Hyperkinetic Movement disorder as a Stroke Presentation in a Tertiary Hospital: A Case Series

Roshan Krytal R. Ulpindo, MD[a] and Neil Lee Ambasing, MD, FPNA[b]

ABSTRACT

Movement disorder, as a presentation of an acute or chronic cerebrovascular disease (CVD) occur in less than five percent of CVD cases. Although a rare presentation of CVDs, stroke is a common etiology of secondary movement disorder. Hemichorea is particularly prevalent following stroke. The objectives of this report are to (1) present nine cases of sudden-onset hyperkinetic movement disorders manifested in acute and chronic stroke patients (2) emphasize the importance of early diagnosis by clinical signs and symptoms identified through computed tomography (CT) and magnetic resonance imaging (MRI), and (3) determine the different anatomic locations involved in this disorder. Hemichorea is the most common hyperkinetic movement disorder seen after stroke with a predilection in older age. It demonstrated that deep vascular lesions had a greater probability of developing movement disorder. Hemichorea-hemiballismus with abrupt onset should be approached as an acute stroke until other potential causes are ruled out. The exact pathophysiology of these abnormal movements remains unclear, although some theories propose dysfunction within the motor circuitry pathway. While many cases resolve spontaneously, medical or surgical interventions may be necessary to manage symptoms, potentially influencing long-term outcomes.

***Keywords**: movement disorder, chorea, hemiballismus, stroke, motor circuitry*

INTRODUCTION

Cerebrovascular disease and movement disorders are common; however, movement disorders are a rare presentation of stroke.[9] Post-stroke movement disorders refer to complications related to ischemic or hemorrhagic strokes that cause damage to brain tissue.[18] Worldwide, the prevalence of post - stroke movement disorder is estimated to range from 1% to 4% [3,7] and represents 22% of all secondary causes of movement disorders.[7] The disorders can be categorized as hyperkinetic (chorea-ballism-athethosis, myoclonus, asterixis, dystonia and tremors) and hypokinetic (Parkinsonism, bradykinesia and akinetic mutism).[10] These disorders often manifest in the acute phase, frequently associated with thalamic infarctions resulting from deafferentation mechanisms. Alternatively, in the chronic phase, they may arise from lesions affecting the contralateral basal ganglia, particularly the subthalamic and lentiform nuclei.[11] Recent insights have also implicated cortical ischemic lesions as contributing factors to chorea following stroke.[6] In this report, we present nine cases of hyperkinetic movement disorder in a tertiary hospital in the Philippines highlighting the onset, etiology of stroke, different anatomic structures involved, pathophysiology and management.

CASE PRESENTATION

There were 9 patients, five males and four females who presented with hyperkinetic movement disorder in Baguio General Hospital and Medical Center (BGHMC) from 2019-2023 mostly within 24 hours of involuntary movements, which was the primary presentation in all cases. Cranial Magnetic resonance imaging (MRI) and Cranial computed tomography (CT) scan were done and showed lesions mostly at the

basal ganglia. All patients were given treatment and noted resolution of symptoms mostly within 2 - 5 days.

Case 1:
A 67-year-old female suddenly presented with a rapid, nonsuppressible, non-rythmic movement of her right forearm and hand for one day before visiting the emergency room. The patient was a known hypertensive on Losartan 100 mg 1 tablet once a day with a 20-pack year smoking history. Her blood sugar level was 121 mg/dl. Other diagnostic tests showed no abnormalities. A cranial MRI showed an acute infarct at the right caudate nucleus with old lacunar infarcts as follows: bilateral caudate, anterior limb of the internal capsule and bilateral external capsules and right lentiform nucleus (Figure 1A, 1B, 1C). The patient refused admission and was started with Aspirin 80 mg 1 tablet once a day, Clopidogrel 75 mg 1 tablet once a day and Rosuvastatin 20 mg 1 tablet once a day at night. She had spontaneous resolution of the involuntary movement a day after the consult. There was no EEG done to the patient.

Case 2:
A 60-year-old male experienced a one-day episode of high-amplitude, non-rhythmic involuntary movements of the left extremities. He is a known case of CVD intracerebral hemorrhage, right basal ganglia MRS 3 since 2019 and a known hypertensive maintain on Losartan 50 mg 1 tablet once a day, Amlodipine 10 mg 1 tablet once a day, Atorvastatin 20 mg 1 tablet once a day at night. Patient was also given Gabapentin 300 mg 1 tablet once a day at night for post stroke pain. Additionally, he had a history of alcoholism and smoking. A Cranial MRI was conducted revealing an acute to subacute parenchymal hemorrhage, right putamen and internal capsule with surrounding vasogenic edema and mass effect (Figure 2A). While the patient was admitted, an EEG was done revealing normal result. The patient was given Haloperidol 10 mg intramuscularly and was started on Valproic Acid 500 mg 2 tablets 2x a day, Clonazepam 2mg 1/2 tablet 2x a day. There was noted improvement on the 4th hospital day and was discharged.

