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# Stroke Associated Hemichorea-hemiballismus: A Case Report

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**Abstract**

The term "movement disorders" encompasses nervous system abnormalities characterized by excess (hyperkinesia) or lack (hypokinesia) of voluntary or automatic movements, often associated with Basal Ganglia dysfunction. Primary and secondary movement disorders differ in their manifestations, with strokes leading to various hyperkinetic and hypokinetic disorders. This case report explores the frequency of post-stroke movement disorders and discusses the prognosis, emphasizing the importance of early recognition for targeted therapies. Neuroimaging reveals the impact on the basal ganglia and thalamus. The discussion delves into the underlying mechanisms, highlighting the complex interplay between cortical and subcortical structures in post-stroke movement disorders. This case report advocates for a comprehensive approach to diagnosis and intervention, contributing to the understanding and management of stroke-associated movement disorders

**Keywords**: NIL

## Introduction

The term "movement disorders" refers to nervous system abnormalities characterized by either an excess (hyperkinesia) or a lack (hypokinesia) of voluntary or automatic movements that are not accompanied by weakness or stiffness. The underlying pathology in most of them is disruption of the Basal Ganglia and its connections' normal functioning. This dysfunction can result from a variety of causes, including genetic factors, neurodegenerative diseases, metabolic abnormalities, vascular events, and certain medications. The specific etiology depends on the type of movement disorder and individual patient characteristics. Primary movement disorders are characterized by abnormal movements as the primary manifestation of the disorder. Conversely, in secondary movement disorders, the abnormal movements stem from another underlying systemic or neurological disorder. Secondary movement disorders arising from strokes exhibit a diverse range of characteristics, with their natural course, prognosis, and treatment strategies differing from those seen in primary (idiopathic) movement disorders. They can manifest either acutely alongside a stroke or as delayed consequences of a stroke. Post-stroke movement disorders can be categorized into hyperkinetic disorders, characterized by excessive and abnormal involuntary movements, and hypokinetic disorders, marked by a scarcity or slowness (bradykinesia) of movement. Movement disorders reported following cerebrovascular events encompass chorea, ballism, athetosis, dystonia, tremor, myoclonus, asterixis, stereotypies, akathisia, tics, vascular parkinsonism, progressive supranuclear palsy, isolated freezing of gait, and cortico-basal syndrome. The frequencies at which various movement disorders appear after

stroke are shown in the table1. (2) The prognosis of primary and secondary movement disorders can differ significantly. Primary movement disorders, often arising from genetic or idiopathic causes, may have a more stable and predictable prognosis, with symptoms typically progressing slowly. The prognosis of post-stroke secondary movement disorders varies based on several factors, including the extent and location of the brain injury, the type of movement disorder, and the effectiveness of rehabilitation efforts. Early recognition allows healthcare professionals to implement targeted therapies that may aid in mitigating the impact of the movement disorder. In some cases, with prompt and appropriate treatment, there may be a partial or complete resolution of the symptoms.

**Table 1 (2) : The frequency of post-stroke movement disorders**

| Movement Disorder | Lausanne stroke registry (%) | Ecuador stroke registry (%) |
|---|---|---|
| **Chorea** | 38 | 36 |
| **Dystonia** | 17 | 29 |
| **Limb shaking** | 10 | |
| **Myoclonus-dystonia** | 10 | |
| **Stereotypic** | 7 | |
| **Asterixis** | 7 | |
| **Tremor** | 3 | 25 |
| **Hemi-akathisia** | 3 | |
| **Dysarthria, dyskinetic hand** | 2 | |
| **Parkinsonism** | | 10 |

