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Case Report

Bilateral Limb-Kinetic Apraxia Following Left Peri-Central Sulcus Hemorrhage

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Abstract

Post-stroke limb-kinetic apraxia is rarely reported. We report a case involving a 39-year-old man who showed bilateral limb-kinetic apraxia due to a unilateral focal lesion of left pericentral sulcus hemorrhage. The patient had full strength in both upper extremities and only mildly impaired proprioception in the right upper extremity. His dexterity, however, was impaired in both hands, resulting in impairment of his ability to perform basic activities of daily living (ADLs). At discharge from the inpatient rehabilitation service, his limb-kinetic apraxia improved to the level of modified independence in basic ADLs. At 6 months after the onset, he maintained the regained dexterity in basic ADLs. It is important to differentiate limb-kinetic apraxia from hemiparesis to understand the neural mechanisms of underlying stroke symptoms and develop an effective rehabilitation treatment.

Keywords: Stroke; Limb-Kinetic Apraxia; Rehabilitation Medicine; Sulcus Hemorrhage; Activities of Daily Living (ADLs).

Introduction

Although post-stroke apraxia is a common and disabling higher-order neurological deficit, there is no standardized evaluation method or treatment for this condition [1,2]. The existence of limb-kinetic apraxia has been questioned in the past [3,4]. Hemiparesis, impairment of elementary motor and sensory innervations in the brain, is the most common symptom of stroke. On the other hand, in apraxia, patients lose learned and skilled movements due to higher-order motor deficits, despite preserved elemental motor and sensory innervation; [2]. herefore, the effective rehabilitation approach for patients with apraxia can be different. We report a case of bilateral limb-kinetic apraxia that occurred after a left postcentral gyrus hemorrhage, extending subcortically and anteriorly. The impaired motor control in this case was distinctly different from hemiparesis and manifested bilaterally from the left hemisphere lesion.

Learning Points/Take Home Messages

- In limb-kinetic apraxia, a patient loses dexterity in fine motor movements (for example, buttoning the shirt), which cannot be attributed to elemental neurological deficits, such as weakness, cerebellar ataxia, or impaired proprioception.
- Although limb apraxia is commonly seen after a stroke, post-stroke limb-kinetic apraxia has rarely been reported, which may be due to the difficulty in differentiating it from hemiparesis.
- Recognizing post stroke limb-kinetic apraxia is important to understand its pathologic neural mechanism and recovery to develop effective and evidence-based medical management.

MATERIALS and METHODS

A single case report in acute inpatient rehabilitation.

Case Presentation

A 39-year-old right-handed man with a medical history of

hypertension, type 2 diabetes mellitus, hyperlipidemia presented with a sudden onset of right lower extremity (RLE) weakness and hypertensive emergency (blood pressure, 204/130 mmHg). The patient had not taken any medications at home and denied any history of illicit drug use. His medical history did not include any neurological disease or head trauma, and his family history was negative for intracranial hemorrhage. The results of his urine toxicology test were negative. The head imaging studies revealed acute intraparenchymal cerebral hemorrhage in the medial left parietal and frontal lobes. The patient started on a nicardipine drip infusion for blood pressure control and later transitioned to oral medications with a goal of the systolic blood pressure 140 mmHg. His blood glucose was controlled with diet and insulin. He temporarily received levetiracetam for seizure prophylaxis. Follow-up head imaging studies did not show significant interval changes. On post-stroke day 15, he was transferred to our facility for acute inpatient rehabilitation.

On initial physical examination, the patient appeared well-built and nourished. He was afebrile, and his blood pressure and heart rate were 116/72 mmHg and 73 beats/min, respectively, with a normal respiratory rate and oxygen saturation on room air. His general examination findings were unremarkable. On neurological examination, he was alert and fully oriented. The motor strength showed normal in both upper extremities (5/5 in the Medical Research Council); however, RLE demonstrated flexion-dominant weakness with foot drop. The finger-nose test showed dysmetria and oscillation in the bilateral upper extremities (BUE). Deep tendon reflexes were scored 1+ throughout, except for 4+ in the right Achilles tendon reflex due to the ankle clonus. Sensory examination was notable for a decreased position sense of the fingers and toes in the right upper extremity (RUE) and RLE. The patient showed no pseudoathetotic movement in the pronator drift examination, suggesting mildly impaired proprioception. The sensation to crude touch and cool temperature was intact. His function was evaluated with Functional Independence Measure (FIM) [12] which was repeated at discharge (Table 1,2).

