

Letter to the Editor

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Inflammation, arrhythmia burden and the thrombotic consequences of atrial fibrillation

We read with great interest the article by Dernellis and Panaretou¹ demonstrating that the use of methylprednisolone among patients with a single previous episode of persistent atrial fibrillation (AF) was associated with less recurrent AF episodes, which may possibly be explained by a reduction in circulating C-reactive protein (CRP) levels. The accompanying editorial² provides a brief overview of the existing evidence for an association between CRP and AF, and concludes that there is “great clinical potential” for anti-inflammatory therapies in the prevention and treatment of AF. We concur with the latter, and wish to suggest that anti-inflammatory strategies may not only reduce the *arrhythmia burden* of AF but also favourably alter the *prothrombotic or hypercoagulable state* associated with AF, which leads to the high risk of stroke and thromboembolism in this condition.

Indeed, we reported high plasma levels of CRP and interleukin-6 (IL-6) among patients with AF, which were independently related to indices of the prothrombotic state in AF (e.g., CRP to fibrinogen, IL-6 to tissue factor).³ In another study of patients with AF, we found a relationship between CRP and the presence of dense spontaneous echocardiography contrast in the left atrium or the left atrial appendage on transoesophageal echocardiography,⁴ which is a well-recognised independent predictor for stroke and thromboembolism in AF. Whilst the prognostic significance of CRP and IL-6 in the prediction of vascular events is well established among general populations,⁵ we have also demonstrated that indices of the prothrombotic state and inflammatory markers (IL6, but not CRP) may predict stroke and vascular events in AF.^{6,7}

Clearly, the effect of anti-inflammatory strategies upon the prothrombotic state needs to be established. Dernellis and Panaretou¹ state that, “The AF inflammatory hypothesis needs more study to be widely acceptable”.

Although further work is needed on dissecting the confounding effect of vascular disease (itself associated with abnormal CRP and inflammatory markers), we broadly agree with their statement. However, we emphasise that the possibility that anti-inflammatory strategies might prevent both the arrhythmia burden *and* the thrombotic consequences of AF must not be overlooked.

References

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Relationship between C-reactive protein concentrations during glucocorticoid therapy and recurrent atrial fibrillation

I read with great interest this potentially landmark study by Dernellis and Panaretou.¹ The authors demonstrated that CRP concentration is a risk factor for recurrent and permanent atrial fibrillation (AF).¹ In addition, they also showed that treatment with oral methyl-prednisolone, given for up to four months following successful cardioversion for ‘persistent’ AF, prevented AF recurrence or the development of permanent AF. The authors have thus suggested a novel way of improving sinus rhythm maintenance in cardioverted patients with AF of short duration (average duration 6.1 h), but without background inflammation.

In their study, 43–44% of included patients were diagnosed with hypertension at inclusion.¹ The authors do not provide information on the number of included patients with cardiovascular disease but without the exclusion criteria of acute myocardial infarction or unstable angina.

In addition, no information is provided on the background medical therapy with respect to the treatment versus placebo groups. Recent studies have demonstrated the efficacy of both angiotensin converting enzyme inhibitors and angiotensin receptor blockers as adjuncts to successful rhythm control in atrial fibrillation.^{2–4} In addition, there is also emerging data to support improved sinus rhythm maintenance with statin treatment.⁵ This is of importance as these mentioned treatments have been known to have anti-inflammatory actions with consequent reduction in C-reactive protein levels.

References

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