Cerebral Blood Flow Changes in Elderly Hypertensive Patients and Cognitive Functions

To the Editor:

We read with interest the article by Beason-Held et al published in the June issue of Stroke.¹ Fourteen treated hypertensive patients and 14 age-matched healthy controls, aged 70.8±7.4 SD years at entry, are examined. The authors, using repeated positron-emission tomography, have found that hypertension is associated with longitudinal regional cerebral blood flow (rCBF) changes, assessed over a period of 6 years, which differ from healthy controls. In hypertensive patients, greater rCBF decreases are observed in prefrontal, anterior cingulate, occipital areas and hippocampus in comparison with controls. However, relevant cognitive impairments are not demonstrated. The duration of hypertension is found to contribute significantly to the rCBF changes over time.

Previous studies of the rCBF in hypertensive patients without cerebrovascular disease also report reductions in blood flow more marked in the frontal, temporal and parietal regions. The perfusion abnormalities are more severe in untreated, compared with treated subjects.²⁻⁴ It has been suggested that blood pressure control may preserve the global cerebral perfusion and reduce the cerebrovascular resistance.⁴

In patients with essential hypertension, without a history of stroke, quantitative volumetric MRI shows atrophies in the thalamic nuclei and temporal lobes, attended with poor memory performance. It is also found that hypertension exacerbates the brain atrophies because of advanced age. The strongest interaction of age and hypertension has been observed in the temporal and occipital lobes.⁵ Besides, it has been shown that hypertension in older subjects without overt vascular disease is associated with cognitive impairment.⁶ It is also found that older hypertensive patients display a cognitive impairment more pronounced in those with poorly controlled blood pressure.⁷

However, Beason-Held et al have not observed cognitive impairment in their hypertensive patients without other vascular risk factors, even in advanced age, although the blood pressure has not been adequately controlled. Undoubtedly, pathogenesis of vascular cognitive impairment is multifactorial, but the interaction of hypertension and age in their occurrence is well documented. The small sample size of this study has also hampered the evaluation of different antihypertensive drug effects over time. Because of a lack of MRI, cerebral small vessel lesions, often seen in hypertension, cannot be discussed. Despite the limitations of this study, the patterns of longitudinal rCBF changes in elderly hypertensive patients could be considered a subclinical stage of cognitive impairment.

Disclosures

None.

Dimiter Hadjiev, MD, PhD, DSc
University Hospital of Neurology and Psychiatry “St. Naum”
Medical University
Sofia, Bulgaria

Petya Mineva, MD, PhD
Medical Faculty
Thracian University
Stara Zagora, Bulgaria

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Dimiter Hadjiev and Petya Mineva

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