

Cortical Hemiballism: A Case of Hemiballismus Associated with Parietal Lobe Infarct

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Abstract

Context: Hemiballismus is characterized by involuntary, irregular, large amplitude, and violent flinging movements of limbs. Stroke (middle and posterior cerebral artery) remains the most common etiology with 2/3 being lacunar. Lesions outside the substantia nigra (STN) can cause hemiballismus, and only a minority by STN lesions, unlike the classical belief. Compared to those arising from STN, cortical hemiballismus is usually less severe with a good prognosis. **Case Report:** A 61-year-old man presented with sudden onset involuntary flinging movements of his right upper extremity accompanied by numbness and tingling. Past medical history was significant for stroke 2 years back with no residual deficits. Vitals signs were blood pressure of 165/84 mm Hg, and heart rate - 82 beats/min. Irregular, arrhythmic, jerky flinging movement, and decreased sensation to light touch in right upper extremity was noted. Magnetic resonance imaging of the brain revealed acute posterior left parietal lobe infarction. He was treated with aspirin and atorvastatin. Thrombolytic therapy was offered but declined. The movements resolved spontaneously over the next 2 days. No further episodes occurred at 3-month follow-up. **Conclusion:** Lesions affecting various areas outside the STN can cause hemiballismus and usually carries a good prognosis with spontaneous resolution. Acute thrombolytic therapy may be considered on an individual basis. Treatment with antipsychotics can be useful for severe and recurring symptoms.

Keywords: Antipsychotics, hemiballismus, hemiballism, infarction, parietal lobe, stroke, subthalamic

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Introduction

Hemiballismus is a rare movement disorder associated with involuntary, large-amplitude, flinging movements of the limbs.^[1-5] While it has been classically described with lesions of the subthalamic nucleus (STN), more and more lesions outside the STN have been reported recently.^[1,3,4] Compared to those arising from STN, cortical hemiballismus is usually less severe with a good long-term prognosis.^[1] It is important to recognize this rare disorder and investigate for possible treatable etiologies. Here, we present a case of hemiballismus

resulting from an acute parietal lobe infarction which was self-limiting.

Case Presentation

A 61-year-old man presented to the Emergency Department with sudden onset of involuntary flinging movements of his right upper extremity. The patient was working out at home when he noticed

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some numbness and tingling in his right arm which slowly progressed to violent shaking. He did not have any vertigo, dysarthria, diplopia, or weakness. Past medical history was significant for stroke 2 years back with no residual deficits. Vitals signs at the time of presentation were blood pressure of 165/84 mm of Hg, heart rate 82 beats/min, respiratory rate 19/min, and normal oxygen saturation on room air. Patient was noted to have irregular, arrhythmic jerky, and flinging movement of the right arm. Neurological examination revealed decreased sensation to light touch in the right upper extremity. Rest of the examination was unremarkable. Laboratory findings, including electrolytes and blood sugar were within normal limits. Computed tomography scan of the head revealed a small old left occipital infarct [Figure 1]. Subsequent magnetic resonance imaging of the brain revealed acute posterior left parietal lobe infarction [Figure 2]. Magnetic resonance angiography revealed mild stenosis of the right P1 segment. Patient was treated with one dose of aspirin 325 mg and continued on aspirin 81 mg and atorvastatin 40 mg. He was offered therapy with tissue plasminogen activator but declined therapy due to risks involved. His involuntary movements were thought to be consistent with hemiballismus secondary to a cortical stroke. The movements resolved spontaneously over the next 2 days. Patient was offered a loop monitor placement to look for any arrhythmias but declined. He was empirically started on warfarin and discharged home. He had not had any further episodes at 3-month follow-up.

Discussion

Hemiballismus is a rare movement disorder characterized by involuntary, irregular, large amplitude, violent flinging movements of the limbs. As the name itself suggests, it involves one side of the body, usually the

arm, leg, proximal in most cases, with facial involvement in approximately half of the cases.^[1-5] Symptoms occur with activity, decrease with rest and disappear with sleep.^[2] Chorea and hemiballismus may present as a spectrum of disease with hemiballismus often evolving into hemichorea, so the term hemichorea-hemiballismus is sometimes used.^[1]

It has been classically reported with lesions of the subthalamic nucleus (STN) with only a few reports describing lesions outside the STN. Lesions of the STN cause lower than normal activation of the internal segment of globus pallidus, leading to disinhibition of the thalamus, resulting in excessive movement manifested as hemiballismus.^[1] However, the pathophysiology underlying cortical hemiballismus remain unclear. Disorganization of the sensorimotor integration in frontoparietal lobes with decreased excitatory output to STN has been postulated.^[3]

Stroke remains the most common etiology giving rise to hemiballismus; common vascular territories involved being middle and posterior cerebral artery territory with 2/3 of the cases involving small vessels with lacunar stroke.^[1,6] The incidence in acute stroke ranges between 0.4% and 0.54% with a prevalence of 1%.^[6] Onset is variable with an average of 4.3 days poststroke and about 12.5% of the patients developing it within 24 h.^[7] Other causes include traumatic brain injury (e.g., subdural hematoma), nonketotic hyperglycemic coma, amyotrophic lateral sclerosis, tuberculomas, vascular neoplasms, systemic lupus erythematosus, systemic vasculitis, demyelinating plaques, and complications from HIV infection such as toxoplasmosis.^[1]

Management involves ruling out reversible causes such as infection, hyperglycemia and neoplastic lesions.

