Letters to the Editor

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Leukoaraiosis and Dementia

To the Editor:
In recent years evidence has been accumulating that white matter abnormalities (so-called leukoaraiosis),1 detected by computed tomographic (CT) brain scan and magnetic resonance imaging (MRI), are associated with hypertension, history of stroke, aging, and dementia despite the uncertainty about its pathophysiology.2-5

The clinical spectrum encompasses asymptomatic subjects, ischemic strokes, gait disturbance, corticobulbar signs, urinary incontinence, and vascular dementia of the Binswanger type. The crucial question is why some patients with ischemic cerebrovascular events and leukoaraiosis develop dementia whereas others do not. Many authors have reported the lack of relationship between dementia and the severity of white matter lesions.6-8

We studied 21 consecutive patients (15 males and six females with a mean age of 67.5 years, range 51–80 years), referred to our institution during 1988 for stroke or TIA, who had a CT scan showing bilateral hypodensity of the deep white matter. We excluded patients with cortical infarcts or radiological criteria positive for normal pressure hydrocephalus.9 All 21 patients underwent neuropsychological assessment using a standard battery of tests exploring intelligence, attention/concentration, abstract/conceptual reasoning, language, and visuospatial skills.10

Twelve patients showed mental deterioration, while nine had only a few defective performances, mainly on attention tasks.

We selected 20 nondemented age- and sex-matched controls from patients admitted in the same period, who had a CT scan with no evidence of any cerebral lesion or leukoaraiosis.

Computed tomography studies were reviewed independently by two neuroradiologists blinded to all clinical information. Severity of white matter hypodensity was graded as 1=mild, 2=moderate, and 3=severe. In both the demented and nondemented patients with leukoaraiosis and in the control group, linear measurements of ventricular size were made according to Albert et al11 and analysis of variance was used to evaluate the different CT measurements:

<table>
<thead>
<tr>
<th>Linear Measurements (Frontal horns/intracranial width)</th>
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<tbody>
<tr>
<td>Leukoaraiosis+dementia (n=12)</td>
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<td>0.37±0.04</td>
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Analysis of Variance

- Leukoaraiosis+dementia vs. leukoaraiosis: df = 1, F = 3.61, p = 0.0727
- Leukoaraiosis+dementia vs. control: df = 1, F = 11.45, p = 0.002
- Leukoaraiosis vs. control: df = 1, F = 1.78, p = 0.19

The mean severity score of leukoaraiosis for the 12 demented patients (2.3±0.75) was not significantly different from that of the nine nondemented patients (1.6±0.72). Demented patients with leukoaraiosis showed a significant enlargement in ventricular size (p<0.002) versus the control group; nondemented patients with leukoaraiosis did not differ (p=0.19) from the control group. The demented patients with leukoaraiosis showed a trend (p<0.07) toward a greater ventricular enlargement versus the nondemented group with leukoaraiosis.

On the basis of our analysis, we suggest that ventricular dilatation may be a main or contributory factor for the development of dementia in patients with ischemic cerebrovascular disease and leukoaraiosis. Our findings are consistent with those of Hershey et al9 who found that 50% of their patients with vascular dementia had enlarged ventricles on magnetic resonance imaging, and with those of Loeb et al12 who found a more significant ventricular enlargement in patients with multi-infarct dementia than in nondemented patients with multiple infarcts.

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