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https://doi.org/10.1016/j.nefroe.2023.03.013

Reply to the letter to the editor regarding "Nonvalvular atrial fibrillation in patients undergoing chronic haemodialysis, should we anticoagulate?"

Respuesta a la carta al director referida a «Fibrilación auricular no valvular en pacientes en hemodiálisis crónica. ¿Debemos anticoagular?»

Dear Editor,

We would like to thank our colleagues Aleix Cases et al. for the observations described in the letter to the editor sent on the occasion of the publication of the article N̈onvalvular atrial fibrillation in patients undergoing chronic haemodialysis, should we anticoagulate?;[1] fundamentally, because we are in total agreement with them and because they have helped to emphasise the conclusions referred to in our original review.[1]

Thus, we agree that tools other than the CHA_2DS_2-VASc have recently been proposed to assess bleeding risk in the haemodialysis (HD) population, given the fact that this *score* has not been shown to be useful in these patients, we did not reference it in our original article. The article referenced in the original review (Ocak et al., reference 40 of the original article) alludes to other *scores* that could not be validated in chronic kidney disease (CKD) to assess the indication for anticoagulation in these patients either.[2] Others like the modified CHADS[3] and the *dialysis risk score*,[4] proposed by Vriese et al., also need to be validated in the HD population. Therefore, we emphasise the low predictive value of these scales used in the general population to assess bleeding risk in HD patients, including the classic HAS-BLED scoring system developed to assess the one-year risk of major bleeding in patients with atrial fibrillation and which was developed in 2010 with data from 3978 patients in the *Euro Heart Survey*.[5]

The authors of the letter to the editor also agree with us regarding the increased risk of calciphylaxis and peripheral vascular disease that the use of vitamin K antagonists (VKA) produces in HD patients, as demonstrated in the articles that we referenced in the original review. We chose to use the term 'vascular health' to include these and other vascular diseases, but perhaps we should have been more explicit. We appreciate the clarification.

Likewise, we agree on the difficulty of achieving adequate coagulation control in HD patients, as described in the headings Ḧigh bleeding risk and lack of efficacyänd Öbjective anticoagulation level difficult to control;̈ on discussing the use of VKAs in the HD population. Additionally, the time in the therapeutic range is low in CKD patients, especially in HD patients, a fact that we should be aware of in this population treated with VKA in order to consider all treatment strategies.

We appreciate the mention of another ongoing clinical trial, such as the *Danish Warfarin-Dialysis Study Safety and Efficacy of Warfarin in Patients with Atrial Fibrillation on Dialysis* (DANWARD) (NCT03862859), which, along with the study mentioned in the original publication (AVK [VKA]-Dial), will provide greater knowledge about the risk-benefit of the use of VKAs in the dialysis population. We would add two further randomised studies with apixaban vs no anticoagulation (EDTA PARIS 2022):

- SACK (Sweden, Norway, Finland, Iceland and Poland), comparing 2.5 mg × 2 tablets apixaban vs no anticoagulation.
- ApiDP (France), nonrandomised, apixaban 5 mg × 2 vs normal kidney function.

Fortunately, the initiation of new clinical trials highlights the concern of this issue in the nephrology and medical community in general.

In the letter to the editor, reference is also made to the warfarin-related nephropathy mentioned in the original review, both acute and possible progression of CKD, included in the recent review of Gómez-Fernández et al.[6] As we pointed out in reference 103 of the original article (Brodsky et al.[7]), oral anticoagulant nephropathy has also been reported with direct-acting oral anticoagulants,[7] as well as the possibility of undiagnosed IgA nephropathy (references 104 and 105 of the original article).[8,9]

We also agree that percutaneous appendage closure is a therapeutic alternative without risk, as inferred from a recent publication[10] already referenced in the letter.

In summary and to conclude, we would like to express our satisfaction, given that, in the points referred to in the letter to the editor and which, again, we are grateful for, the authors

DOI of original article:
https://doi.org/10.1016/j.nefro.2022.05.007.

express their agreement with the conclusions we came to in the original review, to which we refer the reader,[1] and they demonstrate the interest in this subject that is aroused, which translates into a dynamism in the studies that are initiated and in the publications that arise in this regard.

Funding

This study received no specific funding from public, private or non-profit organisations.

Conflicts of interest

The authors have no conflicts of interest to declare.

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https://doi.org/10.1016/j.nefroe.2023.03.014

Comments on the consensus document on autosomal dominant polycystic kidney disease of the SENefro

Comentarios sobre el Documento de consenso de poliquistosis renal autosómica dominante de la SENefro

Dear Editor,

I have read the Consensus Document on Autosomal Dominant Polycystic Kidney[1] that the Spanish Society of Nephrology (Sociedad Española de Nefrología, SEN) drafted and published on its website and I would like to make some of comments on what has been exposed in that document.

They are based on the lack of definition of the term "rapid progressor" by the EMA[2] to establish some criteria that seem to me to be inaccurate. Anyone who starts renal replacement therapy before the mean global age for starting renal replacement therapy in Spain (65 years old) is considered a rapid

DOI of original article:
https://doi.org/10.1016/j.nefro.2021.04.017.