

Hyperkinetic Movement Disorders Caused by Corpus Striatum Infarcts: Brain MRI/CT Findings in Three Cases

Roger E. Kelley, MD

Pawan K. Jain, MD

ABSTRACT

Three patients with hemichorea/hemiballismus/hemidystonia caused by discrete contralateral infarction of the corpus striatum are presented. The infarcts were all small on CT or MRI brain scan and were lacunar in type. Small discrete infarction of basal ganglionic structures allows such adventitious movements to be manifested. Involvement of contiguous areas, seen with larger infarcts, can suppress such movements. The infrequency of such hyperkinetic movement disorders, and the subtle infarct appearance on brain scan, can lead to a delay in the diagnosis.

Key Words: brain, magnetic resonance imaging, computed tomography, hyperkinetic movement disorder.

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Cerebrovascular disease can involve subcortical structures, resulting in movement disorders such as hemichorea, hemidystonia, hemiballismus, and parkinsonism. This is an uncommon occurrence, and it has been theorized that hyperkinetic movements are actually rare because involvement of contiguous subcortical structures serves to suppress such movements.¹ Therefore, in order for hyperkinetic movements to appear, there must be discrete involvement of basal ganglionic or thalamic structures. The authors present three patients who had such a condition and who were found, on either computed tomography (CT) or magnetic resonance imaging (MRI) brain scan, to have subtle, discrete areas of involvement of the corpus striatum. These cases illustrate the interesting anatomic localization associated with such hyperkinesia as well as the potential for misdiagnosis.

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From the Department of Neurology, LSU Medical Center, Shreveport, LA.

Address reprint requests and correspondence to Dr. Roger E. Kelley, Department of Neurology, LSU Medical Center, 1501 Kings Highway, Shreveport, LA 71130.

Patient Reports

Patient 1. A 37-year-old right-handed man was admitted to the hospital on June 24, 1998, with acute onset of right-sided dystonic movements affecting his arm and leg. Past medical history revealed chronic schizophrenia; complete resection of a left frontal grade II astrocytoma, followed by radiation therapy, in 1995; seizure disorder; recently diagnosed hypertension; hyperlipidemia; and a 40 pack-year smoking history. Medications on admission included phenytoin, diphenhydramine, benztropine mesylate, lovastatin, and fluphenazine. He was initially thought to have focal seizure activity.

On examination, his vital signs were unremarkable except for a blood pressure of 164/94 mm Hg. Carotid pulses were 2+ bilaterally with no bruit heard. Cardiac auscultation revealed regular sinus rhythm with no murmur. His mental status and speech were remarkable for a chronic anxious state with somewhat pressured speech. Results of a cranial nerve examination were normal except for a mild right facial asymmetry. He had adventitious movements of his right side, especially of the right upper extremity, with recurrent dystonic posturing. A mild right-sided weakness was detected, and he favored his right leg when he walked.

The results of his routine admission blood work were normal, as were the results of his electrocardiogram. His electroencephalogram demonstrated left frontal slowing and was unchanged from a study performed 1 year before. His CT brain scan (Fig. 1) revealed a left lentiform nucleus infarct. This had not been seen on his previous CT brain scan, but no change was noted in the area of left frontal encephalomalacia which represented the previous tumor resection. His carotid/vertebral duplex scan with transcranial Doppler revealed no significant extracranial or intracranial occlusive disease. He refused performance of an MRI/magnetic resonance angiography as well as consideration of cerebral arteriography. He was discharged home on June 28 in an improved condition, and his right-sided dystonic movements subsided spontaneously and completely within 1 week of presentation.

Patient 2. A 60-year-old right-handed man had a 9-week history of involuntary movement of the right upper ex-