Abnormal movement after stroke is most commonly associated with damage to the basal ganglia (44%) and thalamus (37%) although post-stroke movement disorder involving damage to the cortical regions such as insular or Parieto-insular region has been reported. However, the likelihood of developing abnormal movement after infarction of the deep nuclei and thalamus is three times higher than after cortical infarction. In stroke, hyperkinetic symptoms like chorea are rare—they occur in 1-4% of cases. According to Ching et al.'s study, 27 out of 5,009 patients had a 0.54% incidence of post-stroke hemichorea. According to a related study conducted by the Lausanne Stroke Registry, 38% of all aberrant movements following a stroke are caused by chorea. (3) Hyperkinetic movement disorders are rare (1%), with hemichorea-hemiballism and hemidystonia being the most prevalent kinds. Hemichorea is characterized by rapid, unilateral, involuntary flexion and extension, rotation, or crossing of any region of the body, primarily the distal parts. A severe, violent, arrhythmic, large-amplitude limb excursion from a proximal joint with a rotational feature is known as hemiballismus. These movement impairments are



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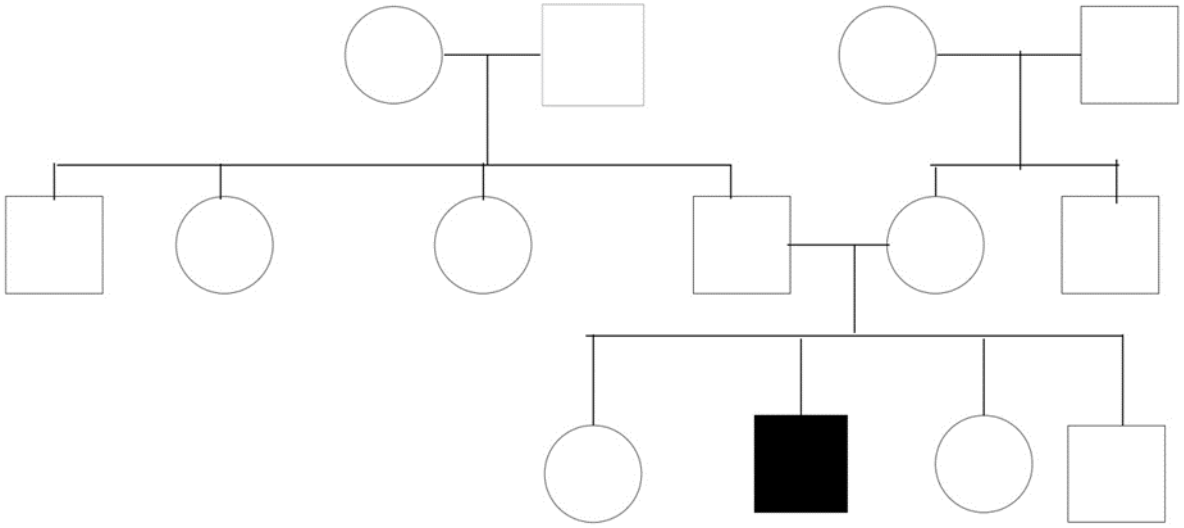
linked to strokes that affect the posterior or middle cerebral arteries. (4) The most frequent types of strokes leading to movement disorders are typically small vessel infarctions occurring in the basal ganglia and thalamus. However, movement disorders can also emerge following cardioembolism, large artery infarction, and intracerebral hemorrhage.

## Case Report

Here we report a case of a 65-year-old hypertensive male, who presented to the OPD with a sudden onset of abnormal body movements for 1 day which started while the patient was sitting in his chair. The body movements were described as dance-like, wide flinging involuntary movements, involving upper limbs more than lower limbs on the left side. The movements were more noticeable proximally than the distal end of the limbs and were progressive. These movements were apparent even at rest which led to impairment in activities of daily living (ADL) such as holding objects. However, no similar complaints were reported in other limbs. There was no impairment in memory, cognitive functioning, or loss of consciousness. The patient denied any occurrence of trauma or any episodes of dizziness, nausea, or vertigo prior to or during the onset of abnormal body movements. The patient denied regular intake of any drug or any substance of abuse. The patient was a non-smoker and non-alcoholic. No similar history of movement disorder was found in the family (Figure 1).

