middle cerebral artery: reference values at rest and during hyper-ventilation in healthy volunteers in relation to age and sex.


Background Factors and Clinical Symptoms of Major Depression With Silent Cerebral Infarction

To the Editor:
In their recent article, “Background factors and clinical symptoms of major depression with silent cerebral infarction,” Fujikawa et al. used magnetic resonance imaging to determine whether depressed patients had or did not have silent cerebral infarction and then compared the two groups in terms of a variety of risk factors for stroke and depression. It is surprising that the authors do not relate their findings to a large body of literature reporting similar results but using different terminology, i.e., leukoencephalopathy, leukoaraisos, deep-white-matter hyperintensity, or subcortical hyperintensity. These terms are essentially used to describe hyperintensities on T2-weighted spin-echo magnetic resonance images of the brain.

Small hyperintensities are related to perivascular spaces; larger hyperintensities (maximum linear dimension, > 5 mm) are usually seen on pathological examination to consist of areas of myelin pallor, infarcts, or lacunes. These large signal hyperintensities are the basis of patient classification by Fujikawa et al.5 A brief review of the literature that links these hyperintensities to depression may be of interest to your readers. Since the publication of our initial report indicating that these hyperintensities are common in elderly depressed patients,9 numerous researchers have noted the high frequency and severity of these hyperintensities in elderly depressed patients compared with control subjects.4,6 Coffey et al.6 also reported that lesions of the basal ganglia were frequent in depressed patients compared with control subjects. Zubenko et al.7 also noted a higher incidence of cortical infarctions and leukoencephalopathy in depressed patients, and Figiel et al.4 reported that the frequencies of large deep white-matter hyperintensities and lesions of the basal ganglia were greater in late-onset depressed patients than in patients with early-onset depressed patients of similar age. Basal-ganglia hyperintensities have also been linked to an increased likelihood of delirium induced by antidepressants or electroconvulsive treatment.5,7

This fairly extensive literature and the report by Fujikawa et al5 suggest that cerebrovascular damage may indeed be important in the pathophysiology of major depression in the elderly and worthy of further study.

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References

Response
We thank Drs Krishnan, Tüpler, and McDonald for their comments on our article.1 In our study, we observed that cerebrovascular damage plays an important role in the pathophysiology of major depression in the elderly and that risk factors for cerebrovascular disease (e.g., hypertension) are related to the onset of senile major depression. It was reported that senile major depression often persisted despite antidepressant therapy and has a poor prognosis.2,3 Figiel et al.4 reported that basal-ganglia hyperintensities are linked to an increased likelihood of delirium with antidepressants.

We suspect that major depression with silent cerebral infarction (especially mixed artery infarction with broad obstruction) persists despite administration of antidepressants and is related to refractory depression in old age. Subsequently, we suspect that major depression with mixed artery silent cerebral infarction can progress to vascular dementia. We would like to study further the response in the elderly to antidepressant therapy and the long-term prognosis for major depression with silent cerebral infarction.

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References

‘Normal’ 99mTc-HmPAO Distribution in Large Subacute Middle Cerebral Artery Infarct

The term “luxury perfusion” is used to describe situations of paradoxical cerebral blood flow (CBF) increase1 or flow values that are high in comparison with metabolic demand.2 The idea prevailed until 1993 that the 99mTc hexamethylpropylene amine oxime (Tc-HmPAO) hyperfixation observed in the subacute stage after cerebral infarct was due to luxury perfusion. However, recent observations have shown that in these circumstances, hyperfixation with 99mTc-HmPAO does not always correspond with CBF increase.3 In the following case, single-photon computed tomography (SPECT) was clearly abnormal with 123I-xe and 99mTc ethylcysteinate dimer (Tc-ECD) but paradoxically normal with 99mTc-HmPAO in the subacute stage of middle cerebral artery (MCA) infarct.

A 34-year-old man came to our hospital on May 21, 1991, with meningeal hemorrhage consequent to rupture of a left carotid artery aneurysm. He underwent surgery 3 days later without...