SELF-CARE		Admission	Discharge	FIM gains
A.	Eating	5	6	
B.	Grooming	5	6	
C.	Bathing	1	5	
D.	Upper extremity dressing	5	6	
E.	Lower extremity dressing	2	6	
F.	Toileting	1	6	
SPHINCTOER CONTROL				
G.	Bladder	2	5	
H.	Bowel	1	5	
TRANSFERS				
I.	Bed <--> chair transfer	3	6	
J.	Toilet transfer	1	6	
K.	Tub shower transfer	1	5	
LOCOMOTION				
L.	Locomotion Walk	1	5	
M.	Locomotor Stair	1	5	
COMMUNICATION				
N.	Comprehension	6	7	
O.	Expression	7	7	
SOCIAL COGNITION				
P.	Social interaction	7	7	
Q.	Problem solving	6	7	

R.	Memory	6	7	
Motor Functional Measures		29	72	43
Cognitive Functional Measures		32	35	3
Total Scores of Admission		61	107	46

Table 1: Functional Independence Measures (FIM).

No Helper	
7	Complete Independence (Timely, Safely)
6	Modified Independence (Device)
Helper - Modified Dependence	
5	Supervision (Subject = 100%)
4	Minimal Assistance (Subject =75% or more)
3	Moderate Assistance (Subject= 50% or more)
Helper - Complete Dependence	
2	Maximal Assistance (Subject = 25% or more)
1	Total Assistance or not Testable (Subject less than 25%)
Leave no blanks. Enter 1 if not testable due to risk	

Table 2: FIM Levels.

Investigations

The patient presented with clumsiness in tasks requiring fine motor control in both hands, such as buttoning the shirt (Video), flipping coins, and picking up a small object and hanging it on the wall. Regarding further evaluations, his Edinburgh Handedness Inventory score was 90% [5] the two-point discrimination on the tip of the index finger was 3 mm bilaterally (within the normal limits), and graphesthesia was impaired only on the right palm. The patient could pantomime in intransitive (e.g., saluting and waving the hand) and transitive movements (e.g., combing his hair, hitting a nail with a hammer, using a toothbrush) on verbal commands, and could mimic hand postures and movements of the examiner in meaningful (transitive and intransitive) and non-meaningful movements. He did not have any problem with sequencing tasks for self-care (no ideational apraxia).

In the stroke risk factor assessment, a transthoracic echocardiogram showed moderately concentric left ventricular hypertrophy with a low normal ejection fraction, and moderate diastolic dysfunction, consistent with his history of hypertension.

In the imaging studies from the acute hospital, the head CT obtained on post-stroke day 1 showed acute parenchymal hematoma involving the medial aspect of the left postcentral to the subcortical white matter measuring 4.4 × 2.6 × 2.2 cm with minimal edema and no significant mass effect, extending rostrally to the subcortical white matter of the medial frontal lobe (Figure 1). CT angiography of the head did not show arteriovenous malformation (AVM), thrombus, vessel occlusion, stenosis, or intracranial aneurysm. CT angiography of the neck did not show any stenosis, occlusion, or aneurysm in the great vessels of the neck. In a brain MRI performed on post-stroke day 2, the hemorrhage was located in the parasagittal left parietal lobe in the postcentral gyrus, which extended downward and rostrally, involving the left subcortical precentral and premotor cortex; also involving the pericetral cingulate gyrus; its size was comparable to that on CT with the addition of surrounding vasogenic edema.

