To the Editor: We read with great interest the article by Cortez et al.1 “Prognostic value of C-reactive protein in resistant hypertension” dealing with the relationship between the levels of C-reactive protein (CRP, a biomarker of systemic low-grade inflammation) and cardiovascular risk in resistant hypertension. The results of their study demonstrated that, after adjustments for potential confounders including traditional cardiovascular risk factors and ambulatory blood pressure and dipping pattern, resistant hypertensive patients with CRP levels above the median value (3.8 mg/l) had a doubled excess risk of major cardiovascular events and a higher risk of cardiovascular death. In addition, a high CRP equally predicted coronary and cerebrovascular events. The authors proposed that, in interaction and sensitivity analyses, CRP levels might be stronger predictors of worse cardiovascular outcomes in younger and obese patients and in those with uncontrolled ambulatory blood pressures with the nondipping pattern.

Current evidence indicates that not only inflammation but also impaired endothelial function may strongly be related to the increased cardiovascular risk associated with resistant hypertension. Quinaglia et al.2 proposed that patients with uncontrolled resistant hypertension might have less nocturnal dipping and a more impaired endothelial response compared with controlled hypertensive subjects.

Conversely, we previously demonstrated that the plasma high-sensitivity CRP levels were inversely correlated with plasma nitric oxide (NO) metabolites in the overall analysis of hypertensive subjects. It was also demonstrated that increased levels of high-sensitivity CRP were associated with reduced endothelial-mediated dilatory responses of the arteries.3 In this context, it is strongly suggested that elevated inflammatory status might be accompanied by the reduced NO production and endothelial dysfunction. Therefore, we would like to know whether impaired endothelial function might be associated with the increased levels of CRP and whether endothelial function might also have a predictive value for cardiovascular outcome in subjects with resistant hypertension in the study of Cortez et al. It would be important to assess more precisely the relationships between inflammation and endothelial dysfunction, and their contribution to the progression of cardiovascular and cerebrovascular diseases in subjects with resistant hypertension.

DISCLOSURE

The author declared no conflict of interest.

REFERENCES