White Matter Lucencies on Computed Tomography, Subacute Arteriosclerotic Encephalopathy (Binswanger’s Disease), and Blood Pressure

Barbara A. McQuinn, MD, and Daniel H. O’Leary, MD

Of 1,643 cranial computed tomography (CT) scans done in a primary–tertiary care private hospital over a 1-year period, 11 (0.67%) showed diffuse confluent white matter lucencies of < 30 Hounsfield units. By retrospective analysis, at least 4 of the 11 were demented. Of these, 3 had clinical evidence of Binswanger’s disease—characterized by progressive dementia, incontinence, variable pseudobulbar signs, and acute and subacute motor deficits. Two additional patients suffered only transient ischemic attacks or lacunar strokes; 2 had syncope; 1 had multiple sclerosis. The remaining patients were neurologically asymptomatic. In this small retrospective series, the severity of CT changes did not distinguish the patients with clinical Binswanger’s syndrome from neurologically less symptomatic patients. Ten of the eleven patients had disordered blood pressure regulation—hypertension, labile systolic pressure, orthostatic hypotension, or a combination of these factors. The severity of CT changes correlated more clearly with blood pressure instability than with clinical encephalopathy. Asymptomatic adult patients with unexplained CT white matter hypodensity and blood pressure disorders may, however, be at risk for the development of subsequent subacute arteriosclerotic encephalopathy. (Stroke 1987;18:900–905)

The neuroradiologic findings in subcortical arteriosclerotic encephalopathy (SAE or Binswanger’s disease) on computed tomography (CT) or nuclear magnetic resonance imaging (NMRI) examinations have been the subject of several recent reports. Clinically, SAE is characterized by subcortical dementia, urinary incontinence, acute and subacute motor deficits, pseudobulbar palsy, long plateau periods, and subacute progression. Hypertension is commonly associated with the disorder. Pathologically, severe arteriolar atherosclerosis and ischemic demyelination of the subcortical white matter, multiple lacunar infarcts, and enlargement of the lateral ventricles are seen. Until the advent of cranial CT scanning, postmortem examination was usually necessary to distinguish SAE from other causes of dementia, and the disorder was considered to be fairly rare. This view has been revised recently; numerous reports have described autopsy-confirmed cases of SAE in which the diagnosis was made during life on the basis of cranial CT evidence of diffuse white matter hypodensity (DWMH) in patients with the above clinical features. This CT scan picture, typical for SAE, is not uncommon—previous series have reported idiopathic DWMH in 1.7–5% of the cranial CT scans surveyed. However, the specificity of DWMH on CT examination for the actual SAE disease state remains unclear. Four to thirty-five percent of patients with DWMH in previous CT surveys were neurologically asymptomatic at the time of the examination. Many investigators assume that such individuals suffer from subclinical ischemic leukoencephalopathy. However, studies that might better characterize which patients with asymptomatic DWMH are at highest risk of later developing symptoms of Binswanger’s disease are lacking. The present study attempts to better define the medical risk factors associated with the development of DWMH, the incidence of symptomatic SAE in DWMH patients, and possible differences in risk factors or CT findings between the symptomatic and asymptomatic groups, by a limited retrospective review of patients scanned for suspected neurologic disease.

Subjects and Methods

All cranial CT examinations of adults on the inpatient ward of a private hospital over a 12-month period were screened for DWMH. Of 1,643 scans, all with bilateral diffuse (confluent throughout the white matter) subcortical hypodensities of < 30 Hounsfield units were studied regardless of diagnosis. The CT examinations were carried out using a GE 8800 unit between June 1984 and June 1985. Eight patients had noncontrast examinations, 2 had i.v. contrast, and 1 had both contrast and noncontrast studies.

The neurologic history and examination results for each patient, obtained at or around the time of the CT scan, were obtained retrospectively from the patient’s chart, as were data concerning blood pressure (BP) abnormalities and other medical illnesses such as diabetes and cardiovascular disease. All patients except Patient 9 had been examined by 1 of 3 academic consulting neurologists on the staff of our institution. The different reasons for neurologic consultation are listed in “Results.” Patient 9’s neurologic findings were obtained from recorded examinations by the internal
### Table 1. Clinical Characteristics of Patients With Diffuse White Matter Hypodensity

