Capsular Hypesthetic Ataxic Hemiparesis

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Twenty-three patients with hypesthetic ataxic hemiparesis underwent computed tomography or magnetic resonance imaging. Twenty-two patients had infarcts of lacunar or slightly larger size in the contralateral posterior limb of the internal capsule. In 15 patients the infarct extended superiorly into the adjacent paraventricular region, and in seven it extended into the lateral thalamus. In eight patients the infarct was limited to the posterior limb of the internal capsule, and in only two patients was an ipsilateral to capsular pontine lacune found. Despite a location similar to that of pure motor and pure sensory lacunar stroke, hypesthetic ataxic hemiparesis correlates with larger infarcts, most often located in the posterior medial superior territory of the anterior choroidal artery. Some infarcts appeared to be localized immediately posterolateral to this region, in the posterior cerebral artery territory. The presence and extent of infarction is better detected by the addition of magnetic resonance imaging to computed tomography. (Stroke 1990;21:24-33)

Recent discussion has questioned the existence of the clinical syndrome of ataxic hemiparesis. Initially attributed to infarction localized to the posterior limb of the internal capsule and verified at autopsy by Fisher and Cole, later necropsy-based findings of Fisher and computed tomographic (CT) review by Huang and Lui established that accompanying hemihypesthesia indicated a capsular rather than a pontine location for lesions causing the ataxic hemiparesis symptom complex. Larsen et al recently confirmed the capsular identity of ataxic hemiparesis using magnetic resonance imaging (MRI).

Foix and Hillemand designated the anterior choroidal artery (AChA) "the artery of the posterior limb of the internal capsule." Bogousslavsky et al described a patient with hemiataxia and ipsilateral hemisensory deficit without pyramidal tract signs and attributed these findings to AChA-territory stroke. The posterior cerebral artery (PCA) thalamogeniculate branch may also irrigate the posterior limb of the internal capsule in its posterior portion. Neither the older literature nor other recent reports typically attribute ataxic hemiparesis to AChA-territory infarction. We describe 23 cases of hypesthetic ataxic hemiparesis with CT- or MRI-documented infarct in the posterior limb of the internal capsule. Special attention is drawn to the pathogenesis and clinical-anatomic correlation of infarction in these cases.

Subjects and Methods

Twenty-three patients with acute stroke and new ataxic hemiparesis with hemisensory deficit had cranial CT scans. Eleven patients had CT both with and without contrast; the other 12 had CT without enhancement. Twelve patients had cranial MRI with a superconducting 1.5-T unit in addition to CT. The lesions appeared as localized areas of abnormal low attenuation on unenhanced CT scans. Some lesions showed partial contrast enhancement. The lesions appeared as areas of increased signal intensity on both spin-density first echo (resonance time [TR] 2000, echo time [TE] 20 msec) and T2-weighted second echo (TR 2000, TE 80 msec) MRI images. No patient died, and only three had cerebral angiography. All patients were seen by the Stroke Service at the University of Illinois and Cook County hospitals between July 1985 and February 1989.

Pyramidal tract weakness was defined as present when face, arm, and leg weakness was accompanied by ipsilateral hyperreflexia or a Babinski's sign. Ataxia was defined by intention tremor of the finger, toe, or heel when movement toward a target was carried out with a to-and-fro deviation of the limb from the ideal course, the deviation increasing as the target was approached. Sensory deficit occurred ipsilateral to the ataxia and motor weakness and consisted of decreased touch, pinprick, temperature, vibration, or proprioceptive sensation compared with the intact hemisemisensory deficit.