**Figure 1: Pedigree analysis**



On examination, the patient was fully conscious and alert with a clear orientation toward his time, place, and personal environment. There was no neck stiffness and no cranial nerve deficits could be observed. The tone was normal in both upper and lower limbs on the right side. However Left side could not be assessed because of persistent involuntary movements during examination. On superficial tendon reflexes examination, the left plantar extensor and right plantar flexor were noted. Deep tendon reflexes were within normal limits on the right side but couldn't be assessed on the right side. Rectal tone was normal and no urinary incontinence was noted. There were no sensory deficits. Cerebellar functions were normal on the right side. The patient was admitted for further evaluation to the inpatient unit. All the routine baseline investigations were sent along with a lipid profile test. A non-contrast computerized tomography scan of the brain was conducted which revealed changes of mild generalized age-related cerebral atrophy grossly, with a suspicious area of hypodensity in the right caudate region.

On further evaluation with a MRI Brain scan (Figure 2), a small, acute infarct was seen in the right caudate showing restricted diffusion. Few punctate gliosis was seen in bilateral frontal subcortical white matter. Prominent ventricular system and cortical sulci suggested diffuse cerebral atrophy. No other significant lesions were seen. To further evaluate the



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source of emboli a CT angiogram of head vessels was performed with 2D and 3D reconstruction of images upon which a mildly narrowed calibre of the right vertebral artery was visualized. Other than that no significant stenotic lesion could be visualized.



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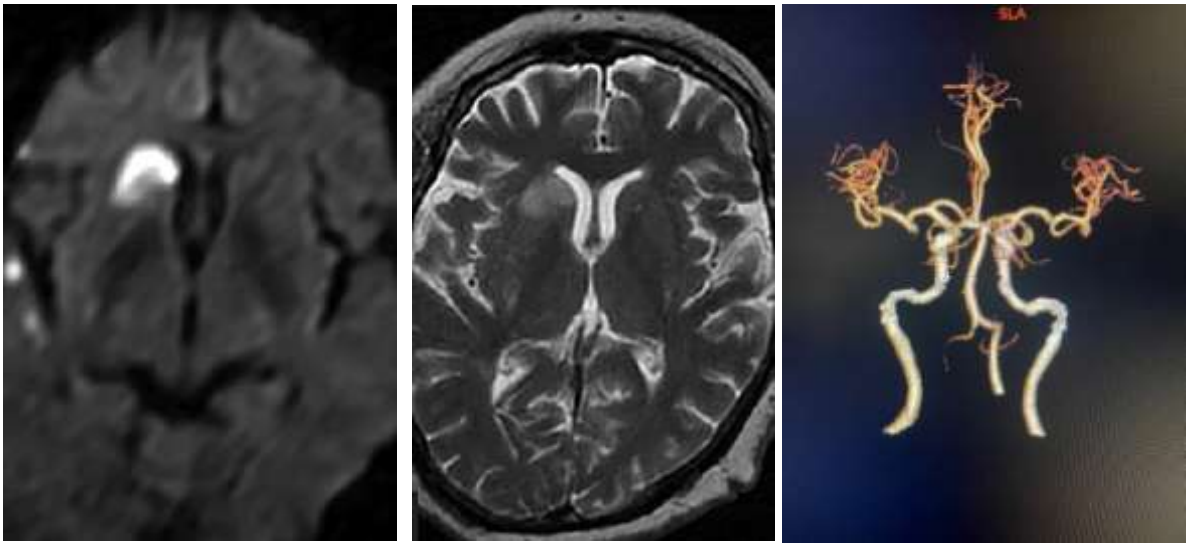
**Figure 1. MRI Brain DWI showing area of diffusion restriction in right caudate suggestive of infarct (Left), T2W sequence showing area of hyperintensity in right caudate (middle) and CT angiogram of brain vessels (right)**



ECG was suggestive of a rate of 80 bpm, sinus rhythm, normal axis with tall R waves in V5 and V6, and deep S waves in V1 and V2 suggestive of left ventricular hypertrophy (as per Sokolov Lyon criteria). A 2-D echo was performed which was suggestive of concentric left ventricular hypertrophy with normal ejection fraction. Laboratory results showed no signs of infection or inflammatory disease(refer to Table 2,3). The patient was diagnosed with acute onset hyperkinetic movement disorder hemi chorea hyper-hemi ballism, an acute cerebrovascular accident-basal ganglia infarct with risk factors of hypertension.

