Transient Involuntary Movement of the Leg (Monoballismus) during Cerebral Angiography

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Summary: Transient involuntary movement of the leg developed during diagnostic or therapeutic angiography in five patients. The movement was jerky, brisk flexion/extension of the hip and knee joints on the side contralateral to acute ischemia. This movement may occur as a result of thromboembolic cerebral ischemia or as an ischemic complication of angiography.

Index terms: Brain, ischemia; Cerebral angiography, complications; Movement

Although persistent involuntary movement of the limb, such as hemichorea and hemiballismus, caused by cerebral ischemia is well known (1–6), transient involuntary movement is an uncommon manifestation of acute ischemia (7–14). Moreover, transient involuntary movement of the leg during cerebral angiography is so rare that we were unable to find any cases reported in the literature. With increasing opportunities to perform diagnostic and therapeutic angiography for cerebral ischemia, we have experienced five cases of such transient involuntary movement. We present them here and discuss their clinical importance.

Case Reports

The transient involuntary movements during angiography in the present series were very similar. The involuntary movement was a brisk, jerky flexion of the hip and knee joints of one limb, most often flexion of both joints to about 20°, with elevation of the knee about 10 to 15 cm above the angiographic table, followed by rapid extension of the limb. The involuntary movement occurred on the side contralateral to the ischemic hemisphere. Except for case 2, the involuntary movement was confined to the one lower extremity, and no such movement was observed in the upper extremity. The movement was involuntary, and the patient could not halt it. No involuntary movement was observed before or after angiography except for case 2. The movement was repetitive, occurring every 10 to 60 seconds during cerebral angiography, with the interval between movements gradually lengthening. The consciousness of the patient did not change during the involuntary movement.

Involuntary movements in cases 3 through 5 were related to cerebral ischemia occurring before angiography; they are summarized in the Table. In these cases, endovascular treatment for acute cerebral ischemia, either local fibrinolysis or percutaneous transluminal angioplasty, was attempted because the patients were referred to us in the ultraacute ischemic stage. In cases 1 and 2, involuntary movement occurred primarily as a complication of diagnostic and/or therapeutic cerebral angiography, and they are described in detail.

Case 1

This 64-year-old man's history included one occurrence of a transient ischemic attack, presenting as weakness of the right upper limb and lasting a half day. Cerebral angiography disclosed marked stenosis (> 90%) of the petrosal portion of the left internal carotid artery. Cerebral blood flow measurement disclosed diffuse, moderate hypoperfusion of the left hemisphere. We performed percutaneous transluminal angioplasty under local anesthesia. Immediately before balloon angioplasty, acute occlusion of the left internal carotid artery occurred, probably because of the reduction in blood flow and the kinking of the proximal internal carotid artery accompanying introduction of the large (8F), stiff guiding catheter. The patient had loss of sensation, followed by weakness, of the right upper limb, especially in the distal portion. This was followed by the involuntary movement of the right lower limb.

Using balloon catheters (Stealth, Target Therapeutics, Fremont, Calif) with inflated balloon diameters of 2.0 and 3.5 mm, respectively, we performed angioplasty at the stenotic portion of the internal carotid artery. Blood flow was restored 40 minutes after the complete cessation of carotid flow. The involuntary movement of the right lower limb soon subsided. The patient recovered completely from weakness and sensory loss in the right upper limb soon after recanalization. No infarction was observed on

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Patients with cerebral ischemia occurring before angiography

Feature	Case 3	Case 4	Case 5
Age, y/sex	71/M	59/M	38/M
Side	R	R	R
Occluded vessel	ICA (C-2 portion)	MCA (M-1 portion)	MCA (M-1 portion)
Cause	Atrial fibrillation, cardioembolism	MCA thrombosis	MCA dissection, embolism
Initial symptoms	Hemianopsia	Hemiparesis, drowsiness	Hemiplegia, drowsiness
Treatment	Fibrinolysis	Percutaneous transluminal angioplasty	Fibrinolysis
Outcome of intervention	Nonrecanalization	Recanalization	Recanalization
Infarction on computed tomography	MCA, PCA	MCA	MCA
Clinical outcome	Hemiparesis, hemianopsia	Hemiparesis	Moderate disorientation

Note.—ICA indicates internal carotid artery; MCA, middle cerebral artery; and PCA, posterior cerebral artery.

the follow-up magnetic resonance images taken 5 days after intervention. The patient was discharged with no neurologic deficit.

Case 2

This 76-year-old woman suddenly had right oculomotor palsy, with no accompanying headache. Cerebral angiography disclosed a large aneurysm at the origin of the right superior cerebellar artery and a small basilar bifurcation aneurysm. The right vertebral artery was larger than the left. During both left and right vertebral angiography, the patient had involuntary movement of the both lower legs. The abnormal movement started when vertebral catheterization began and gradually disappeared within a half day. The involuntary movement on the right side was not coincidental to that on the left side.

