relevance. We look forward to future research that advances the care of CKD patients at risk of OP and fractures.

Conflict of interest

The authors declare no conflicts of interest.

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Yong Wang[a], Wei Zhou[b,*]

[a] *Department of Internal Medicine, Yanbian University Hospital, Yanji, Jilin 133002, China*
[b] *The Children's Hospital, Zhejiang University School of Medicine, National Clinical Research Center for Child Health, Hangzhou 310052, China*

[*] *Corresponding author.*
E-mail address: dracozhou@zju.edu.cn (W. Zhou).

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In response to optimizing osteoporosis management in CKD patients

Respuesta a: Optimizing osteoporosis management in CKD patients

Dear Editor,

In response to Drs. Yong Wang and Wei Zhou, we would like to express our gratitude for their interest in our national ERCOS study[1] and for their additional contributions. We all agree that this is another critical area of patient care that warrants further exploration as highlighted in the recent KDIGO Chronic Kidney Disease-Mineral and Bone Disorder (CKD-MBD) Controversies Conference.[2] Notably, a new conceptual framework was proposed to move to a framework of two clinical syndromes in adults: CKD-associated osteoporosis (OP) and CKD-associated cardiovascular disease. This approach aims to advance towards a personalized care in adults with CKD-MBD.[2]

The ERCOS study (Spanish acronym for CKD-OP)[1] was designed to intentionally include the profile of patients with CKD G3-5D diagnosed of densitometric OP *and/or* fragility fractures. By focusing on patients in whom these two highly prevalent conditions already coexist (as an inclusion criterion), the study does not allow conclusions to be drawn about the prevalence of OP in patients with CKD. Our

results confirmed a previously suspected general therapeutic nihilism, coupled with a notably low sensitivity/awareness of nephrologists regarding OP, despite recent important updates in our guidelines, consensus statements and calls to action.[2–5] We agree with the authors that including patients with CKD without prior OP diagnosis could provide a more comprehensive understanding of bone health across the entire CKD population. In fact, in our manuscript, we specifically emphasized that our results cannot be extrapolated to the entire population and must be interpreted with consideration of other potential biases, such as the small sample size (particularly on dialysis patients) and the involvement of centres and physicians with a specific interest in these conditions, among others.

The authors also suggest that including data from recently published studies on OP treatment outcomes in different CKD populations would have been interesting. Their data on treatment outcomes from recent studies is highly valuable and complements previous publications in *Nefrologia* and other journals.[2,5–7] However, we would like to reiterate that this was not the aim of our study though it could represent a valuable opportunity for a focused and comprehensive review.

Finally, the authors stated that incorporating emerging options such as romosozumab could provide a broader overview of available treatments. We fully agree, but unfortunately, when the ERCOS study was conducted, neither romosozumab nor abaloparatide was available in Spain. In fact, given the significant high prevalence of adynamic bone disease among CKD patients, OP treatment should not be limited to antiresorptives; anabolic or dual-action therapies (teriparatide, abaloparatide, romosozumab, etc.) should also be considered, although data on these agents remains even more limited (e.g. differences in femoral bone density benefits between abaloparatide and teriparatide, and warnings persist for romosozumab).[8,9] Thus, knowledge of bone turnover may also influence the choice of bone-targeted therapies.[2] In most cases, bone formation and resorption markers suffice for assessing bone turnover.[10] However, nephrologists should revive the practice of bone biopsies, at least in cases of complex bone diseases. While treatment decisions for most CKD patients can proceed without biopsies, they remain important for mineralization defects or complex bone diseases not identifiable noninvasively, particularly when bone markers are not aligned.[2,10]

In conclusion, we also fully agree with Drs Yong Wang and Wei Zhou in looking forward to future research that advances the care for CKD patients. This begins with the early diagnosis, classification and codification of patients with CKD, balancing renalism with therapeutic nihilism, and recognizing that bone and mineral disorders in CKD, including CKD-associated OP, may impact cardiovascular health and ageing. Personalized treatment approaches should especially be considered in CKD patients,[2] not only for those already diagnosed with OP and/or fragility fractures but also for those at risk. Given the challenges of conducting necessary randomized clinical trials on this issue, we propose an alternative approach: the prospective registration of key information by relevant scientific societies. This strategy could serve as an intermediate step in generating actionable knowledge and addressing current evidence gaps. Ultimately, we must remember that the best treatment for CKD-MBD is preventing the progression of CKD using the therapies already available.

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Conflicts of interest

Dr. Jordi Bover Sanjuán declares that he has received honoraria for conferences, consultancies and/or travel grants from Abbvie, Amgen, AstraZeneca, Bayer, CSL-Vifor, GSK, Rubió and Sanofi. Dr. Carlos Gómez declares having received fees for conferences and consultancies from Amgen, Italfármaco, FAES, Gedeon-Richter, Rubió, UCB and Sanofi. Dr. Enrique Casado declares having received fees for conferences and consultancies from Eli Lilly, Amgen, UCB, Theramex, Italfármaco, Gedeon-Richter, STADA, Bayer, GPPharma and Rubió. Dr. Juan Navarro states that he has received lecture and consulting fees from Abbvie, Amgen, Vifor Pharma, Rubió and Sanofi.

