Successful therapy reduces levels of vascular endothelial growth factor (VEGF) in patients with hypertension and patients with hypercholesterolemia

Keywords: Vascular endothelial growth factor; Hypercholesterolemia; Hypertension

We read with interest the paper by Sumino et al. [1], who reported the effect of hormone replacement therapy (HRT) on serum levels of vascular endothelial growth factor (VEGF). They suggested that HRT reduced serum levels of VEGF in normocholesterolemic (NC) postmenopausal women (PMW) with hypertension. Conversely, HRT did not affect VEGF serum levels in the PMW with hypercholesterolemia (HC), of whom 74% were treated hypertensives.

We are concerned that their study was based on VEGF measurements in serum samples which has been shown to be inaccurate as this growth factor is released from platelets during clotting [2,3]. Indeed this may explain why some of their observations differed from our recent data.

Hypertension and HC are two of the main risk factors for the development of symptomatic atherosclerosis. We have reported raised plasma levels of VEGF in patients with peripheral artery disease and coronary artery disease when compared to healthy normotensive age and sex matched controls [4]. In a pilot study, we also investigated the effects of antihypertensive therapy on VEGF production in 27 untreated patients (15 men, aged 54 ± S.D. 18) with essential hypertension and 27 age and sex matched healthy controls. Plasma VEGF levels in untreated hypertensive patients were significantly higher than levels in healthy normotensive controls (median 130 pg/ml [IQR 10–2025] vs. 75 pg/ml [20–125] Mann–Whitney test, \( P < 0.05 \)). Furthermore, effective antihypertensive therapy lowered blood pressure and significantly reduced median plasma VEGF levels from 130 pg/ml [10–2025] to 60 pg/ml [10–235] after 2 months of therapy (paired Wilcoxon test, \( P < 0.05 \)). Sumino et al. were unable to detect a significant difference at baseline between their study group which was composed of PMW with or without HC and hypertension (66% of the study group), when compared to the control group, which were also not clearly defined and consisted of 60% hypertensives. This may be attributed to the fact that hypertension itself seems to influence VEGF production, and the proposed efficacy in reducing VEGF with HRT in PMW with NC may in fact be a confounding effect of hypertension and/or associated antihypertensive therapy rather than HRT.

We have also shown a relationship between plasma VEGF and HC. In a pilot study of 20 patients with total cholesterol of > 6.0 mmol/l, and 21 healthy controls, plasma VEGF levels were significantly greater at baseline in patients with HC when compared to controls (median 222 [100–437] vs. 75 [25–111] pg/ml; \( P < 0.05 \)). After 3 months of lipid lowering therapy, median plasma VEGF levels had fallen significantly, to 162 [100–450] pg/ml; \( P < 0.05 \). In contrast to the study by Sumino et al. [1], we suggest that proper management of HC leads to a reduction in plasma VEGF levels, which may in turn delay or prevent the onset of symptomatic atherosclerosis.

References


Funmi M. Belgore,
Gregory Y.H. Lip,
Andrew D. Blann

Haemostasis, Thrombosis and Vascular Biology Unit,
University Department of Medicine,
City Hospital, Birmingham B18 7OH, UK
E-mail: funmib@hotmail.com