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"Echocardiographic response" to sacubitril-valsartan: does it decrease defibrillation implantation, as well as the incidence of malignant arrhythmias? Response

«Respuesta ecocardiográfica» al sacubitrilo-valsartán: disminución de la implantación de desfibriladores, pero ¿también de la incidencia de arritmias malignas? Respuesta

To the Editor,

We greatly appreciate the letter by Jáuregui et al.[1] regarding our recent publication. However, we would like to take the opportunity to clarify certain points.

Due to the retrospective and observational design of our study, we agree that causality cannot be assumed. Patients who were lost to follow-up discontinued sacubitril-valsartan (SV) early, or died before completing titration were excluded because no follow-up evaluation to assess the impact of SV can be made in these circumstances. Of 30 patients excluded, 7 patients (23%) died before completing titration. All of them died due to heart failure progression. No other exclusion criteria related to patients' risk were used.

Currently, SV is an essential part of heart failure treatment because of its proven prognostic benefit in reducing cardiovascular death, including sudden cardiac death and arrhythmic events.[2,3] The prognostic benefits of SV are probably mediated by reduced wall stress, ventricular dilatation, cardiomyocyte injury, hypertrophy, and fibrosis, which are factors related to arrhythmias.[2,4] Therefore, through these positive effects on reverse remodelling, myocardial stretch and fibrosis progression, the arrhythmic risk might be modified by SV treatment.[3] Of note, some of the recent studies evaluating arrhythmic risk cited by Jáuregui et al. included patients from retrospective cohorts who were not treated with SV.[5]

Interestingly, the authors focus on the fact that accurate arrhythmic risk stratification, especially in dilated cardiomyopathy, may include parameters other than left ventricular ejection fraction $\leq$ 35% as late-gadolinium enlargement detected by cardiac magnetic resonance.[5] Although this new approach is exciting and will probably change future clinical practice, it is not yet validated in external populations or included in current guideline recommendations. In our opinion, these new clinical algorithms of arrhythmic risk stratification are not incompatible with the fact that heart failure disease-modifying therapies such as SV should be implemented as early as possible and preferably before consideration of implantation of cardiac devices.

FUNDING

This research received no external funding.

AUTHORS' CONTRIBUTION

L.C. Belarte-Tornero and N. Farré contributed to the conceptualization and writing of the manuscript. All authors discussed and approved the final version of the manuscript.

CONFLICTS OF INTEREST

L.C. Belarte-Tornero report grants, honoraria for lectures and support for attending meeting from Novartis, Rovi, and AstraZeneca. N. Farré report honoraria for lectures from Novartis and Rovi.

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Available online 09 October 2021

https://doi.org/10.1016/j.rec.2021.09.007

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