Results

Lesions that involved the posterior two thirds of the posterior limb of the internal capsule and extended...
FIGURE 1. Magnetic resonance imaging of infarction localized to posterior two thirds of posterior limb of internal capsule, with superior extension into paraventricular region.
superiorly to the level of the body of the lateral ventricle constituted the most common pattern, present in nine patients (8–14, 16, and 18) (Figure 1, Table 1). Eight patients (5–7, 15, 17, 19, 21, and 23) had abnormalities confined to the posterior two thirds of the posterior limb of the internal capsule without visible superior extension. In six patients (1–4, 20, and 22) a distinctive pattern of infarction involving the posterior putamen and the adjacent posterior third of the posterior limb of the internal capsule with superior extension through the upper portion of the posterior internal capsule to the level of the body of the lateral ventricle was demonstrated (Figure 2). Seven patients (1, 3, 7, 9, 13, 19, and 21) had lateral thalamic infarcts adjacent to and involving the posterior limb of the internal capsule (Figure 3); two of these seven patients (9 and 13) had extension superiorly into the paraventricular region. One patient (15) had bilateral, diffuse ischemic changes in both basal ganglia (including the posterior limb of the internal capsule), the lentiform nuclei, and the deep periventricular white matter superior to these structures. Patient 12 had three separate foci of infarction within the posterior limb of the internal capsule: one low in the posterior third, one in the middle third, and another in the deep periventricular region just above the posterior limb.

As a group, the lesions involved the posterior third of the posterior limb of the internal capsule in all 23 patients and showed superior involvement of the upper part of the posterior limb extending paraventricularly in all but eight patients. Nearly a third of the patients also had involvement of the lateral thalamus. In only two patients in whom MRI documented a capsular infarct (15 and 20) were changes noted ipsilaterally in the pons (Figure 4). Patient 4 had an arachnoid cyst of the posterior fossa contralateral to the capsular infarct. Patient 3 had an ipsilateral but noncontiguous PCA infarct. Infarct volume varied from 0.5 to 5.0 cm³ (Table 1) and was thus not limited to lacunar size. MRI detected infarction when CT was negative in four patients, and CT detected infarction when MRI was negative in two (Table 1). The clinical presentations of these 23 patients are given in Table 2. Their ecologic profiles are given in Table 3.
FIGURE 2. Magnetic resonance imaging of infarction in posterior putamen and adjacent posterior one third of posterior limb of internal capsule with superior extension.

FIGURE 3. Magnetic resonance imaging of infarction in lateral thalamus and adjacent posterior limb of internal capsule.
Discussion

Landau recently questioned the very existence of the clinical entity of ataxic hemiparesis and stated that all patients who are weak are clumsy. This is refuted by the observations of Fisher and others that as the patient with ataxic hemiparesis recovers from the acute phase, a disassociation of ataxia and weakness can occur, the latter often fully resolving while the former remains. Reports of accompanying signs of nystagmus and Babinski's reflex in a patient considered to have ataxic hemiparesis indirectly support the coexistence of cerebellar ataxia with pyramidal-type weakness. Often the ataxia appears out of proportion to the weakness. If, as Landau suggests, all patients who are weak are clumsy and ataxic, then one must deny the existence of pure motor hemiparesis. None of our patients had pure motor hemiparesis.

The syndrome of hyposthetic ataxic hemiparesis comprises sensory and motor elements. Bogousslavsky et al suggested that cerebellar ataxia associated with infarction in the posterior limb of the internal capsule is due to a crossed cerebellar diaschisis. Others note that the oral and caudal parts of the ventral medial thalamus receive afferents from the dentatorubrothalamic pathway. Kelly et al and Mori et al found delayed somatosensory evoked responses in six cases of ataxic hemiparesis due to stroke localized to the posterior limb of the internal capsule. Together, these findings suggest that the ataxia of hyposthetic ataxic hemiparesis is caused by dysfunction of the ascending cerebellothalamic, rather than of the descending corticopontocerebellar fibers traversing the posterior limb of the internal capsule.

Afferent lemniscal pathways subserving proprioceptive, kinesthetic, and spinocerebellar tract information may synapse in the ventroposterolateral thalamic nucleus and ascend in a thalamocortical projection through the posterior limb of the internal capsules. Fisher and Curry concluded that the sensory fibers are not arranged in a compact fascicle. The fibers may traverse the thalamocapsular paraventricular region affected in our series. Disruption of these relay pathways could cause a sensory ataxia and might explain the sensory loss in some of our patients. Of note, however, is the infrequency of loss of proprioception among our patients (Table 2).

