Is atrial fibrillation an inflammatory disorder?

I read with great interest the excellent review on the influence of inflammation in the pathogenesis of atrial fibrillation (AF) by Boos et al.1 As the authors have demonstrated, there is compelling evidence supporting the role of inflammation in the pathogenesis of this arrhythmia. I was surprised, however, to find no mention of the possible efficacy of beta-blockers with anti-inflammatory properties in this respect. Carvedilol, in particular, is a slightly beta 1-selective beta-blocker, which also possesses alpha-blockading and antioxidant properties.2 Indeed, part of its reported beneficial effects on ventricular remodelling effects and coronary microcirculation has been attributed to its antioxidant activities.2 Recently, we have provided evidence that carvedilol is probably more efficient than bisoprolol in the prevention of AF recurrences in an unselected patient population.3 In our study, 90 patients undergoing cardioversion of persistent AF were randomized to bisoprolol 5–10 mg once daily or carvedilol 12.5–25 mg twice daily. By intention-to-treat analysis, 23 (46%) patients in the bisoprolol group and 17 (32%) patients in the carvedilol group relapsed into AF, during the 1 year of total follow-up period (P = 0.486). Patients treated with carvedilol had a 14% (hazard ratio = 0.86) lower risk to relapse to AF when compared with patients on bisoprolol group. This issue deserves closer attention, particularly when discussing the limitations of current anti-arrhythmic drugs as far as their anti-inflammatory action is concerned.

References

anti-hypertensive regimes has been associated with a risk reduction of the order of 0.51 (95% confidence interval 0.42–0.62) in the incidence of congestive heart failure,\(^1\) the absence of scrutiny of these drugs, to which the authors allude,\(^2\) has also included the failure to address the issue of whether the anti-hypertensive efficacy of long-acting loop diuretics such as torasemide might be comparable to that of thiazides, and whether, for both classes of drugs, the anti-hypertensive efficacy might be solely attributable to sustained natriuresis. A related issue is whether the protection that thi-azides confer against hypertension-related heart failure might be rivalled, if not surpassed, by diuretics such as torasemide, which potentially possess cardioprotective properties by virtue of additional anti-aldosteronergic effects.\(^3\) The time is long overdue for these issues to be addressed, given the inescapable risk of hyponatraemia (including severe hyponatraemia) inherent in the use of thiazides,\(^4\) by virtue of their physiological actions on the renal tubule and collecting ducts.\(^5\)

References


http://eurheartj.oxfordjournals.org/content/37/6/876.full.pdf