Dear Editor

We read with great interest the article “Postmenopausal Therapy Reduces Catalase Activity and Attenuates Cardiovascular Risk” by Castanho et al., in which the authors investigated serum catalase activity by monitoring the serum concentration of lipids and lipoproteins, cholesteryl ester transfer protein, thiobarbituric acid-reactive substances, nitrate, and high-sensitivity C-reactive protein in postmenopausal women with or without hormone therapy (HT) to determine whether postmenopausal HT administration influences free radical production (proinflammatory marker). They demonstrated that HT in postmenopausal women produces beneficial antioxidant and antiatherosclerotic effects by ameliorating the plasma lipid and lipoprotein profiles, increasing plasma catalase activity, and attenuating the association between cardiovascular risk factors and early atherosclerosis.

Endothelial dysfunction plays a key role in atherogenesis, as it is associated with all major risk factors for cardiovascular disease. Ultrasonography is a noninvasive method to assess endothelial function. Carotid intima–media thickness (C-IMT) is a marker of systemic inflammation and widely used as a prominent marker for cardiovascular diseases. C-IMT may be affected by cardiovascular risk factors, including hypertension, diabetes mellitus, obesity, and tobacco smoking. From this viewpoint, Castanho et al. did not clearly reference detailed patient medical records, which would have been quite useful.

Flow-mediated dilatation (FMD) by insufflating a sphygmanometer cuff is a useful ultrasonographic parameter of the brachial artery to measure vascular responses through variations of the basal artery diameter induced by reactive hyperemia that follows re-establishment of blood flow after a period of vascular occlusion and presents an easy method to assess endothelium-dependent vascular function. Measurement of the ankle-brachial index (ABI) is useful to accurately screen and diagnose peripheral arterial disease (PAD) and allows timely initiation of preventive measures. When ABI is either <0.9 or >1.3, PAD is likely. C-IMT may also affect PAD. For this reason, the present study would be stronger if the authors would have provided information regarding potential PAD.

Finally, C-IMT alone, without other inflammatory markers, may not provide sufficient information to clinicians concerning endothelial inflammation in the patient. Thus, in further studies, we propose that C-IMT should be evaluated together with other serum inflammatory markers, such as FMD and ABI, because they can be monitored rapidly, reliably, and noninvasively at a low cost to identify early stages of atherosclerosis.

References