Stroke in the posterior limb of the internal capsule can cause clinical syndromes of a pure sensory or a pure motor (pyramidal tract) nature. Pure motor or pure sensory stroke has been explained by a small lacune in the posterior limb of the internal capsule located anterolaterally (with or without medial globus pallidus involvement) or medioposteriorly, respectively, causing motor or sensory fiber tract interruption. Lacunes in the posterior limb of the internal capsule in patients with type II pure motor loss reported by Rascol et al never extended upward into the paraventricular region, were always small, and in some instances involved the medial globus pallidus. Crural predominance for motor weakness was frequent in our patients and possibly reflects the predominantly posterior capsular location of the lacunes and somatotopic capsular organization. Groothuis et al ascribed a pure sensory syndrome to lacunar infarction of the medial border of the posterior capsular limb, and Fisher localized pure sensory stroke to the ventroposterolateral thalamus. The majority of strokes in our study of hyposthetic ataxic hemiparesis localized medioposteriorly in the posterior limb of the internal capsule but also extended superiorly paraventricularly and were larger than lacunar in size. Patient 19 had an MRI-detected infarct in a location similar to that in the cases presented by Groothuis et al and Fisher (Figure 3). This patient had evanescent upper motor neuron signs with predominant hemisensory and hemiataxic deficits similar to the clinical presentation of the patient of Bogousslavsky et al.

Ataxic hemiparesis has been attributed to pontine infarction. MRI detected an appropriate pontine infarct ipsilateral to the capsular infarct in two of our patients (Figure 4). Either lesion could explain the contralateral signs of ataxic hemiparesis. Jacob et al suggested that the presence of ipsilateral sensory deficit as well as the absence of nystagmus and dysarthria in patients with ataxic hemiparesis argues for a capsular pathology. All of our patients had hemisensory deficits. Although eight of our 23 patients had dysarthria (Table 2), it was mild when present. Nystagmus was found in Patient 4, in whom a contralateral arachnoid cyst of the posterior fossa was found. As only 12 of our patients had MRI, which is more sensitive than CT for detecting posterior fossa...
TABLE 2. Clinical Presentations of 23 Patients With Hypesthetic Ataxic Hemiparesis

