Delayed Onset Hand Tremor Caused by Cerebral Infarction

Jong Sung Kim, MD

Background and Purpose: Hand tremor has been rarely described as a manifestation of stroke. 

Case Descriptions: Three patients developed delayed onset hand tremor due to cerebral infarctions on the contralateral side, two in the caudate nucleus and the third in the thalamus. The tremors of all three patients were similar to parkinsonian tremor, but less monotonous.

Conclusions: Delayed-onset hand tremor should be considered as one of the movement disorders caused by cerebral infarction. (Stroke 1992;23:292-294)

Involuntary movements such as chorea,1-12 dystonia,13,14 or asterixis15-18 can be produced by cerebral infarction or hemorrhage. However, hand tremor has been rarely described as a manifestation of stroke. I report three patients who showed delayed onset hand tremor due to cerebral infarction.

Case Reports

Case 1
A 45-year-old hypertensive woman developed sudden dysarthria and left-sided weakness in September 1989. She had mild dysarthria, weakness of the lower part of the left face, left-sided hemiparesis, hemihypesthesia, and hyperreflexia. The hemiparesis gradually improved. Three months later, she began to notice a left-hand tremor, which was repetitive, rhythmic (approximately 4-5 Hz), and characterized by flexion-extension movements of all left fingers at the metacarpophalangeal joints. Although slightly spastic, her left arm was free of cogwheel rigidity. The tremor appeared mainly during rest and disappeared during sleep; it diminished when her hands were stretched out or when she used her left arm. The amplitude of the tremor became greater when the patient was anxious. Despite variable amplitude, the frequency of the tremor remained nearly constant.

Brain computed tomographic (CT) scans, performed at 7 days and at 5 months after onset of the stroke, showed identical findings: a cerebral infarct involving the right caudate nucleus, anterior limb of the internal capsule, and anterior part of the putamen (Figure 1). Four-vessel angiogram was normal. 

Case 2
A 65-year-old hypertensive man developed left-sided weakness in March 1989. He gradually recovered until December 1990, at which time tremulous left-finger movements were noticed. Brain magnetic resonance imaging (MRI) performed in April 1991 showed a lacunar infarct involving the right caudate nucleus and the anterior limb of the internal capsule (Figure 2). Another clinically silent ischemic lesion was found in the deep parietal area. Neurological examination showed mild dysarthria, left facial palsy, hemihypesthesia, and clumsiness of the left arm. Mild hyperreflexia in the left extremities without cogwheel rigidity was noticed.

The tremor, which appeared only during the day at a frequency of 5-6 Hz, was characterized by flexion-extension movements of all the left fingers at the metacarpophalangeal joints. It occurred intermittently and became more active when the patient was conversing or writing with his right hand, but disappeared when he used his left arm. When the tremor was highly active, it was often accompanied by mild tremor at the left metacarpocarpal and elbow joints.

Case 3
A 65-year-old diabetic man developed sudden weakness of the left arm in September 1990. Neurological examination showed mild left hemiparesis and markedly decreased sensation of all modalities in the left half of the body. Brain CT scan performed 3 days after onset showed a round infarct involving the right thalamus and the posterior limb of the internal capsule (Figure 3). Two months later, he complained
of unpleasant, painful numbness in the left side of his body, which was partially alleviated by carbamazepine medication.

In February 1991, he developed left-hand tremor, which continued even after carbamazepine was withdrawn. The main component of the tremor consisted of rhythmic (4–5 Hz) flexion-extension movements of the left thumb and index finger. Occasionally, mild adduction-abduction movements of the other fingers were also noticed. The tremor was intermittent; it became more prominent when he used his right hand or was in conversation and diminished when he used his left hand. Repeated brain CT scans revealed the same findings as before. Also observed were intermittent dystonic postures of the left hand: ulnar deviation of the wrist, slight flexion of the fingers, and adduction of the thumb. No cogwheel rigidity was noticed in his left arm.

