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A 78-Year-Old Woman with Sudden Onset of Left-Sided Hemiballismus

Authors' Contribution:

Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
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Patient: Female, 78-year-old
Final Diagnosis: Acute ischemic stroke • hemiballismus
Symptoms: Involuntary movements of the left upper and lower extremities
Clinical Procedure: —
Specialty: Neurology

Objective: Rare coexistence of disease or pathology

Background: Hemiballismus is the most severe form of chorea and is a hyperkinetic disorder characterized by involuntary, high-amplitude movements of the ipsilateral arm and leg, due to lesions of the contralateral side of the central nervous system. Ischemic or hemorrhagic strokes and nonketotic hyperglycemia are predominant etiologies of hemiballismus. Case reports highlighting hemiballismus associated with temporal and parietal lobe infarcts have been published, although research of frontal lobe involvement is limited.

Case Report: A 78-year-old woman presented to the Emergency Department with sudden-onset left-sided hemiballismus. On examination, she was alert, oriented to self and time, and able to follow commands. Her neurologic examination was notable for left-sided hemiballismus, described by the provider as periodic, uncontrolled, and involving a “flinging” motion of the left upper and lower extremities, sparing the face. She was treated with benzodiazepines in the Emergency Department and administered intravenous levetiracetam. Computed tomography of the head without contrast revealed an old left basal ganglia lacunar infarct. The patient was then admitted to the inpatient service, where magnetic resonance imaging of the brain revealed an acute punctate left superior frontal gyrus cortical infarct. Outpatient electroencephalogram revealed right anterior hemisphere dysfunction.

Conclusions: We describe a patient with left-sided sudden onset hemiballismus with an acute infarct of the ipsilateral superior frontal gyrus. This case highlights that brain lesions separate from the basal ganglia can induce hemiballismus, particularly within the frontal lobe, which warrants further research into precentral sulcus functioning and its role in modulating motor activity.

Keywords: Cerebral Infarction • Dyskinesias • Prefrontal Cortex**Full-text PDF:** <https://www.amjcaserep.com/abstract/index/idArt/941840>

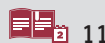
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Background

Hemiballismus is an involuntary, high amplitude hyperkinetic disorder involving the ipsilateral arm and leg, due to lesions in the contralateral aspect of the central nervous system [1]. Ischemic or hemorrhagic strokes and nonketotic hyperglycemia are predominant etiologies of hemiballismus [2]. Other etiologies include neoplasm, tuberculoma, toxoplasmosis, toxic exposure, paraneoplastic syndrome, neuroinflammatory diseases, such as multiple sclerosis, vasculitides, such as systemic lupus erythematosus, and neurodegenerative conditions, including Huntington disease [3].

Hemiballismus was first described and induced by Whittier and Mettler in 1949, when the subthalamic nucleus in monkeys was ablated [4]. It is typically associated with lesions of the subthalamic nucleus [5].

There are a handful of reports that describe lesions outside the subthalamic nucleus. Karmacharya et al described a case of a 61-year-old man with sudden-onset hemiballismus of the right upper extremity secondary to an acute posterior left parietal lobe infarction [6]. Chung et al concluded that post-stroke hemichorea was most often caused by lesions within the lenticular region, followed by subthalamic and cortical lesions [7].

In a recent case report published in 2021, Wei et al highlight a 64-year-old woman with acute left-sided hemichorea in which magnetic resonance imaging (MRI) revealed an acute left frontal lobe infarction in addition to an old right frontal lobe lesion [8].

However, evidence remains limited to suggest hemiballismus is also associated with frontal lobe lesions.

This report is of a 78-year-old woman with sudden onset of left-sided hemiballismus.

Case Report

A 78-year-old woman with a history of hypertension, prediabetes, and restless leg syndrome presented to the Emergency Department (ED) with acute-onset left-sided hemiballismus. This was a first-time occurrence, and she denied toxic ingestion or recent medication changes. The patient had no family history of Huntington disease or other movement disorders. A 10-point review of systems was otherwise negative.

On examination, the patient was alert, oriented, and able to follow commands. The Glasgow coma scale score was 15. Her neurologic examination was notable for left-sided hemiballismus, described by the provider as periodic, uncontrolled, and involving a “flinging” motion of the left upper and lower extremities, sparing the face. There was no facial droop, and cranial nerves II-XII were otherwise grossly intact. Sensory response to light touch was preserved in the bilateral upper and lower extremities. Finger-to-nose and heel-to-shin testing were within normal limits on the right. The left side could not be reliably tested, as underlying movements disrupted voluntary testing, although cerebellar function appeared grossly preserved.