<table>
<thead>
<tr>
<th>Pt</th>
<th>Onset</th>
<th>Motor</th>
<th>Ataxia</th>
<th>Dysarthria</th>
<th>Vertigo</th>
<th>Nystagmus</th>
<th>Sensory</th>
<th>Ambulation</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Stuttering</td>
<td>R: leg &gt; arm &gt; face; symmetric deep tendon reflexes; Babinski's sign</td>
<td>R: arm = leg; gait</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>R: ↓ pinprick, touch: face = arm = leg</td>
<td>+</td>
</tr>
<tr>
<td>2</td>
<td>Stuttering</td>
<td>L: leg; ↑ deep tendon reflexes; Babinski's sign</td>
<td>L: leg</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>L: ↓ pinprick: face = arm = leg</td>
<td>+</td>
</tr>
<tr>
<td>3</td>
<td>Sudden</td>
<td>L: face = leg; symmetric deep tendon reflexes; Babinski's sign</td>
<td>L: leg</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>L: ↓ visual field; ↓ pinprick, touch: face = arm = leg</td>
<td>+</td>
</tr>
<tr>
<td>4</td>
<td>Sudden</td>
<td>R: leg &gt; arm = face; ↑ deep tendon reflexes; Babinski's sign</td>
<td>R: arm = leg; gait</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>R: ↓ corneal reflex; ↓ pinprick, vibration, touch: leg</td>
<td>+</td>
</tr>
<tr>
<td>5</td>
<td>Stuttering</td>
<td>L: arm = leg &gt; face; symmetric deep tendon reflexes; no Babinski's sign</td>
<td>L: arm = leg; gait; ( \downarrow ) OKN to R</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>L: ↓ corneal reflex; ↓ pinprick, touch, vibration: face = arm = leg</td>
<td>-</td>
</tr>
<tr>
<td>6</td>
<td>Sudden</td>
<td>L: leg &gt; arm &gt; face; symmetric deep tendon reflexes; Babinski's sign</td>
<td>L: arm &gt; leg</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>L: ↓ corneal reflex; ↓ pinprick: face = arm = leg</td>
<td>+</td>
</tr>
<tr>
<td>7</td>
<td>Stuttering</td>
<td>L: face = arm = leg; ↑ deep tendon reflexes; Babinski's sign</td>
<td>L: arm = leg</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>L: ↓ corneal reflex; ↓ pinprick, touch, temperature, vibration: arm = leg</td>
<td>+</td>
</tr>
<tr>
<td>8</td>
<td>Sudden</td>
<td>R: leg &gt; arm = face; symmetric deep tendon reflexes; Babinski's sign</td>
<td>R: arm = leg; gait</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>R: ↓ pinprick: arm = leg</td>
<td>+</td>
</tr>
<tr>
<td>9</td>
<td>Unknown</td>
<td>L: leg &gt; arm &gt; face; symmetric deep tendon reflexes; Babinski's sign</td>
<td>L: leg &gt; arm; gait</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>L: ↓ pinprick: face = arm = leg</td>
<td>-</td>
</tr>
<tr>
<td>10</td>
<td>Stuttering</td>
<td>R: leg &gt; face, arm; ↑ deep tendon reflexes; Babinski's sign</td>
<td>R: arm = leg; gait</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>R: ↓ pinprick: face = arm = leg</td>
<td>+</td>
</tr>
<tr>
<td>11</td>
<td>Sudden</td>
<td>R: arm = leg &gt; face; symmetric deep tendon reflexes; Babinski's sign</td>
<td>R: arm = leg</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>R: ↓ pinprick: arm = leg</td>
<td>+</td>
</tr>
<tr>
<td>12</td>
<td>Stuttering</td>
<td>L: leg &gt; arm &gt; face; ↑ deep tendon reflexes; Babinski's sign</td>
<td>L: arm = leg; gait</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>L: ↓ corneal reflex; ↓ pinprick, touch, temperature: face = arm = leg</td>
<td>-</td>
</tr>
<tr>
<td>13</td>
<td>Sudden</td>
<td>R: arm = face = leg; ↑ deep tendon reflexes; Babinski's sign</td>
<td>R: arm = leg; gait</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>R: ↓ visual field, ↓ corneal reflex; ↓ pinprick, touch, temperature: face = arm = leg; ↓ position: arm = leg</td>
<td>+</td>
</tr>
<tr>
<td>14</td>
<td>Sudden</td>
<td>L: leg = arm &gt; face; symmetric deep tendon reflexes; no Babinski's sign</td>
<td>L: arm</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>L: ↓ position: arm = leg; ↓ pinprick, touch, temperature: face = arms = leg; ↓ point location: leg</td>
<td>+</td>
</tr>
<tr>
<td>15</td>
<td>Sudden</td>
<td>L: leg = face = arm; symmetric deep tendon reflexes; no Babinski's sign</td>
<td>L: arm = leg</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>L: ↓ touch: face = arm = leg</td>
<td>-</td>
</tr>
<tr>
<td>16</td>
<td>Sudden</td>
<td>R: leg = arm &gt; face; ↑ deep tendon reflexes; Babinski's sign</td>
<td>R: arm = leg; gait</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>R: ↓ pinprick: face = arm = leg</td>
<td>+</td>
</tr>
<tr>
<td>17</td>
<td>Sudden</td>
<td>L: leg = arm &gt; face; ↑ deep tendon reflexes; Babinski's sign</td>
<td>L: leg = arm</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>L: ↓ pinprick: leg</td>
<td>+</td>
</tr>
</tbody>
</table>