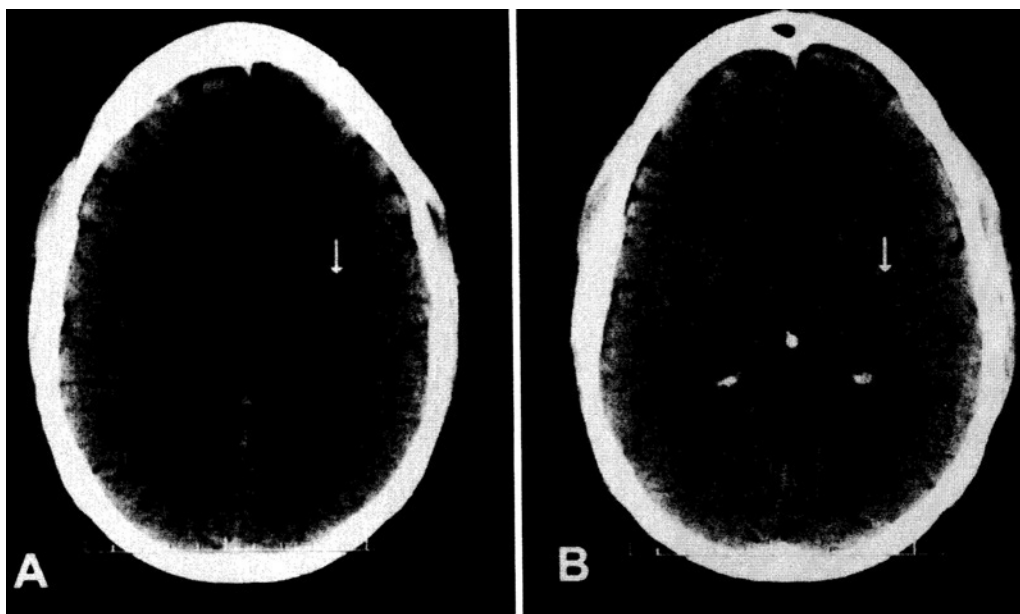


Figure 1. Noncontrast CT brain scan which demonstrates lacunar-type infarction (arrow) of the left lentiform nucleus seen on two transaxial planes (A and B) in Patient 1 on the day of presentation.

tremity and pursing mouth movements. According to the patient's wife the onset was acute, noted at breakfast one morning, but the patient did not seek immediate medical attention. Past medical history revealed longstanding hypertension; a right occipital infarct in September of 1994, which was complicated by a seizure disorder; and longstanding heavy cigarette consumption. He was under treatment with phenytoin, verapamil, and 1 baby aspirin per day. He had no history of neuroleptic medication ingestion, alcohol abuse, head trauma, or toxic environmental exposure. There was no family history of a movement disorder.

His examination on July 18, 1997, revealed pursing mouth movements. Funduscopic examination revealed arteriolar narrowing. He had no Kayser-Fleischer rings. His blood pressure was 118/80 mm Hg. His pulse was 76/minute and regular. He had a soft right midcarotid bruit and bilateral supraclavicular bruits. Cardiac auscultation revealed regular rhythm with no murmur, gallop, or click. He was uncooperative in cognitive assessment, which represented his baseline personality, but no obvious cognitive deficit was present. He had no dysarthria. Cranial nerve examination was remarkable for oro-buccal dyskinetic movements. He had normal motor tone and strength throughout.

He had recurrent choreiform movements of his right upper extremity with a "milkmaid" grip. He had fairly good fine finger movement and foot tapping bilaterally. He was somewhat clumsy on both sides, with diminished rapid alternating movement and fair finger-to-nose and heel-to-shin testing. He had a mildly impaired check reflex on his right side. He tended to favor his left leg when walking and had impaired tandem walking. His deep tendon reflexes were 2+ in both upper extremities and 1+ in

both lower extremities. His Babinski response was negative on the left and equivocal on the right.

His carotid scan revealed relatively minor, bilateral internal carotid artery plaque formation, with approximately 30% stenosis on the right and 30% to 40% stenosis on the left. Previous cardiac evaluation was unremarkable. His July 22, 1997, MRI brain scan, performed with and without contrast, revealed an old right occipital infarct, which had been seen on previous scans, as well as a left putaminal infarct not previously seen (Fig. 2). Over a 1-year follow-up, there has been a progressive marked attenuation of the right-sided choreiform movements, and the oro-buccal dyskinesias have completely resolved.

Patient 3. A 70-year-old right-handed woman was admitted to LSU Medical Center Hospital on March 2, 1998, with recurrent episodes of left-sided numbness. The episodes had been ongoing for 1 week and primarily involved the left face and left upper extremity. The episodes lasted 1–5 minutes each, and several were associated with a sense of weakness in the left arm. Results of her admission neurologic examination were normal, as were the results of her noncontrast CT brain scan. Past medical history was pertinent for hypertension since 1974, non-insulin dependent diabetes mellitus, and an 80 pack-year smoking history. Medications on admission were metformin 850 mg twice a day, piroxicam 20 mg per day, quinapril 40 mg per day, and hydrochlorothiazide 25 mg per day.