**Table 2 : Routine investigations**

| Investigations | Lab values | Reference range | Units |
|---|---|---|---|
| Haemoglobin | 14.8 | 13.5-17.5 | g/dl |
| TLC | 10.82 | 4.00-11.00 | 10^3/mm^3 |
| Platelets | 162 | 150-450 | 10^3/mm^3L |
| BUN | 10 | 7-18 | mg/dL |
| S. Urea | 20 | 15-17 | mg% |
| S. Creatinine | 1.2 | 0.8-1.3 | mg% |
| S. Bilirubin | 1.1/0.2 | 0.3-1.1 / 0.1-0.4 | mg% |
| AST | 64 | 5-40 | IU/L |
| ALT | 62 | 5-35 | IU/L |
| ALP | 108 | 60-150 | IU/L |
| TSP | 5.1 | 6-8 | g/dL |
| S.Albumin | 3.6 | 3.4-5.5 | g/dL |
| S. Na | 138 | 135-155 | meq/L |
| S. K | 5.2 | 3.5-5.5 | meq/L |
| S. Cl | 104 | 98-107 | meq/L |
| HIV | NR | | |
| HCV | NR | | |
| HbsAg | NR | | |
| Blood group | A + | | |



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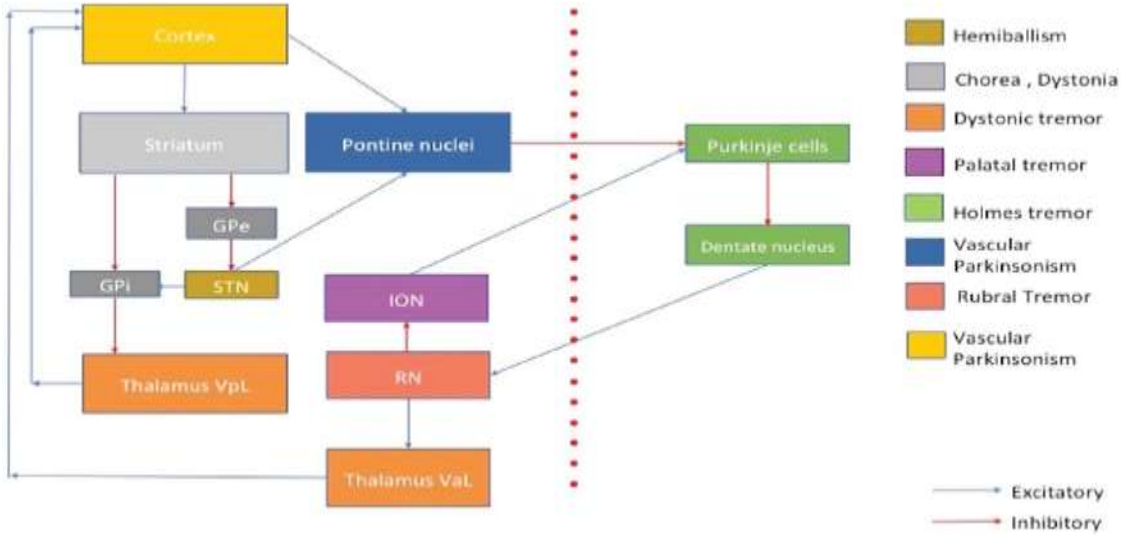
**Table 3 : Lipid profile**

| Investigations | Lab value | Reference range | Units |
|---|---|---|---|
| S. Cholesterol | 130 | <200 | mg% |
| S. Triglycerides | 120 | <150 | mg% |
| HDL | 44 | >60 | mg% |
| LDL | 62 | <130 | mg% |
| VLDL | 24 | <30 | mg% |

**Figure 2: Cerebellar circuit(5)**




## Treatment

The patient was started on antiplatelet (Tab Aspirin 150 mg) and statin (Tab Atorvastatin 10mg), Tablet Telmisartan and Amlodipine 40/5mg was started as an anti-hypertensive, Tablet Tetrabenazine 25mg BD and supportive treatment. The patient was kept under observation for 5 days and was discharged post improvement of symptoms. On 3 months follow-up, the patient's symptoms have resolved.