Case 3:
A 60-year-old male presented with a 6-hour history of uncontrollable jerking and writhing movements affecting the right upper and lower extremities, along with facial involvement, numbness on the right side, and a severe headache. He is a known hypertensive and was non-compliant with medication. After undergoing a cranial MRI an acute to subacute hemorrhage at the left putamen (Figure 2B) was noted. An electroencephalomyogram was done 48 hours from admission revealing a normal result. He was treated with a combination of Amlodipine 5 mg 1 tablet once a day, Losartan + HCTZ 100 mg/12.5 mg 1 tablet once a day, Gabapentin 300 mg 1 tablet once a day at night and Levodopa/Carbidopa 100/25 1 tablet 2x a day. The patient experienced complete resolution of symptoms within 72 hours and was discharged improved on the 7th hospital day.

Case 4:
A 51-year-old female developed involuntary, rapid, jerky movements of the right upper extremity, which was followed by weakness and numbness of the right lower extremities. She had a history of hypertension with uncontrolled blood glucose levels and elevated HbA1c. Upon arrival at the ER the initial capillary blood glucose (CBG) was 341 mg/dl with an HBA1C of 12.3%. A cranial MRI was done revealing acute infarction involving the genu and body of the corpus callosum and pericallosal region (Figure 1D,1E,1F). Time of Flight (TOF) images of the Circle of Willis revealed loss of normal flow related enhancement of the bilateral pericallosal arteries maybe due to slow flow indicating a severe stenosis or occlusion. Patient was started with Aspirin 80 mg 2 tablets once a day, Losartan 5 mg 1 tablet 2x a day, Amlodipine 10 mg 1 tablet once a day, Atorvastatin 20 mg 1 tablet once a day and insulin 70/30 24 units in AM and 14 units in

PM. Her blood sugar levels were within normal ranges (98-133 mg/dl) however with persistent involuntary movements thus Risperidone 2 mg 1 tablet once a day was started; after which, there was total resolution of symptoms after 4 days. An EEG was not performed in the patient.

Case 5:
A 47-year-old female reported with involuntary movements in her left extremities, characterized by stiffening, jerking, and writhing with distractibility but no weakness for one month before visiting the outpatient department. The patient was a known hypertensive on Losartan 50 mg 1 tablet once a day and is living with diabetes was maintained on Metformin 500 mg 1 tablet once a day. Her laboratory result was normal and a cranial MRI showed a chronic infarct at the right caudate nucleus (Figure 1G,1H, 1I). Electroencephalomyogram was not done. She was treated with Cilostazol 100 mg 1 tablet 2x a day, Rosuvastatin 10 mg 1 tablet once a day at night and Metformin 500 mg 1 tablet 2x a day. Her hemichoreaathethosis ceased 2 weeks after administration of Valproic Acid 500 mg 1 tablet 2x a day.

Case 6:
A 66-year-old male with a history of hypertension, diabetes, and cardiovascular disease presented with a 2-week history of rhythmic, low-amplitude involuntary movements in his right extremity. A cranial CT scan showed a chronic infarct at the left putamen (Figure 1J). The EEG was done as an outpatient yielding a normal result. He was started on Aspirin 80 mg 1 tablet once a day and Valproic Acid 500 mg 1 table 2x daily for a symptomatic management of his myoclonus, leading to improvement of symptoms after 3 days.

Case 7:
An 82-year-old male experienced a one-day history of involuntary jerking and writhing movements in his right limbs, along with numbness and slurred speech. A cranial MRI was done revealing an acute infarct at the left thalamus (Figure 1K, 1L, 1M). He was treated with Risperidone 2 mg 1/2 tablet once a day at night, Aspirin 80 mg 2 tablets once a day, Atorvastatin 80 mg 1 tablet once a day at night and Amlodipine 10 mg 1 tablet once a day in pm and noted resolution of symptoms within 24 hours. No EEG was not performed.

Case 8:
A 68-year-old female presented with sudden flailing movements of her left limbs over a 7-hour period, without other symptoms. She had a history of stroke last 2018, CVD ICH, right basal ganglia. She is currently on Losartan 100 mg 1 tablet once a day and Amlodipine 10 mg 1 tablet once a day. A cranial MRI showed a chronic hemorrhage at the right caudate nucleus (Figure 2C). An EEG was not done. The patient was given Clonazepam 2mg 1/2 tablet 2x a day, Losartan and Amlodipine were continued. There was noted decreased involuntary movement prior to discharge and complete resolution three weeks after initiation of treatment.