Her routine admission blood work was unremarkable, as were the results of her cardiac evaluation, carotid non-invasive imaging, and EEG. She reported two further episodes of left-sided numbness after admission and was prescribed intravenous heparin. She had no further episodes while taking heparin and underwent a cerebral arterio-

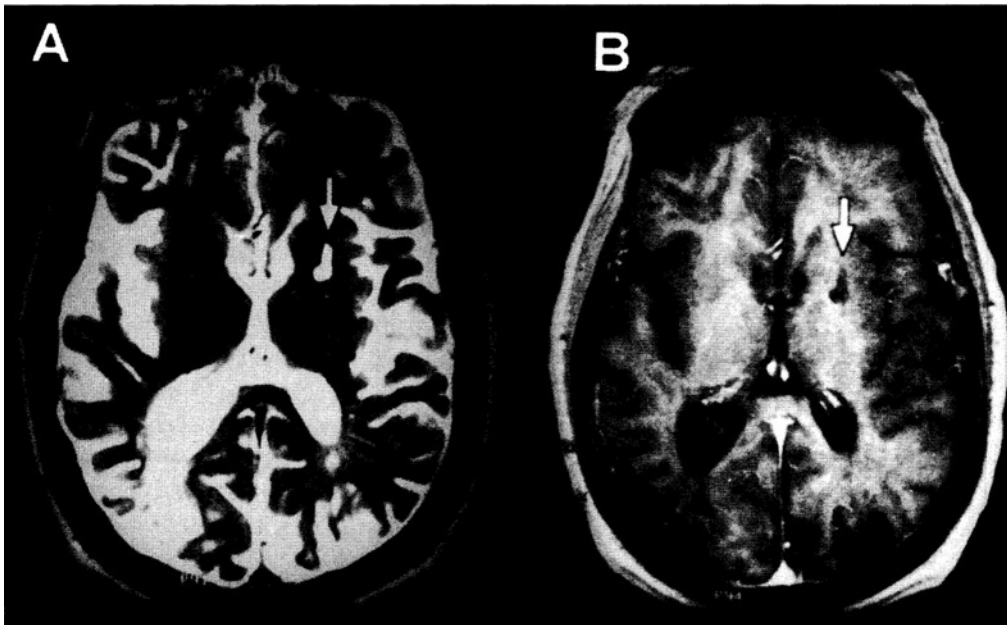


Figure 2. Fast spin echo MRI (1.5-Tesla GE Signa) brain scan which demonstrates: A. discrete putaminal infarct (arrow) on T2-weighted (TR 3500/TE 16, NSA 2) in patient 2. B. absence of contrast enhancement of the infarct (arrow) on the T1-weighted (TR 433/TE 25, NSA 4) image. This study was obtained 10 weeks after the onset.

gram on March 9th. This study revealed nonstenotic plaque formation of both internal carotid origins. She was prescribed 325 mg of aspirin per day after the procedure and was discharged home on March 10th.

On March 28th, she noted the sudden onset of uncontrolled flailing of her left arm accompanied by a sense of gait imbalance, and she was also observed to have orobuccal dyskinesias. In addition, she reported painful dysesthesias of her left arm. When seen in the emergency department on April 5th, she was sent home with a presumptive diagnosis of cellulitis caused by a recent intravenous line, with her movements reflective of local pain. She was not admitted until April 9th and, on neurologic examination, she had prominent oro-buccal dyskinesias with left upper extremity hemiballistic movements. Her mental status was intact and no speech disturbance was noted. There was minor hypersensitivity to pinprick over the left side of her face, and this was more pronounced over her left arm and hand. No gross weakness was noted, but there was somewhat diminished dexterity in the left hand. Her deep tendon reflexes were 1 to 1+ in both upper extremities, 0 to 1 at both knees, and absent at both ankles. The Babinski reflex was positive on her left.

