Involuntary Movements After Anterior Cerebral Artery Territory Infarction

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Background and Purpose—Patients with anterior cerebral artery territory infarction presenting with involuntary movements have rarely been described in the literature.

Case Descriptions—The author reports 9 such patients: 3 with asterixis, 5 with hemiparkinsonism (tremor, rigidity, hypokinesia), and 1 with both. Asterixis developed in the acute stage in patients with minimal arm weakness, whereas parkinsonism was usually observed after the motor dysfunction improved in patients with initially severe limb weakness. Asterixis correlated with small lesions preferentially involving the prefrontal area; parkinsonism is related to relatively large lesions involving the supplementary motor area.

Conclusions—Anterior cerebral artery territory infarction should be included in the differential diagnosis of asterixis and hemiparkinsonism. (Stroke. 2001;32:258-261.)

Key Words: cerebral arteries ▪ cerebrovascular disorders ▪ dyskinesias ▪ movements ▪ Parkinson disease ▪ tremor

Involuntary movements (IMs), such as chorea, dystonia, asterixis, and tremor, may occur as a consequence of stroke,1 most often due to involvement of the basal ganglia or thalamus/subthalamus. However, IMs caused by anterior cerebral artery (ACA) territory infarction have rarely been reported.2 The author describes 9 patients who developed IMs after ACA infarction.

Subjects and Methods

Between May 1995 and December 1999, the author prospectively identified 9 patients with ACA infarction who presented with IMs at the Asan Medical Center. All the patients were examined and followed up by the author. The observed IMs were either asterixis or hemiparkinsonism. To assess the presence of asterixis, the patients were asked to stretch their arms with the wrists dorsiflexed. Legs were also extended with feet dorsiflexed at the ankle. Selected patients (n=4) were videotaped and repeatedly assessed.

Results

There were 4 men and 5 women, ranging in age from 47 to 79 years. No patients received medications, such as antiepileptics or antipsychotics, that might have caused IMs. History taking showed that there were no such IMs before the onset of stroke in these patients. Family history of IMs was also denied. Laboratory results, including serum ammonia, calcium, and electrolytes, were all within normal limits. The clinical findings are briefly summarized in Table 1, and 3 cases are described below. All patients had hemiparesis worse in the leg, and 4 had speech disturbances. None showed significant sensory loss or limb ataxia. Two patients showed alien hand signs; when asked to perform a task using both arms, the action of the right hand was not under conscious control, so that the patients often used the left hand to restrain the right one.3

Involuntary Movements

In all patients, IMs occurred in the arms, most markedly in the fingers, but never occurred in the legs. Four patients had asterixis; 1 (patient 6) had bilateral asterixis, more severe on the side contralateral to the lesion. Asterixis started immediately after the onset of stroke and disappeared within 2 to 7 days. Electromyography, performed in 1 patient (patient 8), confirmed episodes of electrical silence. Six patients had hemiparkinsonism: tremor, cogwheel rigidity, and hypokinesia. The tremors, of 4 to 5 Hz, started to appear gradually as the motor weakness improved at 2 to 4 weeks in the patients with initially severe arm weakness. Tremor occurred mainly during rest in 3 patients; in another 3, it was more prominent on arm stretching. The patients’ gait was slow and hemiparetic, and arm swing was greatly reduced. The dearth of movement was obvious in the involved arm. Patient 9 initially had asterixis that disappeared as her limb weakness progressed. One month later, as her motor dysfunction improved, she developed hemiparkinsonism. L-dopa was tried in 4 patients (patients 1,3,5, and 9); only 1 (patient 3) showed partial improvement of tremor. During the follow up of 2 to 33 (mean 19) months, the tremor and cogwheel rigidity gradually diminished in severity (Table 1).
Imaging Findings and Vascular Study

The lesions, analyzed by brain MRI in 7 patients and CT in 2 patients, are schematically presented in Figure 1. The patients with hemiparkinsonism (patients 1 through 5) usually had relatively large, rostral-dorsal lesions, probably involving the supplementary motor area (SMA), whereas those with asterixis (patients 6 through 8) had small lesions mainly involving the prefrontal area. Cerebral angiography was performed in 7 patients (transfemoral in 3, MR in 4). Atherosclerotic stenosis/occlusion at the proximal ACA was found in 6 patients. Patient 1, with a normal angiogram, had valvular heart disease. To assess the perfusion of the basal ganglia, [99m Tc]-L-ethylcysteinate dimer (ECD) SPECT was performed in 2 patients (patients 5 and 9) and showed perfusion defect in the ACA territory but not in the basal ganglia.