Taking the patient's age and general condition into consideration, we decided to treat only the aneurysm at the origin of the superior cerebellar artery with mechanically detachable coils (Target Therapeutics, Fremont, Calif). One week after diagnostic angiography, we performed coil embolization through the right vertebral artery, using two platinum coils. When the right vertebral artery was catheterized, the patient had the involuntary movement of the leg again, but this time it was confined to the left leg. This involuntary movement disappeared soon after the coil embolization. The cause of the involuntary movements on two separate occasions is thought to be reduced blood flow induced by catheterization of the vertebral arteries. Although the patient's right oculomotor palsy persisted, she was otherwise neurologically healthy.

Discussion

Nonepileptic, involuntary movements involving the unilateral body are referred to by such terms as *hemiballismus* (2, 3, 5), *limb shaking* (12, 14), *involuntary tonic spasms of a limb* (15), and *repetitive involuntary movement* (13). Such movement usually involves one side of the body (hemiballismus), but occasionally involves one limb (monoballismus). These movements are described in such terms as wavering, jerking, shaking, flinging, flapping, kicking, shivering, twitching, or trembling of the arm and/or leg.

Ischemic lesions in the subthalamic nucleus of Luys may cause hemiballismus on the contralateral side (2). Other types of lesions in the cerebral cortex, striatum, globus pallidus, thalamus, internal capsule, or brain stem may result in a similar clinical picture (2, 3, 5, 6, 15–18). These regions are efferent or afferent pathways or projection areas of the subthalamic nucleus. Not only ischemic lesions, but hemorrhagic lesions, brain tumors, cerebral trauma, and demyelinating disease in the same regions also may result in hemiballismus (1, 3, 16, 19, 20).

From the vascular anatomic point of view, the blood supply to the subthalamic nucleus comes from the anterior choroidal artery, the posterior communicating artery, and/or the posterior cerebral artery (21). Temporary or permanent ischemia of the subthalamic nucleus or its related areas attributable to stenosis or occlusion of the internal carotid artery (11, 12), the middle cerebral artery, the posterior cerebral artery, or the basilar artery (8, 11, 18) can cause hemiballismus. Hemiballismus as a symptom of vertebrobasilar insufficiency also has been reported (8, 22). In the case of ischemia of the posterior circulation, bilateral involuntary movement of the limbs can occur, as in our case 2, because blood supply from the basilar artery is bilateral. It is unclear why the involuntary movement involved only the leg, whereas the upper limb was not involved. Because the affected lesion, such as the subthalamic nucleus, has a somatotopic representation (23),

the lesions representing the lower limb may be preferentially involved.

The involuntary movement of the leg can occur only when the pyramidal tract is intact. The blood supply to the pyramidal tract should be spared for the involuntary movement to be expressed. Lesions in the basal ganglia involving the pyramidal tract do not cause the involuntary movement. Thus, in none of the cases reported here, except case 5, did the patient have severe hemiparesis on admission. In case 5, the patient was hemiplegic on admission, but he recovered from hemiplegia after fibrinolysis, suggesting reversible ischemia of the pyramidal tract.

Though involuntary movements of the limb may last as long as several years, brief episodes of hemiballismus are rare (4, 6, 7, 9, 22). There are several possible explanations for the transience of the involuntary movement during angiography: (*a*) the ischemic damage regresses after resolution of the initiating pathogenic event, either hemodynamic or thromboembolic; (*b*) the ischemia extends to the pyramidal tract, thus halting the manifestation of the involuntary movement; or (*c*) the angiographic procedure causes the ischemia, thus it should be regarded as an angiographic complication.

Several mechanisms of ischemia might occur only during angiography including: (*a*) reduction of blood flow by catheterization. (The extreme condition is the occlusion caused by wedging or kinking of the vessel); (*b*) vasospasm by mechanical manipulation, resulting in reduction of blood flow; and (*c*) embolic complications. To ameliorate these conditions, it is necessary to normalize the blood flow by removing the catheter or reopening the occluded vessel by angioplasty or fibrinolysis, if possible. Contrast material also may cause adverse effects to the brain by hyperosmolality (24) and toxic effects (25). When cerebral blood flow is stagnated, intracranial vessels may be excessively exposed to the contrast material, a possible solution is to use an isotonic, less toxic nonionic contrast material.

Involuntary movement of the limb can occur as a symptom of transient ischemia, which is a warning sign of possible major stroke (7, 10–13). Focal seizure with tonicoclonic movements also is known to occur in patients with previous or acute cerebral ischemia (26). However, Yanagihara et al (13) reported that involuntary movements occurred as transient hemodynamic ischemic episodes in patients with the carotid occlusive disease whose electroencephalograms showed no epileptiform activities at rest or even during the involuntary movement. Although we have not examined any of our patients with electroencephalograms, we think their involuntary movements were caused by cerebral ischemia because our patients had clinical pictures similar to those of Yanagihara's series, and they had no seizure history either before or after angiography. Thus involuntary movements caused by cerebral ischemia should be carefully differentiated from focal seizures (4, 12, 13, 17).

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