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Letters to the Editor

Is the level of resistin appropriate for predicting atrial fibrillation?

We read the paper entitled "Increased level of resistin predicts development of atrial fibrillation" by Ozcan et al. [1] with interest. These authors stated that plasma resistin and the high sensitivity C-reactive protein levels were the only independent predictors of atrial fibrillation (AF) and that elevated levels of plasma resistin were related to the paroxysmal AF group and the persistent AF group, but not to the permanent AF group.

Although several risk factors have been identified for the development of AF, the pathogenesis is multifactorial and not totally understood. Different factors are included in this self-perpetuating process such as volume and/or pressure overload in the heart, fibrosis, oxidative stress, and inflammation. Since cardiac inflammatory disorders such as myocarditis, pericarditis, and post-periocardiotomy syndrome frequently are accompanied by AF, clinicians concentrate upon the idea that AF is closely associated with inflammation according to these clinical observations [2]. Previously, inflammatory markers have been assessed in the cardiac tissue, intracardiac blood, and peripheral blood in patients with AF. Furthermore, in one study, higher C-reactive protein (CRP) and interleukin-6 levels were detected in the left atrium than in the coronary sinus during AF and the authors concluded that there appears to be intracardiac sequestration of inflammatory cytokines, potentially pointing to an important mechanism of atrial remodeling [3]. However, if the blood samples of this study had been collected from left atrium or coronary sinus, results different from that study could have been obtained, and especially the atrial inflammation could be more accurately evaluated.

The authors also stated that the permanent AF was associated with higher levels of CRP than paroxysmal AF, implying that CRP levels may be related to the burden of AF, but the same thing is not valid for resistin levels. The reason for these results may be due to the small number of patients included in the study as well as drug usage. Namely, circulating resistin levels are found to be decreased by the anti-diabetic drug rosiglitazone [4]. Moreover, reduced circulating resistin levels after rosiglitazone treatment have been reported by Moore et al. [5]. If the detailed history of drug usage of the patient population had been questioned, different results might have been obtained.

Furthermore, inflammation appears to play an important role in the development of thromboembolic complications associated with AF. The relationship between inflammation and AF-related thromboembolism was supported by the observation in which interleukin-6 and CRP are markedly elevated in patients with dilated left atrium and an inadequately functioning left atrial appendage. Moreover, CRP has also been shown to be correlated with identified clinical stroke risk stratification schemas ($CHADS_2$, SPAF) [6]. In the current study, it was not specified whether any thromboembolic events occurred in the one-year follow-up or if there was any relationship with predefined risk scores. The relation of the resistin levels with these factors should have been important in the prediction of thromboembolic events.

Finally, we would like to have much more detailed information from the authors of this study regarding drug usage and occurrence of thromboembolic events in follow-up. It would be much more accurate if they had collected the blood samples from the coronary sinus or left atrium. According to the results of this study, we think that plasma resistin levels may not be an appropriate marker in the prediction of the development of AF. To clarify this issue, further large-scale studies are needed.

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