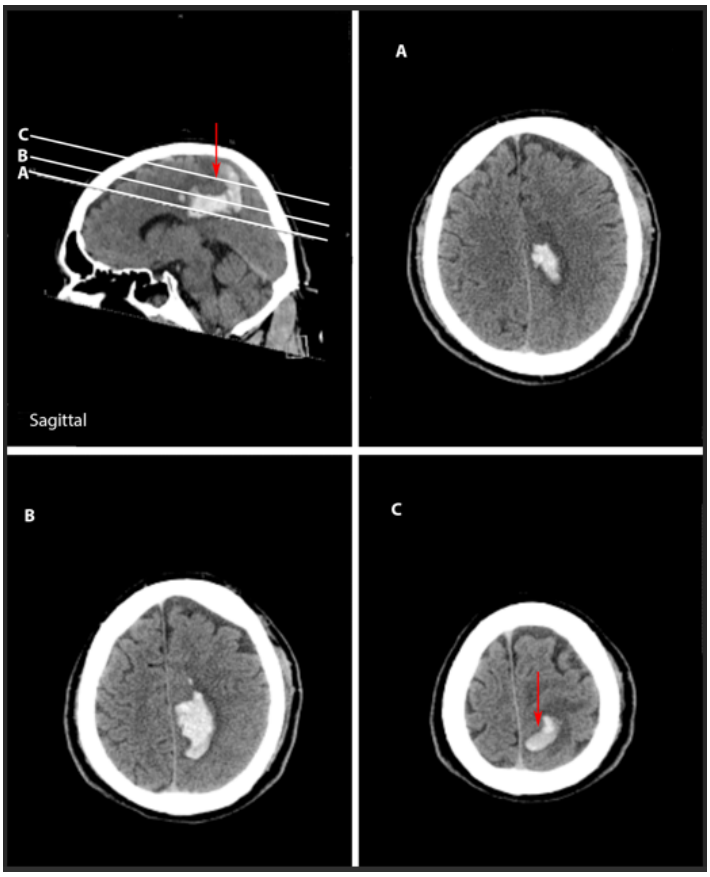

Figure 1: Head CT showing acute intracerebral hemorrhage on post-stroke day 1. Sag. A midline sagittal image showing the medial aspect of the left hemisphere. The hematoma is spreading down from the left postcentral gyrus to the subcortical white matter anteriorly, involving the left medial subcortical frontal lobe. The lines A, B, C represent the cut level of the axial image. A. An axial image of line A with hematoma in the subcortical white matter of the left medial frontal lobe. B. An axial image between levels A and C, with the hematoma spreading in the subcortical white matter of the left medial post-central gyrus anteriorly to the medial frontal lobe. C. An axial image showing the hematoma in the left post-central gyrus. The arrow is pointing at the left central sulcus.

Differential Diagnosis

While the patient's awkward, slow, and clumsy hand movements in the RUE could indicate sensory ataxia, he had similar symptoms in the left upper extremity as well, which was shown to be intact in the sensory examination. Additionally, the clumsiness did not improve with visual guidance, which is observed in sensory ataxia. Two-point discrimination was within the normal limits on the fingers of both hands. Based on the findings of physical examinations, the symptoms presented in both upper extremities were ascribed to limb-kinetic apraxia with a lesion in the left postcentral gyrus. Ideomotor and ideational apraxias were ruled out because the patient was able to perform pantomimes, mimic the movements of the examiner, and conceptualize tasks with the upper extremities [1,4,6].

Regarding the etiology of his intracerebral hemorrhage, due to his young age of onset and the atypical localization of the hemorrhage, the differential diagnosis included AVM, brain tumor, and vasculitis. However, head CT and brain MRI findings did not support these diagnoses.

Treatment

The patient received conventional inpatient rehabilitation therapy with physical, occupational, and speech therapies for 3 hours/day, 6 days/week, including repetitively practicing tasks in basic ADLs using both hands. Initially, he required total assistance for ambulation and toileting, maximal assistance for lower body dressing, moderate assistance for transfers, and stand-by assistance for other basic self-care tasks (Table 1).

His co-morbidities of hypertension and type 2 diabetes mellitus were managed medically, and his insulin was switched to metformin. His blood pressure was well controlled with amlodipine and lisinopril and stayed within 140/90 mmHg.

Outcome and Follow-Up

Twenty-three days later, at discharge from the inpatient rehabilitation service, the patient achieved modified independent, except for bathing and tub transfer with supervision in basic ADLs. He achieved modified independence for walking on even surfaces with an ankle foot orthosis (AFO) on the RLE and a crutch, but still required supervision for walking on uneven surfaces and ambulating stairs. At the subsequent clinic visit 6 months later, he was ambulating at a modified independent level utilizing only the AFO. He only showed weakness in the right foot drop on examination and maintained and further improved to independence in basic ADLs.