<table>
<thead>
<tr>
<th>Patient Age/Sex</th>
<th>Cardiovascular or systemic illness</th>
<th>Hypertension</th>
<th>Orthostatic hypotension</th>
<th>Diabetes</th>
<th>Subacute neurologic events</th>
<th>Acute cerebrovascular events</th>
<th>Incontinence</th>
<th>Dementia</th>
</tr>
</thead>
<tbody>
<tr>
<td>1/77/F</td>
<td>CHF and AS/AI valvular disease</td>
<td>Yes, difficult to control; wide fluctuations in BP</td>
<td>Yes, frequently symptomatic; believed related to multiple hypertensive medications</td>
<td>No</td>
<td>No</td>
<td>Yes; frontal ICH; multiple bilateral lacunar infarcts</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>2/64/M</td>
<td>Renal failure; constrictive pericarditis; CAD</td>
<td>Yes, difficult to control; wide fluctuations in BP</td>
<td>Not mentioned</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>3/52/F</td>
<td>L focal seizure, unknown etiology</td>
<td>Yes</td>
<td>Not mentioned</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>4/71/M</td>
<td>No</td>
<td>Yes</td>
<td>Not mentioned</td>
<td>No</td>
<td>Intermittent confusion over 2 yrs</td>
<td>R parieto-occipital cortical infarct</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>5/89/M</td>
<td>Abrupt onset dementia 3 wks; S/P lumbar sympathectomy for PVD</td>
<td>No</td>
<td>Yes, systolic BP change of 33 mm Hg, lying to sitting; fluctuations in systolic BP of 70 mm Hg during typical day</td>
<td>No</td>
<td>Pseudobulbar affect; jaw jerk</td>
<td>No</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>6/72/F</td>
<td>Angina; CAD</td>
<td>Yes</td>
<td>Yes (diabetic autonomic neuropathy)</td>
<td>Yes</td>
<td>Gait deterioration</td>
<td>CVA, minor deficits x 2; 1 lacunar infarct</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>7/78/M</td>
<td>Atrial fibrillation</td>
<td>Yes</td>
<td>Not mentioned</td>
<td>Yes</td>
<td>No</td>
<td>Yes; TIA x 2</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>8/79/F</td>
<td>R subdural hematoma evacuated 6 mos previously; depression; impulsive character disorder x yrs</td>
<td>No</td>
<td>Not mentioned</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>9/92/F</td>
<td>Syncope x 2; CHF; atrial fibrillation</td>
<td>Wide daily variation in systolic BP (&gt;100 mm Hg/day) on no medications</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>10/73/F</td>
<td>Abnormal visual, brainstem evoked potentials; presumed MS</td>
<td>Yes</td>
<td>Not mentioned</td>
<td>No</td>
<td>Partial Brown-Sequard syndrome</td>
<td>No</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>11/64/F</td>
<td>Heavy EtOH abuse; peripheral neuropathy</td>
<td>No</td>
<td>Not mentioned</td>
<td>No</td>
<td>Paresthesias; leg weakness</td>
<td>No</td>
<td>Yes; isolated impairment of memory; confabulation</td>
<td>No</td>
</tr>
</tbody>
</table>

CHF, congestive heart failure; AS/AI, aortic stenosis/aortic insufficiency; BP, blood pressure; CAD, coronary artery disease; ICH, intracerebral hemorrhage; L, left; R, right; PVD, peripheral vascular disease; CVA, cerebrovascular accident; TIA, transient ischemic attack; MS, multiple sclerosis.
medicine academic staff. Special note was made of the results of the bedside mental status examination, which routinely included tests of recall, orientation, language, and general information. However, the retrospective nature of the study precluded standardization of mental status examination techniques used by the 3 neurologists.

Patients were considered hypertensive if they regularly sustained BP > 140/90 mm Hg or if they were receiving antihypertensive medications because that diagnosis had been previously established. The daily vital signs record of all patients was examined for the presence of wide fluctuations in systolic BP. Patients were considered to have orthostatic hypotension if it had been noted that their systolic BP fell by > 25 mm Hg when changing between lying, sitting, or standing positions. Patients were considered diabetic when medications were required to control blood glucose. Both the nursing notes and the recorded general and neurologic evaluations were screened for the mention of urinary or fecal incontinence.

DWMH was identified in 11 of 1,643 consecutive CT scans (incidence, 0.67%). None of the involved areas enhanced with i.v. contrast. There were 7 women and 4 men in the group (ages 52–92, mean 74); 8 of these 11 patients had no history of antecedent neurologic disorders known to cause DWMH. The remaining 3 patients had histories of disorders that might contribute to CT leukoencephalopathy: Patient 1 had survived a left frontal lobe intracerebral hemorrhage (ICH) 1 year prior to her examination, Patient 8 had had a craniotomy for subdural hematoma evacuation 6 months previously, and Patient 10 (see Table 1) was presumed to have multiple sclerosis (MS) on the basis of clinical and evoked potential findings. Of note is that Patient 1 did not show DWMH on CT scan at the time of her ICH, and her more recent scans showed equivalent degrees of DWMH in each hemisphere (Figure 1). Reasons for neurologic evaluation of the 11 patients included syncope in 2, memory loss and confusion in 4, recent transient ischemic attack (TIA) in 1, temporary acute confusional state and renal failure in 1, first seizure in 1, late-life MS in 1, and aggressive personality disorder in 1.