Discussion

The three patients described here showed delayed-onset hand tremors as manifestations of cerebral infarctions. These were resting tremors that became active when the patients were anxious or concentrating on something, disappeared when they slept, and diminished when they used the involved arms. Like the parkinsonian tremor, these were characterized by a rhythmic (4–6 Hz), “pill-rolling”-type finger tremor. However, they were less monotonous than parkinsonian tremor, occurred intermittently, and had an amplitude that fluctuated more randomly than the usual parkinsonian tremor.

Movement disorders such as chorea (or ballism),1-12 dystonia,13,14 or asterixis15-18 have been frequently described as manifestations of basal ganglionic or capsular strokes. However, hand tremor has been mentioned only rarely in the literature. A few patients with arm tremors associated with basal ganglionic or thalamic arteriovenous malformations have been described,19-21 and Grimes et al13 presented a patient with a caudate infarct who showed a coarse arm tremor in addition to delayed onset posthemiplegic dystonia. I also have seen patients with thalamic infarct who showed delayed-onset arm dystonia with action tremor (unpublished observation). However, these tremors were not characteristic of parkinsonian tremor, but were coarse action tremors usually greater proximally than distally. Recently, Dewey and Jankovic10 collected 21 patients with hemichorea–hemiballism. One of these patients showed a 5- to 7-Hz intention and resting tremor that the authors described as parkinsonian tremor. How-
ever, their patient did not suffer from stroke, but had a cystic glioma in the midbrain.

The patients described here lacked cogwheel rigidity and bradykinesia and thus could not be classified as having a vascular parkinsonism, but their tremors may share a pathogenic basis identical with those associated with idiopathic parkinsonism. Although the pathogenesis of parkinsonian tremor is still unknown, modified outputs from the basal ganglia were thought by some to cause a development of rhythmic activities in a portion of the thalamus, probably the nucleus ventralis intermedius. The rhythmic signal could follow a path to the motor cortex and descend to the spinal motor neuron pool via the pyramidal tract.

It is noteworthy that there were time gaps ranging from 3 to 21 months between onset of stroke and initiation of the tremor. In patients 1 and 2, the CT and MRI scans showed infarcts involving the caudate nuclei. It could therefore be speculated that, during those time gaps, the lesions may have influenced the thalamus to generate rhythmicity. Dooling and Adams, Grimes et al, and Pettigrew and Jankovic proposed similar mechanisms to explain the pathogenesis of delayed-onset athetosis or dystonia in their patients. Grimes et al also noticed thalamic atrophy on the ipsilateral side of the lesions, which could provide possible evidence for the thalamic modulation from the basal ganglia.

In our third patient with delayed onset intermittent dystonia and hand tremors, the lesion was situated in the thalamus, probably affecting the ventrolateral nucleus (Figure 3). It remains obscure how the involuntary movement of this patient acquired rhythmicity. Podlas et al and Lobos-Antunes et al described patients with coarse arm tremor secondary to thalamic arteriovenous malformations. The latter authors suggested that the ischemic lesion involving the substantia nigra may have caused the tremor in their patients. However, this postulation could hardly be applied to our patient with a discrete lesion in the superior-lateral portion of the thalamus. Rather, an alteration of the thalamic function itself seems more likely to have produced rhythmicity in this patient.

Regardless of the pathogenesis, it could be concluded from the three cases described here that delayed-onset hand tremor should be considered as one of the movement disorders caused by stroke.

Acknowledgment

I wish to thank Prof. Onyou Whang for her helpful suggestions in preparing this manuscript.

References


KEY WORDS • cerebral infarction • tremor
Delayed onset hand tremor caused by cerebral infarction.

J S Kim

Stroke. 1992;23:292-294
doi: 10.1161/01.STR.23.2.292

Stroke is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1992 American Heart Association, Inc. All rights reserved.
Print ISSN: 0039-2499. Online ISSN: 1524-4628

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://stroke.ahajournals.org/content/23/2/292

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Stroke can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Stroke is online at:
http://stroke.ahajournals.org/subscriptions/