The patient underwent repeated brain MRI examinations with and without contrast 5 months after the onset, which confirmed the absence of a secondary intracerebral hemorrhage and the stable hematoma in the left medial postcentral gyrus, involving the same extent of the anatomical structure of the brain (Figure 2).

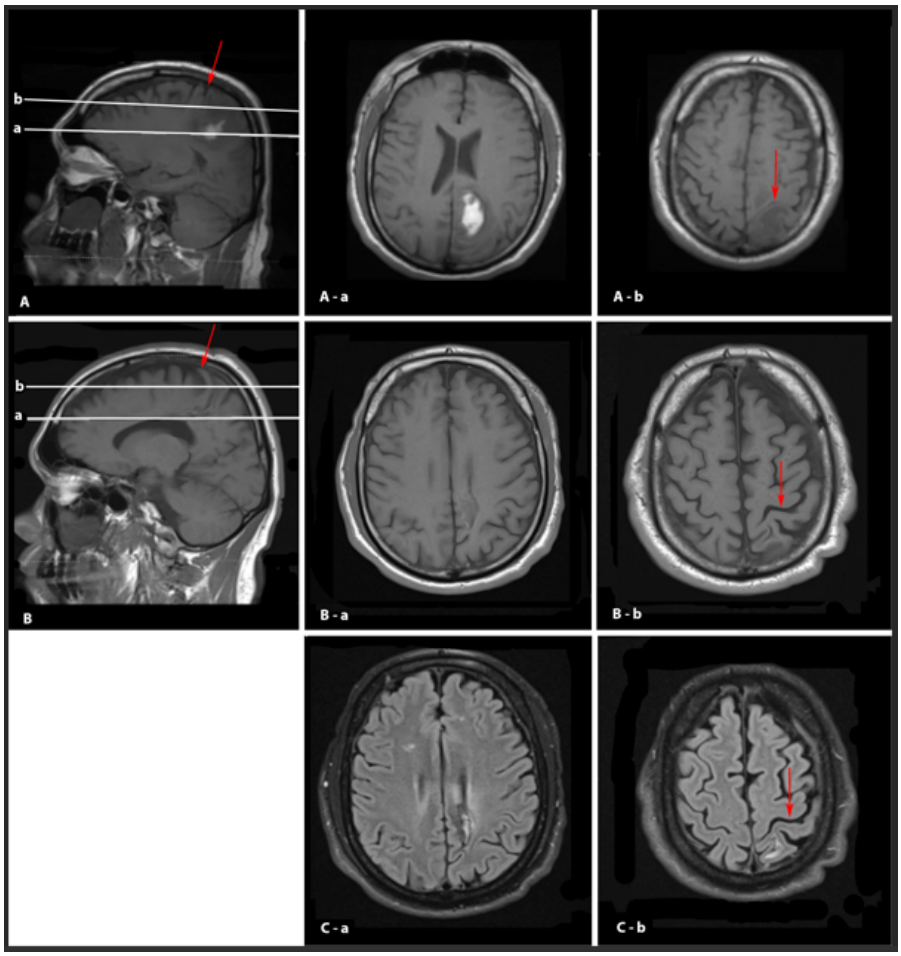

Figure 2: Brain MRI. The upper row images were obtained in the acute phase on post-stroke day 2. The middle and lower row images were obtained in the chronic phase, 5 months post-stroke. Row B demonstrates T1 sequence images while row C illustrates the same images but in FLAIR sequence. The upper row: A. T1 midline sagittal image showing the medial aspect of the left hemisphere. A-a. T1 axial image at line a of the sagittal image. The hematoma with a high-intensity signal in the subcortical white matter of the left postcentral gyrus is surrounded by edema. A-b. T1 axial image at line b of the sagittal image. Hematoma with mass effect on the left central sulcus. The middle row. B. T1 midline sagittal image showing the medial aspect of the left hemisphere. Hematoma receding and edema resolved with parenchymal atrophy in the involved area. B-a. A T1 axial image at the level of line a of the sagittal image, involving the same anatomical structure of the injured area that was occupied by the acute hematoma. B-b. A T1 axial image at the level of line b of the sagittal image, involving the same anatomical structure of the injured area that was previously occupied by acute hematoma. C-a. A FLAIR axial image at the level of line a of the sagittal image B, involving the same anatomical structure of the injured area that was occupied by the acute hematoma.C-b. A FLAIR axial image at the level of line b of the sagittal image B, involving the same anatomical structure of the injured area that was previously occupied by acute hematoma. The red arrow indicates the left central sulcus.