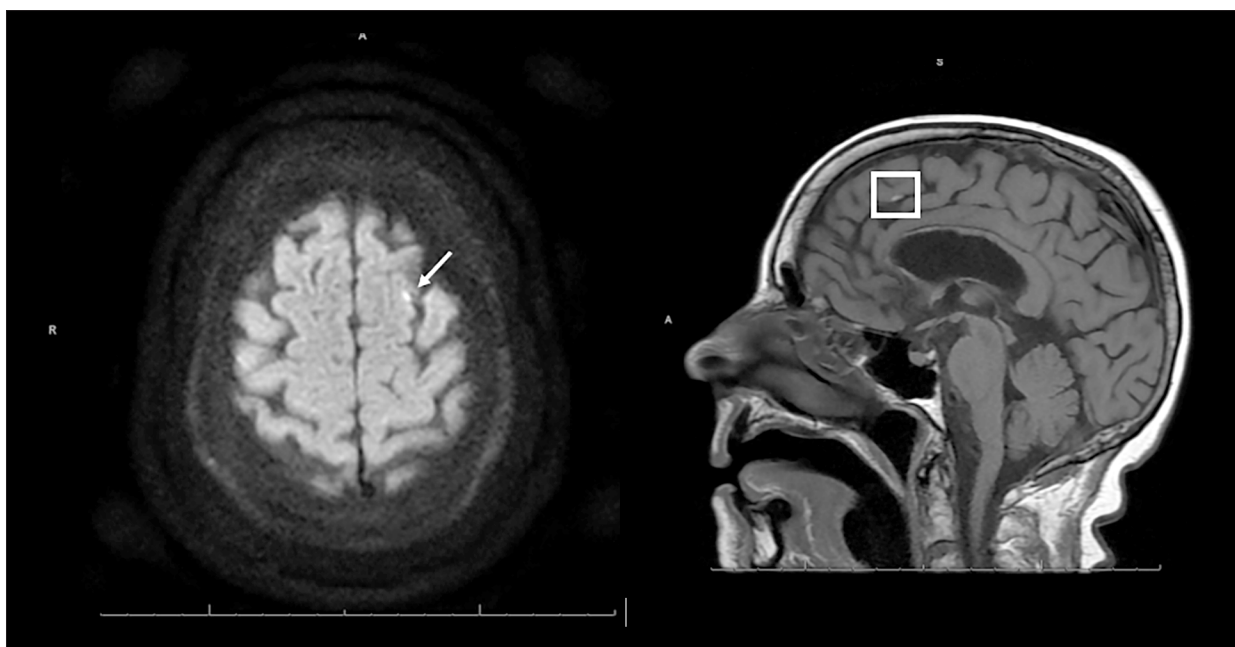


Figure 1. Magnetic resonance imaging of the brain via diffusion-weighted imaging on the axial view (top image) and fluid-attenuated inversion recovery via the sagittal view (bottom image) revealed a punctate acute left superior frontal gyrus cortical infarct.



Figure 2. Electroencephalogram revealed a focal, intermittent, slowing of the right anterior hemisphere, involving the frontocentral and frontotemporal regions, without clear seizures or epileptiform discharges (highlighted via blue arrows).

The patient's uncontrolled movements were controlled with sequential doses of intravenous lorazepam (2 mg, 2 mg, 2 mg, 1 mg) totaling 7 mg and 1 g levetiracetam. The laboratory studies in the ED were largely unremarkable. Her creatine kinase levels were elevated, at 476 U/L. Computed tomography (CT) of the head without contrast revealed an old 1.5-cm left basal ganglia lacunar infarct. An initial attempt at MRI failed due to ongoing movement. A second attempt at MRI was taken with the patient under general anesthesia and accompanied by an anesthesiologist. MRI of the brain with and without contrast revealed a punctate acute left superior frontal gyrus cortical infarct (Figure 1).

The patient was admitted inpatient for further monitoring. Further workup revealed a positive urine culture of greater than 100 000 col/mL *Escherichia coli* and *Proteus mirabilis*. Hemoglobin A1C on day 1 of admission was 5.3%. The fasting glucose level within a year prior to presentation was 99 mg/dL (reference range, 60-99 mg/dL). Inpatient laboratory test results revealed a vitamin B1 level of 279 nmol/L (reference range, 70-180 nmol/L), positive serum antinuclear antibody test, elevation of Sjögren B antibody of 3.3, and normal thyroid stimulating hormone, copper, and ceruloplasmin levels. Human immunodeficiency virus and syphilis panels were also negative.