Only 4 of the 11 patients were considered demented (incidence, 36%). Admittedly, this figure may be falsely low as a result of insensitivity of the bedside mental status examination for mild degrees of dementia; also, the retrospective nature of the study precluded use of a standardized testing regime. One patient (Patient 11) had a Korsakoff's-type dementia and peripheral neuropathy, presumably related to chronic
alcohol abuse, and she did not share the risk factors of vasculopathy or hypertension with the rest of the group. Her CT scan is shown in Figure 2. Because this patient exhibited no hemispheric deficits, incontinence, pseudobulbar palsy, or other nonalcohol-related neurologic signs, the classification of her dementia as a manifestation of SAE rather than of alcoholic Korsakoff's syndrome is uncertain. The other 3 demented patients fulfilled most of the clinical criteria for SAE (see Table 1) and were considered to suffer from that syndrome. Mental status examinations of all 3 patients recorded marked deterioration of judgment, memory, and abstract reasoning with near-normal language skills. The remaining 7 patients in our study had normal recorded examination of mental status, and no mention was made of incontinence or subacute motor deterioration. Two of these 7 patients (Patients 1 and 7) had experienced acute cerebral ischemic events (strokes and TIAs), but in isolation these were not considered specific for clinical SAE. Thus, only 3 patients of the 11 (27%) definitely had the full clinical syndrome of symptomatic SAE, or Binswanger's disease.

Ten of the 11 patients (91%) had disordered BP regulation, due to either hypertension, widely fluctuating systolic BP in otherwise normotensive patients, orthostatic hypotension, or a combination of these factors. Six of the 11 had prior evidence of cerebral or systemic atherosclerosis. Of the 8 apparently neurologically asymptomatic patients, 7 (88%) had hypertension, 2 diabetes, 4 ischemic cardiac disease, 2 prior acute cerebrovascular events, and 1 renovascular disease. Three of the 8 (38%) routinely exhibited daily fluctuations in systolic BP of > 70 mm Hg; 1 had symptomatic orthostatic hypotension. Of the 3 patients with clinical Binswanger's disease, 2 were hypertensive, 1 had diabetes, 1 had cardiovascular disease, and 2 had prior acute cerebrovascular events. Two of these 3 had moderately symptomatic orthostatic hypotension, with severe lightheadedness precipitated by standing.

The incidence of hypertension within the entire group of 11 patients was 82% (9 of 11); of extreme systolic BP swings, 45% (5 of 11); of diabetes, 27% (3 of 11); of cardiac disease, 45% (5 of 11); of TIA or stroke, 36% (4 of 11); and of renal and peripheral vascular diseases, 9% (1 of 11). There were no significant differences in risk factor incidence or severity between the 3 patients with definite signs of SAE dementia and the other 8 patients.

Discussion
Only a minority of patients with DWMH in our series (27%) had strong clinical evidence suggesting
SAE. The percent of such patients without clinical findings has been sizable in other reports as well, ranging from 4 to 35%.1,2,13 DWMH in the present study was much less specific for clinicalBinswanger’s disease than for the presence of either systemic vasculopathy or disordered BP regulation (Figures 3 and 4).

Four of the 5 patients with the most radiologically extensive DWMH also suffered the most labile systolic BP. These patients also had significant systemic atherosclerosis, and the apparent association between DWMH and BP swings could therefore be artifactual. An alternative speculation is that these 2 factors might constitute additive risks for the development of DWMH.

Patient 5’s case is significant in that the association of labile BP with DWMH is apparently independent of hypertension. Though never hypertensive, Patient 5 developed new-onset dementia, incontinence, and a gait disorder approximately 4 weeks after a lumbar sympathectomy, which had resulted in severe chronic symptomatic orthostatic hypotension (Figure 5).

In a large autopsy series of elderly persons, neuropathologic findings of SAE were seen in 3.8%, regardless of neurologic symptoms.4 However, the degree of leukoencephalopathy in that study correlated with the degree of neurologic impairment, as it did in another recent CT survey.1 We were unable to find a similar correlation between the extent of radiographic DWMH and the severity of recorded clinical SAE. The prevalence of symptomatic Binswanger’s dementia may have been underestimated in our sample due to the limitations of retrospective analysis of mental status testing. On the other hand, our sample population is biased toward the neurologically impaired in that patients were scanned for suspected neurologic illness.

On CT scan, DWMH was almost always associated with disordered BP regulation, systemic atherosclerosis, or both. In individuals with the above risk factors, it is reasonable to consider that DWMH may reflect the neuropathologic changes of SAE within the subcortical white matter. The significance of similar white matter changes on NMRI is not addressed by the current study, but an analysis of BP abnormalities in patients with abnormal white matter NMRI signals would also be of interest. Careful clinical and neuropathologic follow-up of “asymptomatic” DWMH patients identified by either technique, especially those with generalized atherosclerosis and unstable BP, may shed further light on the true spectrum of clinical SAE and associated risk factors.

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