Discussion

This case involved a 39-year-old right-handed man with cerebral hemorrhage in the left postcentral gyrus, who presented with bilateral limb-kinetic apraxia. The patient responded well to acute inpatient rehabilitation interventions and was able to maintain his functional gains at the 6-month follow-up. In the follow-up imaging study performed 5 months after the stroke onset, the lesion involving the parenchymal structure was stable. The hemorrhage was most likely caused by hypertension.

Limb apraxia is defined as a loss of learned and skilled movements derived from higher-order motor deficits with preserved elemental motor and sensory innervation [2]. Liepmann classified it into ideomotor, ideational, and limb-kinetic apraxias. The most commonly described form of post-stroke apraxia is ideomotor apraxia (dysfunction of a skilled movement production system), followed by ideational apraxia (dysfunction of a skilled movement conceptual system) [2,3].

Limb-kinetic apraxia consists of "impaired, coarse, mutilate execution of simple movements," according to Liepmann, and presents as a unilateral, awkward execution of correctly planned movements. [3] Limb-kinetic apraxia has been largely neglected, and some studies have even questioned its existence [3,4]. Recently, limb-kinetic apraxia was reported in neurodegenerative diseases, such as Parkinson's disease. The pathological mechanism underlying this form of apraxia remains unestablished [7]. Since the definition of apraxia excludes elementary motor and sensory impairment, stroke patients with weakness, ataxia, and/or sensory impairment are basically excluded when the apraxia appears on the same stroke-affected limb. Some may consider limb-kinetic apraxia merely as sensory ataxia or attribute the clumsiness from limb-kinetic apraxia to a part of hemiparetic weakness itself. In typical post-stroke hemiparetic weakness, the patient may show increased or decreased muscle tone, inability to perform isolated joint movements (synergy), hypokinesia, and bradykinesia, which can make it difficult to differentiate hemiparesis from limb-kinetic apraxia. This case, however, did not show weakness, hypokinesia or bradykinesia in either upper extremity. The patient showed impaired results in the finger-to-nose test bilaterally as well as clumsiness and difficulty in performing functional tasks in both upper extremities. He showed mildly impaired proprioception in the fingers of his right hand. In sensory ataxia, the performance is mostly corrected with visual guidance; however, he was unable to correct his performance with it. Additionally, his symptoms presented bilaterally, making sensory ataxia less likely. He did not have any lesions in the cerebellum, brainstem, or basal ganglia, which would exclude cerebellar ataxia or Parkinsonism. However, the hematoma extended into the left medial frontal subcortical white matter, which may have affected the fine motor control of both hands.

Liepmann attributed limb-kinetic apraxia to brain injury in the contralateral sensory-motor cortices of the affected upper extremity [3,4]. In our case, the main stroke lesion was located in the left postcentral gyrus but presented as bilateral limb-kinetic apraxia. Heilman et al. reported left hemisphere dominancy in limb-kinetic apraxia, supporting the bilateral manifestation of limb-kinetic apraxia with a left hemisphere lesion in the presented case [8].

There is no standardized evaluation method or rehabilitation therapy for apraxia. Patients with apraxia improve with repetitive training in a specific task, but the beneficial effect is not generalized. Moreover, the training effect is not retained after the training ends [9]. The effectiveness of rehabilitation therapy for apraxia remains unknown. [7,9,10] In our case, the improvement in function was retained at the 6-month follow-up after completion of the inpatient therapy program, which may suggest that limb-kinetic apraxia has a better prognosis than the other types of apraxia. Jang and Seo reported rapidly recovered post-stroke left hemiparesis with intensive rehabilitation therapy, ascribing the impaired motor control to limb-kinetic apraxia instead of hemiparetic weakness, which is ascribed to the injury of the corticofugal tract, not to the corticopyramidal tract [11,12].

Conclusion

In conclusion, Apraxia is a common and disabling symptom occurring after a stroke attack; however, no standardized evaluation methods or evidence-based management has been established. Recognition of limb-kinetic apraxia and differentiating it from hemiparesis and other types of apraxia is important to advance the evaluation and evidence-based management of post-stroke apraxia.

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