Patient 1

A 79-year-old hypertensive woman with valvular heart disease suddenly developed left hemiparesis (II/V, on the 0-V Medical Research Council scale, in the arm and II/V in the leg), and urinary incontinence. She was apathetic and nearly mute. The muscle strength gradually improved, and 1 month later she experienced only minimal hand weakness, although leg weakness (II/V) remained. Sensation was normally perceived. There was no ataxia on the finger-to-nose test. She started to show a resting tremor of 4 to 5 Hz in her left fingers. The tremor was rhythmic and of variable amplitude. Also observed were cogwheel rigidity, hypokinesia in the left limbs, and a loss of arm swing during walking. There was no tremor in the foot. Brain MRI showed an acute infarct in the frontal lobe of the right ACA territory. MR angiography showed normal results, although a distal portion of the ACA was not evaluated. L-dopa (200 mg)/benserzide (50 mg) 3 times a day did not improve her symptoms and was discontinued. Nevertheless, the tremor gradually decreased in intensity; 3 years later, mild cogwheel rigidity remained in the left arm but without tremor.

Patient 6

A 55-year-old hypertensive man suddenly developed right hemiparesis (II/V, on the 0-V Medical Research Council scale, in the arm and II/V in the leg), and urinary incontinence. She was apathetic but nearly mute. The muscle strength gradually improved, and 1 month later she experienced only minimal hand weakness, although leg weakness (II/V) remained. Sensation was normally perceived. There was no ataxia on the finger-to-nose test. She started to show a resting tremor of 4 to 5 Hz in her left fingers. The tremor was rhythmic and of variable amplitude. Also observed were cogwheel rigidity, hypokinesia in the left limbs, and a loss of arm swing during walking. There was no tremor in the foot. Brain MRI showed an acute infarct in the frontal lobe of the right ACA territory. MR angiography showed normal results, although a distal portion of the ACA was not evaluated. L-dopa (200 mg)/benserzide (50 mg) 3 times a day did not improve her symptoms and was discontinued. Nevertheless, the tremor gradually decreased in intensity; 3 years later, mild cogwheel rigidity remained in the left arm but without tremor.

Clinical Data of the Patients

<table>
<thead>
<tr>
<th>Patient/Sex/ Age, y</th>
<th>Limb Weakness (Arm/Leg) (Initial—at Time of IMs)</th>
<th>Other Symptoms</th>
<th>Type of IM</th>
<th>Interval Between Onset of Stroke and IMs</th>
<th>Course of IMs</th>
</tr>
</thead>
<tbody>
<tr>
<td>1/F/79 L, II/II—slight/II</td>
<td>Apathetic, mute, UI</td>
<td>Tremor (resting &gt;), CR, HK</td>
<td>1 mo</td>
<td>Mild CR at 36 mo</td>
<td></td>
</tr>
<tr>
<td>2/M/66 R, II/I—IV/II</td>
<td>Apathetic, mute</td>
<td>Tremor (postural, action &gt;), HK</td>
<td>1 mo</td>
<td>Improved at 6 mo</td>
<td></td>
</tr>
<tr>
<td>3/F/77 R, VI—IV/III</td>
<td>Apathetic, stuttering speech</td>
<td>Tremor (resting &gt;), CR, HK</td>
<td>21 d</td>
<td>Mild tremor and CR at 10 mo</td>
<td></td>
</tr>
<tr>
<td>4/F/62 R, II/I—IV/II</td>
<td>Apathetic, mute</td>
<td>Tremor (postural &gt;), CR</td>
<td>14 d</td>
<td>Mild CR at 8 mo</td>
<td></td>
</tr>
<tr>
<td>5/M/76 R, IV/II</td>
<td>Apathetic, UI</td>
<td>Tremor (postural &gt;), CR, HK</td>
<td>Simul</td>
<td>Improved at 12 mo</td>
<td></td>
</tr>
<tr>
<td>6/M/55 R, slight/III</td>
<td>Apathetic, speech difficulty, alien hand sign</td>
<td>Asterixis†</td>
<td>Simul</td>
<td>Improved at 7 d</td>
<td></td>
</tr>
<tr>
<td>7/M/47 R, slight/IV</td>
<td>Apathetic, speech difficulty</td>
<td>Asterixis</td>
<td>Simul</td>
<td>Improved at 3 d</td>
<td></td>
</tr>
<tr>
<td>8/F/67 L, slight/IV</td>
<td>Dysarthria</td>
<td>Asterixis</td>
<td>Simul</td>
<td>Improved at 2 d</td>
<td></td>
</tr>
<tr>
<td>9/F/59 R, none/slight*—IV/III†</td>
<td>Apathetic, transcortical aphasia, echolalia, alien hand sign</td>
<td>Asterixis—tremor (resting &gt;), CR, HK</td>
<td>1 mo (tremor)</td>
<td>Mild tremor at 24 mo</td>
<td></td>
</tr>
</tbody>
</table>

UI indicates urinary incontinence; CR, cogwheel rigidity; HK, hypokinesia; and Simul, simultaneously.

*At the time of asterixis; †at the time of tremor; ‡bilateral.

Schematic drawings of the lesion location in patients with ACA infarction. Numbers indicate patient number. Images of patients 2 and 8 were obtained by CT scan; all others are MRI.
test did not reveal ataxia. On stretching of the arms, there was asterixis in both hands, which was worse in the right. MRI showed an acute infarct in the left ACA territory. Transmural angiography showed an occlusion of the A2 portion of the left ACA. The asterixis gradually decreased in intensity, and was no longer observed 1 week later.