Previous reports have inconsistently correlated neuroimaged, autopsy-proven, and clinically inferred abnormalities localized to the thalamus, internal capsule, and corona radiata with the hypesthetic ataxic hemiparesis symptom complex.2-4,7,19,20,35-38

infarcts, no definite statement can be made about possible concomitant pontine infarcts in the other 11 patients. None of these 11 patients, however, had pontine strokes by CT or definite clinical signs of posterior fossa disease.
Mohr et al\textsuperscript{39} attributed a sensory motor deficit to infarction in the posterolateral thalamocapsular region. Discrepant accounts of thalamic involvement in reports of hyposthetic ataxic hemiparesis may be due to the insensitivity of CT for depicting infarction in gray matter nuclei such as the thalamus, as well as to the fact that various anatomic sites of destruction may lead to similar neurologic deficits.\textsuperscript{19,35,40} Involvement of the thalamus was detected only by MRI in three of seven patients in our series. An answer to whether there is thalamic ischemia in each instance of hyposthetic ataxic hemiparesis will require MRI studies as well as pathologic verification. Such correlation thus far is absent in the varied reports of infarction correlated with this syndrome. The majority of patients with hyposthetic ataxic hemiparesis in our series had infarcts localized by CT or MRI to the posterior third of the posterior limb of the internal capsule and contiguous paraventricular region. This distribution is similar to that found by Fisher and Cole,\textsuperscript{2} by Huang and Lui,\textsuperscript{4} and by Perman and Racy,\textsuperscript{36} whose patients with ataxic hemiparesis also had sensory deficits. Verma et al\textsuperscript{35} and de Renzi and Nichelli\textsuperscript{19} localized infarcts in a similar clinical syndrome to a more anterior and rostral portion of the posterior limb of the internal capsule. Others have correlated ataxic hemiparesis without sensory deficit to infarction of the posterior limb of the internal capsule and adjacent corona radiata as seen by CT.\textsuperscript{34,41-44} Using MRI, Larsen et al\textsuperscript{3} localized infarcts in the ataxic hemiparesis syndrome to the posterior limb of the internal capsule. Their case demonstrated no superior extension or thalamic involvement. The lesion responsible for hyposthetic ataxic hemiparesis may lie at a variable superior–inferior extent in the posterior limb of the internal capsule and may affect the lateral thalamus more frequently than is depicted by CT. As only slightly more than half of our patients underwent MRI, it may be that the number of patients with thalamic and superior extension into the paraventricular region is greater than indicated by our findings.

The exact vascular supply of the area infarcted in our patients with hyposthetic ataxic hemiparesis is uncertain. The AChA and its penetrating branches may supply the lateral portions of the thalamus, the lateral geniculate, and the superficial superior part of the ventrolateral thalamic nuclei, as well as the posterior limb of the internal capsule in its posterior two thirds, sometimes as high as the superior angle of the lateral segment of the globus pallidus, and the retrolenticular portions, the tail of the caudate nucleus, the medial globus pallidus, and the posterior putamen.\textsuperscript{17,45-50} Damasio's\textsuperscript{51,52} CT template and Beevor's\textsuperscript{53} anatomic localization of the AChA territory include what appears to be a superior extension into the paraventricular region. This appears at first glance to contrast with most reports of the area supplied by the AChA. It may be that when viewed at 20\textdegree{} cuts parallel to the canthomeatal (orbitomeatal) line (as is customary with CT), the caudatal, capsular, and superior lateral thalamic areas supplied by the AChA appear paraventricular. In addition, some would view this paraventricular area as being the corona radiata and thus supplied by the lateral striate arteries of the middle cerebral artery (MCA). However, it is not possible to exclude edema as the cause of MRI or CT findings at this site. It will be noted that by pathologic–anatomic studies, Kaplan and Ford\textsuperscript{46} deny the extension of lateral striate MCA branches into the corona radiata. Partial or complete infarction of the AChA territory may occur and depends on the caliber of the affected vessel (AChA stem, branch, or collateral), the extent of its ramification zone, and available collaterals from the MCA, posterior communicating artery (PCoA), or PCA.\textsuperscript{45,54} Our Patient 12 had three separate infarcts, each of which corresponded to occlusion of an AChA pene-
TABLE 3. Ecologic Profile of 23 Patients With Capsular Hypesthetic Ataxic Hemiparesis