## Discussion

Lesions affecting any part of the motor circuitry, whether subcortical (affecting the basal ganglia, thalamus, internal capsule, diencephalon, and mesencephalon) or cortical (affecting the primary motor, supplementary motor, and premotor cortical areas) can show up as post-stroke movement disorders. Although the precise mechanisms underlying the pathogenesis of these conditions have not been fully characterized, disturbances in the crosstalk between inhibitory and excitatory circuits resulting from vascular damage are proposed as the underlying cause. The gamma-aminobutyric acid (GABA)ergic and dopaminergic systems play a key role in post-stroke abnormal movement.(1) Classically, it is caused by lesions in the subthalamic nuclei; however, it can be associated with lesions in other basal ganglia.

Information is relayed from the cortex to the thalamus and back by the core subcortical part of the motor circuitry, the basal ganglia. The two pathways that make up the cerebellar circuitry are the GMT (Guillain Mollaret triangle) or dentate-rubro-olivary pathway and the cortico-cerebello-cortical or dentate-rubro-thalamic pathway (figure 2).It has been noted that compared to cortical strokes, aberrant motions



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are more likely to develop after strokes that impact subcortical regions. (5)

The above figure illustrates the basal ganglia and cerebellar circuitries involved in the generation of movement disorders. The cortex has an excitatory influence over the striatum from where two pathways arise: direct and indirect basal ganglia pathways. Through the direct pathway, the globus pallidus interna (Gpi), which is also unable to inhibit the thalamic VpL nucleus, receives inhibitory signals from the striatum. As a result, the thalamus influences the cortex excitatory through the direct pathway. The globus pallidus externa (GPe), which is further unable to inhibit the subthalamic nucleus (STN), receives inhibitory signals from the striatum through the indirect pathway. As a result, the GPi, which has an inhibitory effect over the thalamic VpL nucleus, is now excited by the STN. The thalamus thus becomes inhibitory to the cortex through the indirect pathway. The pontine nuclei in the cerebellar circuitry receive excitatory signals from the cortex, cross the midline, and then exert an inhibitory effect on the cerebellum's Purkinje cells. Because of this, the Purkinje cells can no longer inhibit the dentate nucleus and can instead continue as the excitatory dentatorubrothalamic pathway that crosses over to the cortex. Additionally, the STN promotes the excitatory cerebellar impact over the cortex by exerting an excitatory influence over the pontine nuclei. The dentate nucleus on one side, the red nucleus (RN), and the inferior olivary nucleus (ION) on the other comprise the Guillain-Mollaret triangle (GMT). By sending out inhibitory signals, the red nucleus prevents the ION from exciting the Purkinje cells. Hypokinetic movement disorder results from damage to the direct basal ganglia route, whereas hyperkinetic movement disorder results from involvement of the indirect pathway. Unbalances in the neurotransmitter system in the basal ganglia or well-defined lesions of the nuclei are usually the causes of involuntary movements that follow a basal ganglia infarction. Recent evidence suggests that most movement disorders induced basal ganglia lesions result from defects in functional connectivity rather than from a single lesion. (5)

A decrease in the functioning of the indirect pathway leads to hyperkinetic movement disorders such as chorea and ballism. Nevertheless, certain studies in animal models provide compelling evidence for the theory that lesions in the subthalamic nucleus (STN) can also trigger hyperkinetic movements.

Certain movements can emerge suddenly in conjunction with a stroke, while others have a delayed onset and may even progress over time. Acute lesions disrupt the normal functioning of motor circuits, resulting in abnormal movements that typically cease when normal function is restored. In many cases, these abnormal movements begin to appear after an improvement in motor weakness, causing a delay in their presentation. This phenomenon underscores the concept of neuronal plasticity, where a parallel network forms to compensate for motor deficits, and the formation of pathological circuits gives rise to these abnormal movements.Most frequently, abnormal movements occur on the side opposite to the stroke site (contralateral). However, there have been instances where abnormal movements manifest on the same side as the lesion (ipsilateral). The clinical characteristics of movement disorders caused by basal ganglia circuit lesions closely resemble those seen in post-stroke movement disorders associated with different brain regions. In cases of movement disorders resulting from basal ganglia infarction, they typically present unilaterally on the opposite side of the ischemic lesion. This is attributed to a compensatory increase in the activity of structures on the side opposite to the stroke site, leading to abnormal movements on the same side as the lesion.

