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Inflammation, fibrillation (AF) compared with control group. Moreover, the ratio of circulating intermediate atrial appendage (LAA) flow during sinus rhythm. That the extent of atrial fibrosis as a hallmark for high-density lipoprotein cholesterol (HDL-C) and pro-oxidant cytokines. One of the important limitations of our study was using an automatically counted monocyte numbers rather than the proportion of monocyte subsets. Thus, the study by Suzuki et al. may support and confirm our previous findings when the ratio of circulating intermediate CD14+CD16+ monocytes to HDL-C levels has been calculated and analysed. Because MHR combines the two detrimental processes like inflammation and oxidative stress, it could be used as a novel marker for prediction of the severity of atrial remodelling. However, additional large-scale prospective studies in different populations are needed to confirm the role of MHR and/or circulating intermediate CD14++CD16+ monocytes/HDL-C in the pathophysiology and prognosis of AF.

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References

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Monocyte-to-HDL-cholesterol ratio and left atrial remodelling in atrial fibrillation: author’s reply

We thank Dr Canpolat1 for his interest in our recent study.2 Many studies have established a strong association between inflammation, oxidative stress, and atrial fibrillation (AF); the pathogenesis and progression of AF seem to be simultaneously influenced by multiple factors.3,4 Canpolat et al. showed in their study that among commonly measured clinical factors, the monocyte to high-density lipoprotein cholesterol (HDL-C) ratio (MHR)—a combined inflammatory and oxidative stress marker—was independently associated with AF recurrence after cryoenergy ablation and correlated with the left atrial (LA) diameter.5 In our study, we investigated the role of intermediate CD14++CD16+ monocytes in the pathogenesis of AF and showed that a monocyte subgroup, intermediate CD14++CD16+ monocytes, was independently associated with the presence of AF. In addition, intermediate CD14++CD16+ monocytes reflected LA functional remodelling. Of note, the total monocyte count in healthy controls and AF patients without any other obvious co-morbidities was not predictive for the AF status.2

In their letter to the editor, Canpolat et al. now hypothesize that the ‘intermediate CD14++CD16+ monocytes to HDL-C ratio’ is able to predict severity of LA remodelling. To this end, we performed a sub-analysis of our data including MHR and intermediate CD14++CD16+ MHR (iMHR). In addition, for a complete analysis, we looked at classical CD14++CD16– MHR (cMHR) as well as non-classical CD14+CD16+ MHR (nMHR). Univariate logistic regression analysis revealed that the MHR (odds ratio (OR): 1.041; 95% confidence interval (CI): 1.004–1.078, P = 0.027) and iMHR (OR: 2.057; 95% CI: 1.303–3.251, P = 0.002) both influenced the presence of AF. Furthermore, parameters such as body mass index, diastolic blood pressure (DBP), total cholesterol (T-choi), HDL-C, triglycerides (TG) and proportion of classical CD14++CD16– monocytes, and intermediate CD14++CD16+ monocytes also influenced the AF status. There was no statistical significant association of cMHR and nMHR values with AF (OR: 1.040; 95% CI: 0.997–1.085, P = 0.070 and OR: 1.259; 95% CI: 0.976–1.623, P = 0.076, respectively). Although iMHR was strongly associated with the presence of AF (P = 0.002), the cell proportion of intermediate CD14++CD16+ monocytes (collinear to MHR and iMHR) of all parameters had the strongest association with the presence of AF in univariate analysis (P = 0.001).

In contrast to intermediate CD14++CD16+ monocytes, the iMHR did not show significant correlation with any clinical and laboratory parameters including the duration of AF, echocardiogram parameters, BNP level, and LA volume in AF patients.

There are several limitations inherent to the different design of our present study and in the study of Canpolati et al. First, to avoid bias of other systemic diseases, our study population was limited to patients without any co-morbidities. Secondly, mean HDL-C level in our study