined in 33 patients using the (IgG Ab, Roche Cobas), 31 patients developed SARS-CoV-2 (91.6%) antibodies 39 days (21-104) after infection.

Chronic kidney disease patients on haemodialysis are known to present frequent infections plus a suboptimal response to vaccines, partly due to alterations in both innate and adaptive immunity[1,2].

CKD is one of the risk factors that have been associated with greater mortality from SARS-CoV-2 infection[3,4], since not only do these patients present immune system alterations, but further exposure to hospital centres renders them more vulnerable to contracting the infection.

Not a great deal is known about humoral response to SARS-CoV-2 infection in patients on haemodialysis. Sakhi et al. reported that seroconversion presented in 89% of the cases at a median 67 days post-diagnosis[5], and while these data are similar to our population in terms of seroconversion, the development of antibodies in our cohort occurred earlier, at a median 39 days.

In conclusion, immunological response to SARS-CoV-2 infection in our patients on a haemodialysis programme was good, with seroconversion occurring in the majority of the patients. Nevertheless, this population's future response to the SARS-CoV-2 vaccine is unknown.

Conflicts of interest

MJ Soler declares that he/she has provided scientific consulting or given presentations with Mundipharma, Fresenius, Bayer, Novo Nordisk, Janssen, Boehringer, Eli Lilly, AstraZeneca and Esteve, which were not related to this work.

Nestor Toapanta, Zaira Castañeda, José Zúñiga, Natalia Ramos, María Azancot state that they have no conflict of interest.

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Reticulocyte hemoglobin content and iron therapy in CKD

Concentración de hemoglobina reticulocitaria y ferroterapia en la ERC

ARTICLE INFO

Dear Editor,

We read with great interest the review entitled, "*Iron therapy in the management of anaemia in non-dialysis chronic kidney disease: the perspective of the S.E.N. [Sociedad Española de Nefrología (Spanish Society of Nephrology)] anaemia group*"[1]. In this manuscript, the authors provide an update on the management of iron deficiency in patients with chronic kidney disease (CKD).

It is clearly shown that the diagnosis and treatment of absolute iron deficiency is simple and that there is a broad consensus on that matter[1–3]. The same does not apply to functional iron deficiency. In such clinical situation, caused in most cases by inflammation, there is an increase in hepcidin synthesis (due to IL-6) in the liver[4]. Hepcidin blocks ferroportin, the only cell channel that exists for exporting cellular iron into the bloodstream, thereby reducing suitable availability of iron in the bone marrow. This leads to deficient haemoglobin synthesis in the reticulocytes[5]. Unlike mean corpuscular haemoglobin, whose value diminishes after several weeks, bone marrow iron deficiency may be estimated in a few days based on reticulocyte haemoglobin content (CHr)[5]. Therefore, all the guidelines recommend the percentage of hypochromic red blood cells or CHr as the best laboratory parameters for the diagnosis of functional iron deficiency (1B)[6–8].

Inexplicably, the authors[1] state that we must continue to use the classic markers (serum ferritin and transferrin saturation - TSAT), suggesting that the *new markers are less accessible, more expensive and somewhat unreliable*. We cannot convey this concept, since following the widespread introduction of automated cell counters, most laboratories can now measure number, volume and CHr and thus detect iron deficiency at an early stage[6]. Moreover, not only are red blood cell markers not expensive, they are also the most rewarding option in comparison to the different tests that assess FID and response to treatment in patients with CKD on haemodialysis or not[8]. Finally, these markers are very reliable. Mast et al.[9] demonstrated, in patients undergoing a bone marrow examination for other reasons, that their predictive value for iron deficiency is higher than the classic parameters (serum ferritin or TSAT). We have also seen the excellent correlation between CHr and the classic markers[10], which is why we believe that at this point in time they are accessible, cost-effective and very reliable and that their use should be recommended in accordance with the guidelines[6–8].

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