Hemichorea-hemiballism stands out as the most prevalent movement disorder following a stroke. Neuroimaging studies have consistently linked hemichorea or ballism with the subthalamic nucleus (STN), caudate, and putamen. In a comprehensive study involving 2500 patients conducted by Ghika Schimd et al., 29 patients exhibited post-stroke abnormal movements, with 11 of them (38%) experiencing hemichorea-hemiballism.(6)

Another study by Alarcon et al., which involved 56 patients, found that chorea was the most frequently observed movement disorder, affecting 20 patients (36%). Hemiballism has historically been associated with lesions in the contralateral STN but can also be linked to lesions in other areas of the brain. Lesions in the thalamus or lentiform nucleus are more commonly linked to hemiballism than those in the subthalamus. Alarcon et al. reported that among eight



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patients with hemiballism, one had subthalamic lesions, one had a pallidal lesion, and six had thalamic lesions.

A syndrome featuring hemiballismus and acute limb pain has been documented in cases of anterior parietal artery strokes. In another study involving 25 patients with hemiballism by Vidakovic et al., only one patient had an isolated contralateral subthalamic nucleus lesion, while others had various central nervous system parts involved, including the pons, midbrain, basal ganglia, thalamus, and cortex. (7)

Hemichorea is frequently associated with lesions in the lentiform nucleus or of the thalamus, but it has also been observed in cases involving the subthalamus, striatum, posterior limb of the internal capsule, corona radiata, frontal lobe, parietal lobe, temporal cortex, external capsule, and pons. Hemiballism typically occurs immediately with the onset of a stroke, although there have been reported cases with delays of up to 5 months. In contrast, hemichorea tends to manifest within a few days after a stroke.

Hemichorea-hemiballism is usually seen contralateral to the lesion site but there have been few case reports where it was found to be present on the ipsilateral side of the lesion. One patient had developed ipsilateral hemiballism postoperatively due to an infarct in the right anterior cerebral artery territory. All lesions were contralateral to the side of the body afflicted in a clinicoradiological examination of vascular hemichorea by Galiano et al., with the exception of one instance involving a left thalamic hematoma and ipsilateral hemichorea.

While small vessel disease is commonly associated with these movement disorders, it's important to note that vascular malformations such as cavernous malformation, moyamoya disease, and carotid stenosis can also be observed in some cases. In a study involving 42 patients with moyamoya disease, with an average onset age of 21.4 years, chorea was observed in 24 patients, and dystonia in 8 patients. Notably, after undergoing bypass surgery, all patients experienced an improvement in symptoms, suggesting that the underlying pathogenic process in moyamoya disease is reversible cerebral ischemia. Occasionally, ballism and chorea may manifest bilaterally or affect only one limb (monochorea). Many patients with ballism exhibit distal choreiform movements, and during the recovery phase, hemiballism often transitions into hemichorea and hemidystonia.

This study seeks to highlight a distinctive manifestation of movement disorder linked to an underlying stroke etiology. Early detection of this atypical presentation could

potentially alter the treatment trajectory, resulting in an improved prognosis. Numerous isolated case reports have documented similar presentations, emphasizing the need for a comprehensive examination and assessment. Establishing an algorithm for prompt identification and intervention in patients with movement disorders secondary to stroke is crucial, and analyzing these individual cases contributes to the development of such a framework.

Conclusion: Stroke-associated movement disorders are distinct entities requiring special consideration while evaluating patients with stroke. The pathophysiology of such disorders is relatively complex which needs further research in this area.

**Informed Consent**

Informed consent of the subject was taken for use of personal information, displaying test results and imaging.

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