Authors' response

Sir,

We thank Kotani and colleagues for the appreciation of our work[1]. In view of the published data[2-4], we agree that oxidative stress might be potentially involved in the pathogenesis of atrial fibrillation. Increased blood viscosity has been identified as a sensitive marker for underlying oxidative stress[5,6]. It is tempting to speculate that our findings might be regarded as surrogate parameter for oxidative stress, showing an association between haematocrit and the occurrence of atrial fibrillation after acute myocardial infarction. The fact that in our study the haematocrit level did not show a similar impact on the occurrence of atrial fibrillation in a manner proportional to the haemoglobin and red blood cell counts might be purely owed to the small sample size and the retrospective study design. The blood count levels in our predominantly male study population (61%) were more in the upper normal range (95% percentiles: haemoglobin 15.9 mg/dl, haematocrit 47.8 per cent, erythrocyte count 5.3 T/l). We agree with Kotani and colleagues that the measurement of oxidative stress and antioxidant capacity might reveal valuable additional information and should be addressed by further studies. We also agree that unification of the timing of blood sample collection and additional clinical variables (*e.g*. heart rate and blood pressure prior to onset of atrial fibrillation, fluid therapy, *etc*.) is of potential interest. However, due to the retrospective nature of our nested-case control study we were not able to provide this information and should, therefore, be evaluated by future prospective studies. The present study was not designed to reveal the underlying pathophysiological mechanisms of new onset of atrial fibrillation after acute myocardial infarction, but was purely conducted to identify novel predictors, that might be objective of further studies.

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