Background: The relevance of the association between inflammation and atrial fibrillation (AF) is not firmly established. The clinical importance is considerable because inflammation is usually not targeted as a treatment option, minimizing a probable benefit.

Methods: We have used a case-control study to assess whether proposed risk factors that have a genetic component and are readily detected in circulating blood are causally related to AF. We studied a group of 249 AF patients (p) without structural heart disease. Both p. and control group (n=375) were less than 70 years. The studied variables were C-reactive protein (CRP) and monocyte chemoattractant protein-1 (CCL2).

Results: Plasma CRP [1.49 (0.7-3.5) vs. 1.38 (0.4-2.9) mg/L; p=0.029] and CCL2 [117.6 (104-135) vs. 78.9 (61-109) pg/mL; p<0.001] concentrations were significantly higher in AF p. than in controls. However, when segregated between paroxysmal (n=140) and permanent (n=109), the difference for CRP was only observed in p. with a permanent condition [1.79 (0.9-4.5) vs. 1.38 (0.4-2.9) mg/L; p=0.022]. Plasma CCL2 was raised in both subgroups and no difference among them was observed (P=0.716). Multivariate analysis revealed that circulating CCL2 was significant [OR=1.02 (1.01-1.02), P<0.001] and CRP was negligible [OR=0.97 (0.93-1.02), P=0.256] to explain the presence of AF. Odd ratios for AF as a function of genotype did not differ from 1.0 for any of the individual CRP and CCL2 polymorphisms, or any combinations. However, we found that plasma protein levels increased in association with certain individual genotypes both in controls and p.. Plasma CRP concentration was significantly higher in carriers of: rs1130864 A/A (P<0.001), rs1417938 A/A (p<0.001), rs12728740 A/A (p<0.001) and rs3093077 C/C (P=0.009). For CCL2 this association was only found in carriers of rs3760399A/G polymorphism (P=0.002).

Conclusions: Elevated plasma CRP concentration per se does not increase AF risk. Values obtained for CCL2 suggest that inflammation is probably a consequence of AF. Our data also suggest that the effect of the duration of the episode should be further studied in the assessment of the actual role of the different markers of inflammation.