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Jordi Bover [a,*], Carlos Gómez-Alonso [b], Enrique Casado [c], Juan Francisco Navarro-González [d]

[a] *Servicio de Nefrologia, Hospital Universitario Gemrans Trias y Pujol, IGTP, REMAR y RICORS 2040, Badalona, Barcelona, Spain*
[b] *Unidad de Gestión Clínica de Metabolismo Óseo-Mineral, Hospital Universitario Central de Asturias, Oviedo, Asturias, Spain*
[c] *Servicio de Reumatología, Parc Taulí Hospital Universitari, Institut d'Investigació i Innovació Parc Taulí (I3PT-CERCA), Sabadell, Barcelona, Spain*
[d] *Unidad de Investigación y Servicio de Nefrología, Hospital Universitario Nuestra Señora de la Candelaria, Santa Cruz de Tenerife, Islas Canarias, Spain*

[*] *Corresponding author.*
E-mail address: jbover.ics@gencat.cat (J. Bover).

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Obinutuzumab en glomerulonefritis focal y segmentaria resistente al tratamiento

Obinutuzumab in focal segmental glomerulonephritis resistant to treatment

Sr. Director,

El obinutuzumab, es un anticuerpo monoclonal anti-CD20 humanizado de tipo II del isotipo IgG 1, de uso inicialmente en neoplasias de células B resistentes a rituximab (RTX). Actualmente están en curso estudios aleatorizados, con buenos resultados comparados con otros anti-CD20 en glomerulonefritis membranosa (GNM) y nefropatía lúpica (NL)[1,2].

Presentamos el caso de un varón de 63 años con antecedentes de hipertensión arterial, en seguimiento en consulta externa de nefrología desde 2015 por síndrome nefrótico clínico y analítico: proteinuria de 10 g/24 h, albumina 2,4 g/dl, colesterol 308 mg/dl, sistemático de orina con proteínas +++ y hematíes dismórficos además de edemas en miembros inferiores.

Se amplía el estudio glomerular (autoinmunidad, serología, complemento, inmunoglobulinas) resultando negativo, y presentando función renal normal. Se realiza biopsia renal en julio/2015 con resultado compatible con glomerulonefritis de cambios mínimos (GNCM).

Inicialmente (en 2015) se trató con pauta de corticoides con prednisona a dosis 1 mg/kg/día/durante 16 semanas presentando respuesta parcial con proteinuria de 2,2 g/24 h. Ante un nuevo brote de síndrome nefrótico clínico con empeoramiento de edemas y bioquímico con proteinuria 8,7 g/24 h, albumina 2,7 mg/dl en diciembre/2016, al intentar bajar la dosis de prednisona a 5 mg/día, se le catalogó como corticodependiente.

Se reinició tratamiento con prednisona a dosis de 1 mg/kg/día con lo que presentó mejoría parcial: descenso de la proteinuria a 2,4-3,5 g/24 h en junio/2017. En vista de las recaídas y la corticodependencia del paciente y siguiendo guías terapéuticas KDIGO se decidió asociar al tratamiento tacrolimus desde junio/2017 a julio/2018, con proteinuria mínima de 2 g/24 h en el control de febrero del 2018[2,3].

Posteriormente presentó nuevo brote de la enfermedad a nivel bioquímico con proteinuria de 5,8 g/24 h e hipoalbuminemia por lo que se suspendió el tacrolimus. Se volvió a tratar con corticoides y se añadió ciclofosfamida vía oral 100 mg/24 h/por vía oral/durante 8 semanas.

En noviembre del 2019 presentó un nuevo brote de síndrome nefrótico siendo tratado con micofenolato de mofetilo (MMF), como cuarta línea de tratamiento, disminuyendo la proteinuria a 0,5 g (mayo 2022). Se suspendió en mayo del 2022 debido a cuadro de hematuria macroscópica y tumoración renal izquierda. Se realizó nefrectomía parcial, siendo compatible la pieza histológica con hematoma organizado. Se aprovechó la pieza histológica para remitirlo de nuevo a anatomía patológica y realizar microscopia electrónica, dada la mala evolución del paciente con GNCM. El resultado al microscopio óptico: Enfermedad por cambios mínimos, extensa nefritis tubulointersticial aguda. Con el microscopio electrónico: glomerulonefritis focal y segmentaria (GNFYS). Se realizó estudio genético no observándose mutaciones asociadas. Con este resultado y tras un nuevo brote bioquímico en octubre/2022 (proteinuria 8 g/día, sistemático de orina activo y albumina 2,2 mg/dl), se reinició inicialmente MMF, cambiando posteriormente a tacrolimus, ante la falta de respuesta.