Patient 9
A 59-year-old hypertensive woman suddenly developed involuntary, shaking movements of the right forearm after 3 episodes of transient, right-sided weakness. Neurological examination showed that there was a slight weakness in the right leg. The gross arm shaking rapidly diminished in intensity and was observed as a pattern of asterixis only when her hands were stretched. Over the next 3 days, her limb weakness progressed to III/V, when the asterixis disappeared. She also developed transcortical mixed aphasia and alien hand sign. The arm weakness gradually improved; 1 month later, only slight clumsiness of the hand was observed while the leg was still severely weak (III/V). She started to show a resting tremor of 4 to 5 Hz in the right fingers. There was cogwheel rigidity and slight spasticity in the right arm and a loss of arm swing on gait. The tremor and rigidity gradually improved, and 2 years later there only remained a very mild, intermittent hand tremor.

Discussion
I found that there are 2 distinct types of IMs caused by ACA infarction: asterixis and hemiparkinsonism.

Hemiparkinsonism
The parkinsonism usually occurred after the patients’ limb weakness improved in the setting of initially severe hemiparesis. Their resting and monotonous tremor was similar to that of idiopathic Parkinson’s disease (IPD), but the tremor occurred more distinctly in half of the patients when their arms were stretched. However, whether this postural tremor differentiates it from a tremor of IPD remains unclear, because the patients with IPD occasionally show prominent postural tremor.4 The patients’ hemihypokinesia may be attributed at least in part to the pyramidal dysfunction. However, the dearth of arm movement and the loss of arm swing during gait were clearly more prominent than would be expected from the mild pyramidal dysfunction. Moreover, the cogwheel rigidity cannot be explained by hemiparesis or hemispasticity. Rather, the delayed onset of symptoms in patients with severe limb weakness and the observation that parkinsonian symptoms occurred in the arm but not in the leg suggest that the extrapyramidal symptoms are manifested only after the recovery of the initially severe pyramidal dysfunction.

Previously, frontal lobe tumors have been repeatedly described as a cause of hemiparkinsonism.5–8 However, parkinsonism due to ACA infarction has received surprisingly little attention.2,9 Our imaging studies showed that hemiparkinsonism is usually related to SMA or cingulate gyrus involvement (Figure 1, panels 1 through 5). Leenders et al8 hypothesized that local tissue pressure on the basal ganglia produced hemiparkinsonism in their patient with frontal meningioma. However, this mechanism seems unlikely in our patients, considering that there was no compressive effect on the basal ganglia on MR imaging, including coronal cuts (data not shown), and that the symptoms occurred in the subacute-chronic but not the acute stage. Thus, a more plausible explanation for the hemiparkinsonism would be a functional disconnection of the striatum-SMA circuitry.8 There has been ample evidence of anatomic connections between the striatum and the SMA,10–12 and the disconnection of the basal ganglia output to the SMA by an ACA infarction could produce parkinsonism. Previously, Playford et al13 demonstrated an impaired basal ganglia facilitation of medial frontal activation in patients with IPD. Dick et al9 even suggested that IPD symptoms actually reflect functional deafferentation of the SMA from the basal ganglia. The disconnection does not appear to be related to an alteration of the basal ganglionic blood flow, because SPECT studies showed normal perfusion in the basal ganglia in our patients. In a study using PET scan, Miyagi et al10 found decreased glucose metabolism and normal dopamine metabolism in the basal ganglia of a patient with meningioma at the SMA area that produced hemiparkinsonism. They speculated that the tumor might have caused synaptic dysfunction of the striatum as a whole, but without an impairment of the presynaptic dopaminergic nerve terminals. Consistent with this observation, L-dopa was not effective in most of our patients. Nevertheless, the hemiparkinsonism spontaneously improved in all the patients.

Asterixis
Unlike hemiparkinsonism, the asterixis correlated with initially mild limb weakness associated with small, anteriorly located lesions (Figure 1, panels 6 through 8). Asterixis, one type of negative myoclonus,14 results from a sudden cessation of electrical activity in the extensor muscles, due possibly to an intermittent inhibition of the spinal neuronal system that mediates voluntary tonic extension of the limb. Asterixis may occur after focal cerebral lesions involving the midbrain, thalamus, parietal lobe, or the frontal cortex.14–16 However, ACA infarction as a cause of asterixis has received little attention. Young and Shahani14 suggest that sustained muscle contraction is related to the neural subsystem involving the medial frontal cortex, parietal lobe, and the ventrolateral thalamus. This system may have been disrupted in our patients with lesions mainly involving the prefrontal cortex. It is noteworthy that asterixis was bilateral in patient 6. Although concomitant involvement of the contralateral frontal cortex may be possible, previous studies have also reported bilateral asterixis due to unilateral brain lesions,15,17 which suggests that the focal unilateral ACA infarction itself produced bilateral asterixis.

References


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