<table>
<thead>
<tr>
<th>Pt/age/sex</th>
<th>Migraine</th>
<th>Cocaine</th>
<th>Diabetes mellitus</th>
<th>Hypertension</th>
<th>Carotid disease</th>
<th>Lues, other</th>
<th>Carotid angiography</th>
<th>Duplex ultrasonography</th>
<th>Smoking</th>
<th>Alcohol use</th>
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<tbody>
<tr>
<td>1/61/F</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>Angina pectoris</td>
<td>-</td>
<td>Normal</td>
<td>Not done</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>2/81/M</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>LVH</td>
<td>-</td>
<td>Bifurcation plaque</td>
<td>Not done</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>3/68/F</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>LVH, LAE, old MI</td>
<td>-</td>
<td>Not done</td>
<td>Not done</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>4/59/F</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>Normal</td>
<td>Not done</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>5/62/M</td>
<td>-</td>
<td>+/-</td>
<td>+</td>
<td>+</td>
<td>CAC</td>
<td>Chol, TG</td>
<td>Bifurcation plaque</td>
<td>Not done</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>6/56/M</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>LVH, old MI</td>
<td>Chol, TG</td>
<td>Bifurcation plaque</td>
<td>Not done</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>7/54/F</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>CHF, AS</td>
<td>-</td>
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<td>Not done</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>8/56/F</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>Not done</td>
<td>Bifurcation plaque</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>9/68/F</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>Paraproteinemia</td>
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<td>Not done</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>10/63/M</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>Not done</td>
<td>Not done</td>
<td>Unknown</td>
<td>Unknown</td>
</tr>
<tr>
<td>11/74/M</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>Normal</td>
<td>Not done</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>12/60/M</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>CAC, old MI</td>
<td>-</td>
<td>Bifurcation plaque</td>
<td>Not done</td>
<td>+</td>
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<td>+</td>
<td>+</td>
<td>-</td>
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<td>Normal</td>
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<td>Not done</td>
<td>Normal</td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td>14/58/M</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>Angina pectoris, old MI, LVT</td>
<td>-</td>
<td>Normal</td>
<td>Not done</td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td>15/79/F</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>LVH, old MI, MAC</td>
<td>-</td>
<td>Normal</td>
<td>Not done</td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td>16/45/F</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>LVH, LAE</td>
<td>-</td>
<td>Not done</td>
<td>Normal</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>17/75/M</td>
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<td>-</td>
<td>-</td>
<td>-</td>
<td>LVH</td>
<td>-</td>
<td>Bifurcation plaque</td>
<td>Not done</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>18/46/M</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>LVH</td>
<td>-</td>
<td>Normal</td>
<td>Not done</td>
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<td>20/51/M</td>
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<td>21/26/M</td>
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<td>22/42/M</td>
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Pt, patient; F, female; M, male; LVH, left ventricular hypertrophy; LAE, left atrial enlargement; MI, myocardial infarction; CHF, congestive heart failure; AS, aortic stenosis; CAC, coronary artery calcification; LVT, left ventricular thrombus; MAC, mitral annulus calcification; Chol, cholesterol; TG, triglycerides.

Helgason and Wilbur

Capsular Hypesthetic Ataxic Hemiparesis

Trator. Alternatively, the PCA thalamogeniculate branch may irrigate the posterior portion of the lateral thalamic nuclear mass, the posterior portion of the internal capsule, and the putamen.8-51,53,55 Our Patients 1-4, 20, and 22 had infarcts localized by CT or MRI to this anatomic area. The MCA lenticulostriate branches irrigate the posterior limb of the internal capsule anteriorly, the striate ganglia, and may extend contiguously into the adjacent corona radiata.51-53 No infarct in any patient in our series corresponded to this anterior lateral location.