No large vessel occlusions were seen on CT angiogram of the head and neck. An MRI of the cervical spine with and without contrast revealed no clinical signs of infection or neoplasm.

The patient was discharged on hospital day 5, with an improvement in her symptoms. She was prescribed levetiracetam in

the outpatient setting indefinitely and reported no recurrence of hemiballismus after discharge.

Outpatient laboratory test results revealed negative autoimmune and paraneoplastic encephalitis panels. An outpatient electroencephalogram revealed right anterior hemisphere dysfunction (Figure 2).

Discussion

This case highlights that brain lesions separate from the subthalamic nucleus and basal ganglia can induce hemiballismus, particularly within the frontal lobe.

The patient highlighted in this case report was a 78-year-old woman who presented with acute-onset left-sided hemiballismus involving the upper and lower extremities. MRI did reveal an acute infarct of the left superior frontal gyrus, although this did not necessarily explain her symptomatology. There was no evidence to suggest an infectious or toxic-metabolic etiology.

Research has shown that the dominant (left) superior frontal gyrus is involved in working memory and spatial processing [9]. Additionally, the activation of the non-dominant (right) superior frontal gyrus plays a key role in modulating inhibitory control and motor urgency [10].

In our patient, it would be expected that a lesion of the left superior frontal gyrus would cause contralateral deficits involving the right extremities, although there is some evidence to

suggest ipsilateral frontal lobe involvement in hemiballismus, as highlighted by Wei et al [8]. Nevertheless, more research is needed on the functioning of the precentral sulci, as these remain a topic of investigation.

The majority of hemiballismus cases have a good prognosis and resolve spontaneously, as seen in our patient. Treatment of hemiballismus involves dopamine depletion (VMAT2 inhibitors) and dopamine antagonists. Severe cases refractory to this therapy can be treated with deep brain stimulation or pallidotomy [11].

References:

1. Grandas F. Hemiballismus. Handbook of Clinical Neurology. Elsevier; 2011
2. Hawley JS, Weiner WJ. Hemiballismus: current concepts and review. Parkinsonism Relat Disord. 2012;18(2):125-29
3. Rocha Cabrero F, De Jesus O. Hemiballismus [Updated 2023 Aug 23]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2023. Available from: <https://www.ncbi.nlm.nih.gov/books/NBK559127/>
4. Whittier JR, Mettler FA. Studies on the subthalamus of the rhesus monkey; Hyperkinesia and other physiologic effects of subthalamic lesions; With special reference to the subthalamic nucleus of Luys. J Comp Neurol. 1949;90(3):319-72
5. Postuma B, Lang AE. Hemiballismus: Revisiting a classic disorder. Lancet Neurol. 2003;2(11):661-68
6. Karmacharya P, Shrestha P, Adhikari J, et al. Cortical hemiballismus: A case of hemiballismus associated with parietal lobe infarct. N Am J Med Sci. 2015;7(12):572-74
7. Chung SJ, Im JH, Lee MC, Kim JS. Hemichorea after stroke: Clinical-radiological correlation. J Neurol. 2004;251(6):725-29
8. Wei J, Zhang Y. Hemichorea in a patient with ipsilateral cortical infarction: A case report. BMC Neurol 2021;21(1):420
9. du Boisgueheneuc F, Levy R, Volle E, et al. Functions of the left superior frontal gyrus in humans: A lesion study. Brain. 2006;129(Pt 12):3315-28
10. Hu S, Ide JS, Zhang S, Li CR. The right superior frontal gyrus and individual variation in proactive control of impulsive response. J Neurosci. 2016;36(50):12688-96
11. Suchowersky O, Shetty A. Treatment of hemiballismus. Therapy of Movement Disorders. 2019;255-57

Conclusions

Hemiballismus has a multitude of etiologies, although it commonly involves cerebrovascular infarcts, particularly lesions of the subthalamic nucleus, or nonketotic hyperosmolar hyperglycemia [11]. This case report highlights a patient with hemiballismus with a known lesion of the ipsilateral superior frontal gyrus, as confirmed by advanced imaging. This warrants further research into the functioning of the precentral sulcus and its role in modulating motor activity.

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