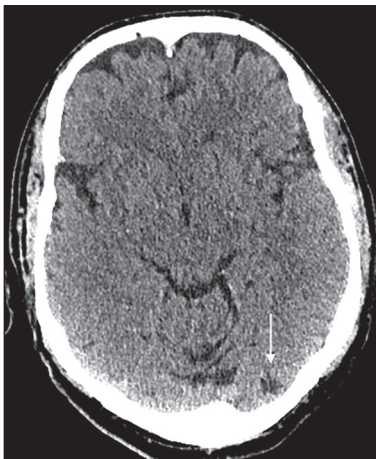


Figure 1: Computed tomography scan of the head revealing a small old left occipital infarct (white arrow)

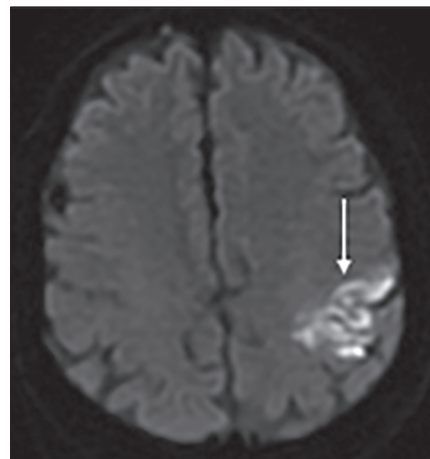


Figure 2: Diffusion-weighted sequences in magnetic resonance imaging of the brain showing restricted diffusion (white arrow) in posterior left parietal lobe consistent with acute parietal lobe infarction

Prevention of injury with padding and restraints to prevent injury is important in acute stages while supportive care with good hydration is required to prevent complications such as dehydration and rhabdomyolysis. It is unclear whether hemiballismus with underlying acute stroke by itself is an indication for thrombolytic therapy. Although there have been few reports where it was used with good outcomes,^[8,9] it is important to rule out stroke mimics and consider individual patient factors. Pharmacologic therapy with dopaminergic blockade can be considered for severe or recurring symptoms. Typical neuroleptics (haloperidol, perphenazine) or atypical antipsychotics (risperidone, clozapine) may be started at a low dose and titrated as tolerated. Catecholamine-depleting agents such as reserpine and tetrabenazine may be considered when long-term therapy is required as they carry a lower risk of tardive dyskinesia. For refractory cases, stereotactic neurosurgical procedures may be considered in good surgical candidates. It is, however, important to note that in many cases, symptoms tend to subside allowing withdrawal of drugs.^[1]

Conclusion

Lesions affecting various areas outside the STN can cause hemiballism, and only a minority is caused by STN lesions unlike the classical belief. It usually carries a good prognosis with majority having spontaneous resolution. Acute thrombolytic therapy may be considered on an individual basis. Treatment with antipsychotics can be useful for severe and recurring symptoms.

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Conflicts of interest

There are no conflicts of interest.

References

1. Postuma RB, Lang AE. Hemiballism: Revisiting a classic disorder. *Lancet Neurol* 2003;2:661-8.
2. Hao M, Qin X, Gao H. A case of Hemichorea-Hemiballism Induced by Acute Infarction of Bilateral Corona Radiata and Cortex. *Cell Biochem Biophys*. February 2015. doi:10.1007/s12013-015-0608-6.
3. Wu MC, Yen RF, Lin CH, Wu RM. Hemiballism in a patient with parietal lobe infarction. *Neurology* 2013;80:e22.
4. Srivastava T, Singh S, Goyal V, Shukla G, Behari M. Hemichorea-hemiballism associated with frontoparietal bleed. *J Neurol* 2006;253:653-4.
5. Daoud A, Chand P, Cruz-Flores S. Segmental ballism due to acute ischemic lesions of the caudate nucleus and parietal lobe: Case report and review of the literature. *Clin Neurol Neurosurg* 2011;113:898-900.
6. Ghika-Schmid F, Ghika J, Regli F, Bogousslavsky J. Hyperkinetic movement disorders during and after acute stroke: The Lausanne Stroke Registry. *J Neurol Sci* 1997;146: 109-16.
7. Alarcón F, Zijlmans JC, Dueñas G, Cevallos N. Post-stroke movement disorders: Report of 56 patients. *J Neurol Neurosurg Psychiatry* 2004;75:1568-74.
8. McCollum D, Silvers S, Dawson SB, Barrett KM. Resolution of acute onset hemichorea-hemiballismus after treatment with intravenous tissue plasminogen activator. *Neurohospitalist* 2013;3:131-4.
9. Zidverc-Trajkovic J, Jovanovic DR, Marjanovic I, Radojicic A, Beslac-Bumbaširevic L. Successful intravenous thrombolysis in a stroke patient with hemiballism. *Neurologist* 2011;17:205-7.