Two recent reports of the clinical syndrome associated with thalamocapsular infarction, one attributed to AChA-territory infarction, mention hypesthetic ataxic hemiparesis.55-59 Bruno et al60 did not see hypesthetic ataxic hemiparesis in their series. Ataxia as a component of the AChA syndrome is first mentioned by Bogousslavsky et al.7 However, previous autopsy- and CT-based accounts of capsular ataxic hemiparesis with sensory loss do correspond to the anatomy of the AChA territory described by Beevor,53 Damasio,51,52 and others.2,4,19,20,36-38,51-55 Thus, hypesthetic ataxic hemiparesis may be a more common manifestation of occlusion of AChA mainstem or penetrator branches than has been recognized. None of the patients reported in this article were included in our previous reports of AChA-territory infarction.10,12,13 Hypesthetic ataxic hemiparesis was also attributed to lateral lenticular striate (MCA)-territory infarction by Ghika et al.56 Thalamogeniculate branch (PCA) occlusion has been associated with contralateral ataxia, motor weakness, and sensory loss. Usually, other symptoms are also present.61

Angiographic and necropsy data confirming the vascular supply of the infarcted tissue in this series of patients with hypesthetic ataxic hemiparesis do not
exist. Angiography would be of little help due to its limitations in detecting AChA or PCA penetrator branch occlusion. As it is not always identified by internal carotid artery angiography, the absence of the AChA on angiograms would not prove its occlusion.62–64

The cases in our series followed the general rule that small-vessel disease, typical of hypertension, diabetes, and lues, is likely responsible for infarction in the AChA territory (Table 3). Four patients had potentially vasospastic etiologies of infarction (history of migraine and cocaine use; Table 3). Patient 3 had an ipsilateral PCA-territory infarct. As the PCA forms collaterals with the AChA, it is impossible to rule out large-vessel disease in Patient 3. Recovery from hypesthetic ataxic hemiparesis was good. Only two patients manifested moderate or severe neurologic dysfunction at 3 months’ follow-up. This is in contrast to the prognosis reported for the AChA syndrome in general10–13 and may reflect the unilateral and more posterior medial and superior location of infarction in patients with hypesthetic ataxic hemiparesis.

Although lacking necropsy or angiographic proof of the site of vascular occlusion, our study may reflect a previously unemphasized clinical symptom complex associated with infarction in the AChA territory. On the whole, these capsular-territory infarcts associated with hypesthetic ataxic hemiparesis are of lacunar or small-vessel disease, typical of hypertension, diabetes, and lues, and are difficult to distinguish radiologically from other cases of typical AChA-territory stroke. As a larger experience with small-vessel disease, typical of hypertension, diabetes, and lues, is likely responsible for infarction in the AChA territory (Table 3). Four patients had potentially vasospastic etiologies of infarction (history of migraine and cocaine use; Table 3). Patient 3 had an ipsilateral PCA-territory infarct. As the PCA forms collaterals with the AChA, it is impossible to rule out large-vessel disease in Patient 3. Recovery from hypesthetic ataxic hemiparesis was good. Only two patients manifested moderate or severe neurologic dysfunction at 3 months’ follow-up. This is in contrast to the prognosis reported for the AChA syndrome in general10–13 and may reflect the unilateral and more posterior medial and superior location of infarction in patients with hypesthetic ataxic hemiparesis.

Although lacking necropsy or angiographic proof of the site of vascular occlusion, our study may reflect a previously unemphasized clinical symptom complex associated with infarction in the AChA territory. On the whole, these capsular-territory infarcts associated with hypesthetic ataxic hemiparesis are of lacunar or moderately larger size and frequently involve the lateral thalamus and posterior medial portions of the posterior limb of the internal capsule, with superior extension into the paraventricular region. The medial globus pallidus is not involved. The subgroup of hypesthetic ataxic hemiparesis–associated infarcts localized to the posterior putamen and posterior third of the posterior limb of the internal capsule may represent PCA branch–territory stroke. Although they may differ in size and location from those infarcts leading to pure motor or pure sensory loss, the infarcts in our patients are difficult to distinguish radiologically from other cases of typical AChA-territory stroke. As a larger experience with the hypesthetic ataxic hemiparesis stroke syndrome accrues, a full spectrum of clinical manifestations should come into view. Better imaging techniques65 may further define the site(s) of infarction and the pathogenesis of neurologic dysfunction.

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