Cerebral Atherosclerosis and Mild Alzheimer’s Disease

To the Editor:

Qiu et al. observed in persons aged 75 years and older that higher pulse pressure is associated with increased risk for Alzheimer’s disease (AD) and dementia in older adults. In this respect, I agree with the authors that these conditions are caused by moderate or severe cerebral atherosclerosis, because the loss of arterial compliance leads to a rise in pulse pressure. Likewise, I believe that poor cerebral perfusion (hypoperfusion) can increase dementia risk. Therefore, I wish to add our personal experience in 85 patients (68 women and 17 men) with AD (unpublished observations). First, nongenetic AD represented 95.5% of all cases and the mean age at onset of the symptoms was 66 years (range, 45 to 84 years). Similar findings in 60 patients were previously reported by us elsewhere. Second, in all patients with AD, the onset of the symptoms were insidious, course undulating (periods of clinical improvement alternating with those of worsening), and progressive. Clinical data suggest that the etiologic agent is of vascular origin. Third, the early stage (mild degree) of this disease was characterized by (1) impairment of recent memory in 68.2% of cases (typical course, because this symptom is the cardinal change of AD) or (2) behavioral and personality changes in 31.8% (atypical course, because this symptom is the cardinal change of AD). Fourth, the earliest stages of AD are caused by progressive hypoperfusion and hypometabolism in the intraparenchymal territory of the anterior choroidal and anterior perforating arteries due to atherosclerotic plaques located at the mouths of these collateral branches. By contrast, an omental transplantation on the optic chiasma, carotid bifurcation, and the APS can ameliorate the clinical feature of AD.

Fourth, tomographic and neurosurgical findings demonstrated 3 important observations in the circle of Willis: (1) moderate and/or severe atherosclerosis of the supraclinoid portion of the internal carotid arteries and their terminal branches; (2) a variable number of exsanguinated and collapsed anterior perforating arteries originated from the carotid crotch and circle of Willis, and (3) some perforating branches with residual blood flow centripetal to the origin of the vessels. So, these pathological changes provoke progressive hypoperfusion and hypometabolism into the intraparenchymal territory of the anterior choroidal and anterior perforating arteries due to atherosclerotic plaques located at the mouths of these arterial branches. Thus, the neuronal injury by ischemia and ischemic penumbra triggers a pathophysiologic cascade of events such as inhibition of the Krebs cycle, elevation of the extracellular concentrations of glutamate and aspartate, and free radicals formation, among another changes. By contrast, in late stages (moderate and advanced degrees), irreversible damage to the biological molecules, neuronal death, astrogliosis, and cerebral atrophy occur. Therefore, based on the above-mentioned observations and our experience with omental transplantation on the APS in 9 patients with sporadic AD (mild degree in 1 case and moderate degree in 8), we believe that the complete reversal of symptoms in the mild AD patient was due to revascularization of cholinergic and neuropeptidic nuclei within the subcommissural regions, the olfactory tracts, and the left medial temporal lobe. At present, 5 years after one patient’s surgery, her quality of life is good and she can participate in activities of daily living similar to any normal woman of her age. In 8 other cases with moderate AD (progressive worsening of memory, behavioral and personality changes, higher cortical dysfunction, and posture and gait disturbances), we observed only neurological improvement, and the results were better during the first weeks after the surgery than in the following months. These clinical results suggest that this surgical procedure can cure (mild AD) or improve (moderate AD) this disease.

For these reasons, we believe that the earliest stages of AD are caused by progressive hypoperfusion and hypometabolism in the intraparenchymal territory of the anterior choroidal and anterior perforating arteries due to atherosclerotic plaques located at the mouths of these collateral branches. By contrast, an omental transplantation on the optic chiasma, carotid bifurcation, and the APS can improve this disease by revascularization of the subcommissural regions and neighboring areas.

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