Response to Magnesium and Vascular Dysfunction in Malignant Hypertension

We are grateful to Van Laecke and Vanholder\textsuperscript{1} for their interesting observations and comments. We agree that molecular mechanisms of the persistent endothelial dysfunction in patients with malignant hypertension (MHT) observed in our study remain to be clarified, although this aspect was beyond the scope and aims of our published study because of the rarity of MHT as a condition.\textsuperscript{1}

It is plausible that possible electrolyte abnormalities (including magnesium) and also subclinical renal dysfunction in MHT patients could contribute to the endothelial and vascular dysfunctions. Indeed, magnesium transport abnormalities may be relevant to the vascular biology of hypertension.\textsuperscript{2} However Touyz and colleagues\textsuperscript{3,4} made their observations on MHT patients of black ethnicity origin, and this may not be the case in white patients, who are mainly represented in our study.

We also feel that the findings by Shechter et al\textsuperscript{5} that magnesium supplementation improves brachial artery flow-mediated dilation in patients with coronary artery disease, are not entirely relevant to our study in MHT, where no significant changes in flow-mediated dilation were seen in MHT compared with high-risk hypertension.\textsuperscript{5} Similarly, an interesting study referred to by the authors of the letter has been performed in renal transplant recipients, but this is a disease condition with numerous features of advanced vascular and metabolic abnormalities.

We did not attempt to perform sophisticated multivariate regression analysis to avoid introduction of possible error related to inadequate size of the study population. However, in MHT patients from our published study, serum creatinine levels had no significant predictive value for microvascular endothelial dysfunction ($\beta=-0.08$; $P=0.79$). Therefore, it is unlikely that rather small changes in creatinine levels between the groups had a significant impact on the study results. Although patients with MHT hypertension received less statins, they tended to receive more angiotensin-converting enzyme inhibitors, thus resulting in mutually smoothing effects on endothelial dysfunction. Nevertheless, we entirely agree with Van Laecke and Vanholder\textsuperscript{1} that further research is warranted to shed more light on the mechanisms of endothelial perturbation in MHT.

Disclosures

None.

Alena Shantsila
Girish Dwivedi
Gregory Y.H. Lip

University of Birmingham Centre for Cardiovascular Sciences
City Hospital
Birmingham, United Kingdom

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Alena Shantsila, Girish Dwivedi and Gregory Y.H. Lip

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