A noncontrast CT brain scan on the day of admission showed normal results. She was prescribed oral haloperidol 0.5 mg thrice daily, gabapentin 100 mg thrice daily for pain control, and ticlopidine 250 mg twice daily. She reported a significant improvement in her left-sided pain, and her abnormal adventitious movements were markedly attenuated within 1 week of treatment. An MRI brain scan was obtained on April 13th and it demonstrated an infarct within the region of the right corpus striatum, primarily involving the putamen (Fig. 3). The anatomic lo-

calization of the corpus striatum infarcts in the three patients is demonstrated in Figure 4.

Discussion

The corpus striatum is composed of the caudate nucleus, the globus pallidus, and the putamen. The latter two structures are often grouped together as the lentiform nucleus. It is generally felt that vascular lesions of the corpus striatum are the most common explanation for hemichorea and/or hemiballismus.¹⁻⁵ Lacunar-type infarction of the contralateral putamen and caudate nucleus, demonstrated by CT brain scan, has been associated with hemichorea-hemiballismus.¹ The authors of this report theorized that the rarity of such a movement disorder in stroke was attributable to either coexistent involvement of the internal capsule, resulting in hemiparesis (which does not allow the abnormal movements to be expressed) or coexistent involvement of the globus pallidus (which has the potential to suppress the movements).⁶ Hemiballismus is most commonly seen as a result of lesions of the subthalamic nucleus; however, involvement of the striatopallidal pathway, which is GABAergic and inhibitory to pallidal neurons, can lead to disinhibited motor thalamic structures.⁷ In a report of 25 patients with hemiballismus, cerebral ischemia or hemorrhage was the presumptive cause in 19 subjects.⁸ Of interest, 16 subjects also had hemichorea. It is reported that hemichorea seen in association with a striatal ischemic lesion can also be related to thalamic disinhibition.⁹ The lesion localization was not identified in most of these cases, and discrete involvement of the subthalamic nucleus was observed in only six patients. In certain cases of hemichorea and/or hemiballismus, the vascu-

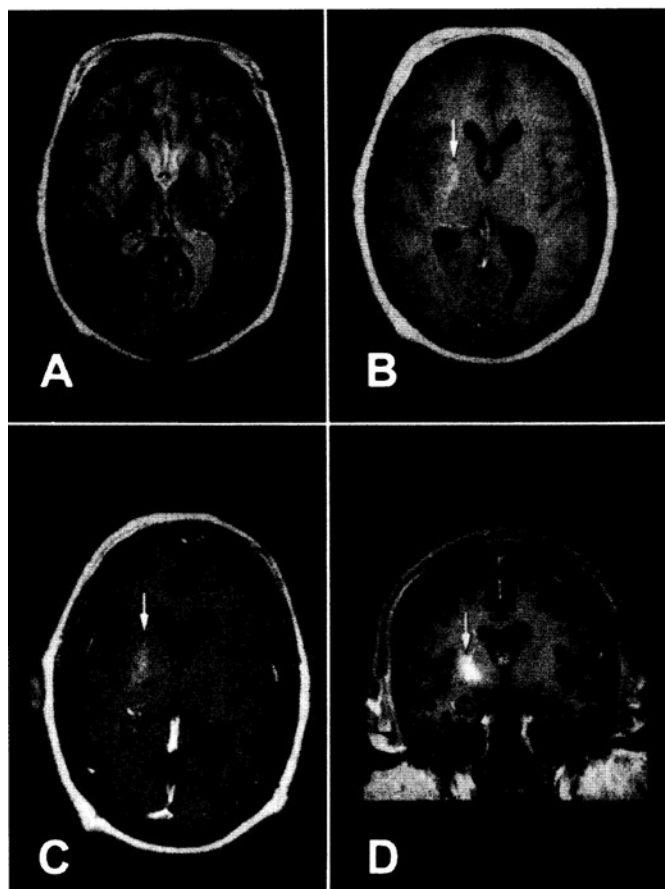


Figure 3. A. T2-weighted (TR 3800/TE 90, NSA 2) fast spin echo, MRI brain scan in Patient 3 which reveals no distinct infarct. B. T1-weighted (TR 560/TE 15, NSA 4) image demonstrates a subtle area of increased signal intensity (arrow) in the region of the right putamen. C. Contrast-enhancement of the right putaminal infarct (arrow) seen on transaxial plane. D. Most dramatic demonstration of the contrast-enhancing infarct (arrow) is seen on coronal plane. The increased signal intensity seen on the noncontrast T1-weighted image was interpreted to represent hemorrhagic transformation of the infarct. This study was obtained 2 weeks after the onset.

lar lesion involves the caudate nucleus and the internal capsule with ¹ or without ⁵ involvement of the putamen.

