LETTER TO THE EDITOR

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Autoimmunity-related atrial fibrillation incidence: an emerging conundrum meriting further investigation: authors' reply

This is a response to the Letter to the Editor, 'Autoimmunity-related atrial fibrillation incidence: an emerging conundrum meriting further investigation' by Moysidis *et al.* https://doi.org/10.1093/europace/euad076, about the article, 'Autoimmune diseases and new-onset atrial fibrillation: a UK Biobank study' by MJ Tilly *et al.* https://doi.org/10.1093/europace/euac244.

We greatly appreciate Moysidis *et al.*[1] for taking the time to read and comment on our recently published study regarding the association between autoimmune disease and new-onset atrial fibrillation (AF) in the UK Biobank.[2] In their letter, Moysidis *et al.* further elaborate on the complicated links through which autoimmune disease, potentially through underlying inflammation, and AF are related. We appreciate their additional summarization of the supporting and contradicting results that have been published earlier. We also commemorate their additional comments on the relation of other markers reflecting underlying inflammation, including inflammatory biomarkers and anti-inflammatory treatment, with AF.

We agree that further investigation is essential to shed further light on the complexity of the relation between inflammation and cardiac arrhythmia. Indeed, in line with our recent conclusions, multiple studies have shown that inflammatory biomarkers are associated with AF.[1] However, while large-scale, population-based studies are excellent to identify novel risk factors, the conclusions regarding the underlying pathophysiology often remain hypothesis generating. Single biomarkers, especially inflammatory markers, lack sensitivity and specificity to accurately predict the risk of AF.[3] Therefore, identification of novel therapeutic options requires more in-depth examination of the underlying inflammatory pathways. A recent review has shown that immunity-related cardiac remodelling is inherently connected to atrial electrical, structural, and neural remodeling.[4] Moreover, (ventricular) cardiac remodelling has been proved to be related to multiple, but not all, inflammatory pathways.[5] By investigating these inflammatory pathways with respect to atrial structural, electrical, and biochemical remodelling, the underlying pathophysiology of AF development may be uncovered. Moreover, through understanding of the exact mechanisms underlying AF, new therapeutic options for AF prevention and management may be uncovered.

Conflict of interest: None declared.

References

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