There have also been cases of multiple small vessel infarcts, involving subcortical structures of both hemispheres, which result in hyperkinetic movement disorders.¹⁰⁻¹² In addition, infarcts of the basal ganglia can result in focal (contralateral) dystonia.^{13,14} Dystonia has been described after both striatopallidal and thalamic stroke.¹⁵ The corpus striatum appears to be the most common area of involvement in hemidystonia.¹⁶ It has been reported that focal vascular lesions of the basal ganglia are associated with movement disorders in roughly half of all cases.¹⁷ However, according to one recent series,¹⁸ hyperkinetic movement disorders were seen in only 1% of patients with acute stroke. The outcome is considered excellent in most cases,¹⁹ but others report that resolution of the hyperkinetic movements is unpredictable.²⁰

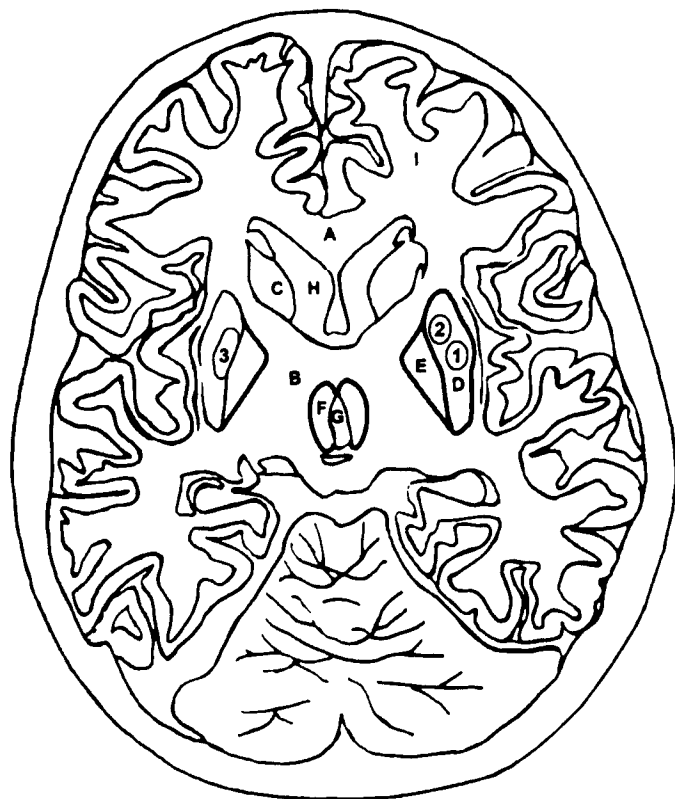


Figure 4. Schematic anatomical diagram of the brain transaxial plane which demonstrates the area of striatal infarction in patients 1, 2, and 3. A = genu of corpus callosum, B = genu of internal capsule, C = head of the caudate nucleus, D = putamen, E = globus pallidus, F = thalamus, G = third ventricle, H = frontal horn of lateral ventricle, and I = frontal cortex.

Our three patients all had small lacunar-type infarcts which were, by definition, discrete and subtle. Two patients were taking phenytoin at the time of presentation, and phenytoin has the potential to promote chorea.²¹ However, the choreiform movements were not diffuse, they were correlated with a vascular movements of the corpus striatum, and the movement disorder remitted over time. One patient was taking both phenytoin and fluphenazine, and phenothiazines are well known to promote extrapyramidal symptoms. This patient had complete remission of his hemidystonia within a week of acute presentation, and this was despite continuation of both medications. One of our patients was initially thought to have focal seizure activity, another waited 9 weeks before seeking medical attention, and the third was originally misdiagnosed as having a local inflammatory process with hyperkinetic movements caused by pain. Our cases illustrate that hyperkinetic movement disorders after small basal ganglion-thalamic infarction can still be a diagnostic challenge. These infarcts have the potential to be missed on CT or MRI brain scan unless one is specifically looking for the most frequent anatomic areas of involvement.

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