Case 9:
A 59-year-old male, hypertensive on Losartan 50 mg 1 tablet once a day, person with diabetes on Metformin 500 mg 1 tablet 2x a day and post stroke patient (2017) MRS 2 on Cilostazol 50 mg 1 tablet once a day came in with a one-year history of rapid, shock-like contractions with irregular amplitude and rhythm of bilateral upper extremities. Upon arrival at the ER the initial CBG was 249 mg/dl with an HBA1C of 12.7%. The MRI revealed encephalomalacia from previous hemorrhagic process, right corona radiata and basal ganglia probable extension to the left basal ganglia via the Canal of Gratiolet (Figure 2D). An EEG was done showing an unremarkable result. Patient was maintained on Valproic Acid 500 mg 1 tablet once a day, Insulin 70/30 20 units in AM and 10 units in PM; and other medications were continued.

Figure 1. Cranial Imaging of Ischemic Stroke Patients Presenting with Hyperkinetic Movement Disorder on the first day of consult.

In case 1, Brain magnetic resonance image (MRI) shows the acute cerebral infarct in the right caudate nucleus (A: DWI B: ADC; C: T2FLAIR). In case 4, MRI shows the acute cerebral infarcts involving the genu and corpus callosum and pericallosal region (D; Diffusion weighted image (DWI), E: Apparent Diffusion Coefficient (ADC) and F: Fluid attenuated inversion recovery (FLAIR)). In case 5, MRI shows a chronic infarct at the right caudate nucleus (G: DWI, H: ADC, and I: T2FLAIR). In case 6, Cranial CT showed chronic infarct at the left lentiform nucleus (J). In case 7, MRI shows the acute cerebral infarct involving the left thalamus seen as hyperintensity in DW1 and T2Flair (K, M) and with corresponding signal drop on ADC (L).

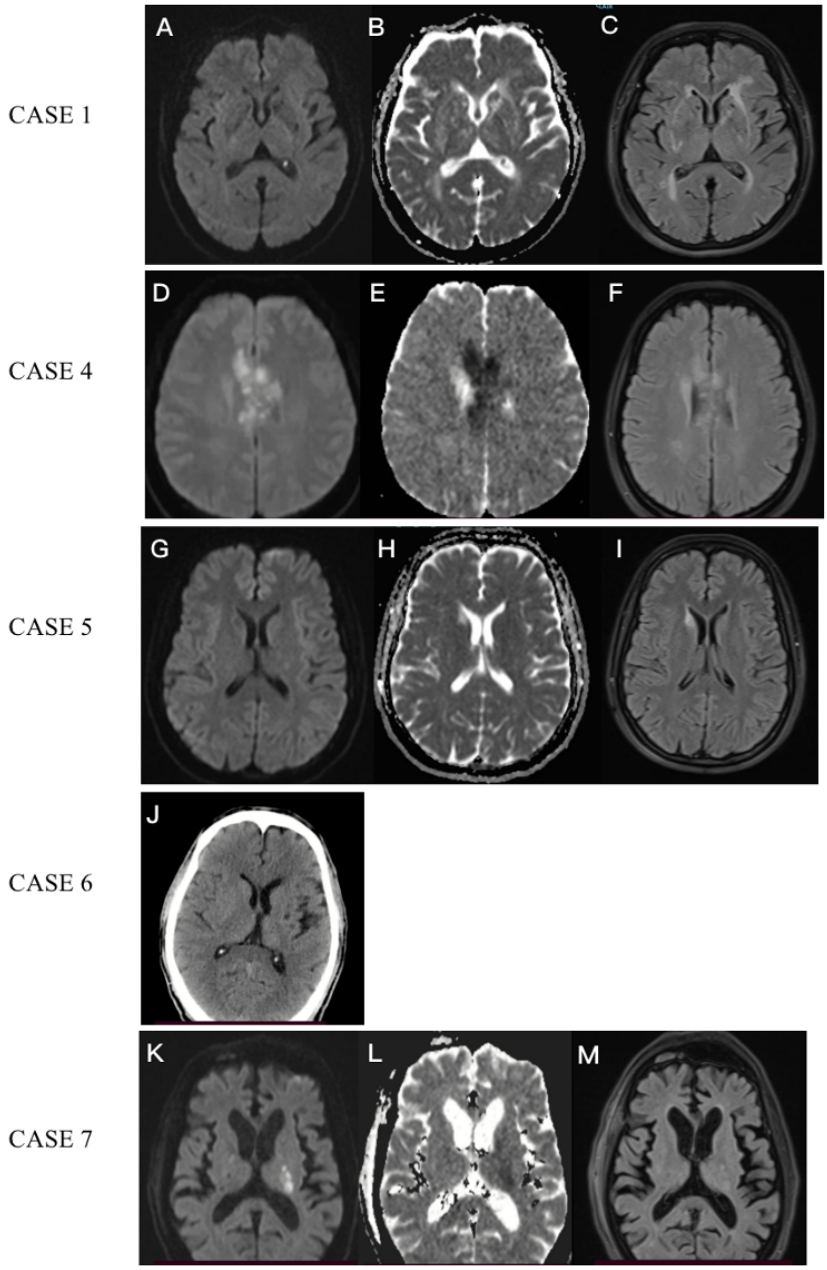
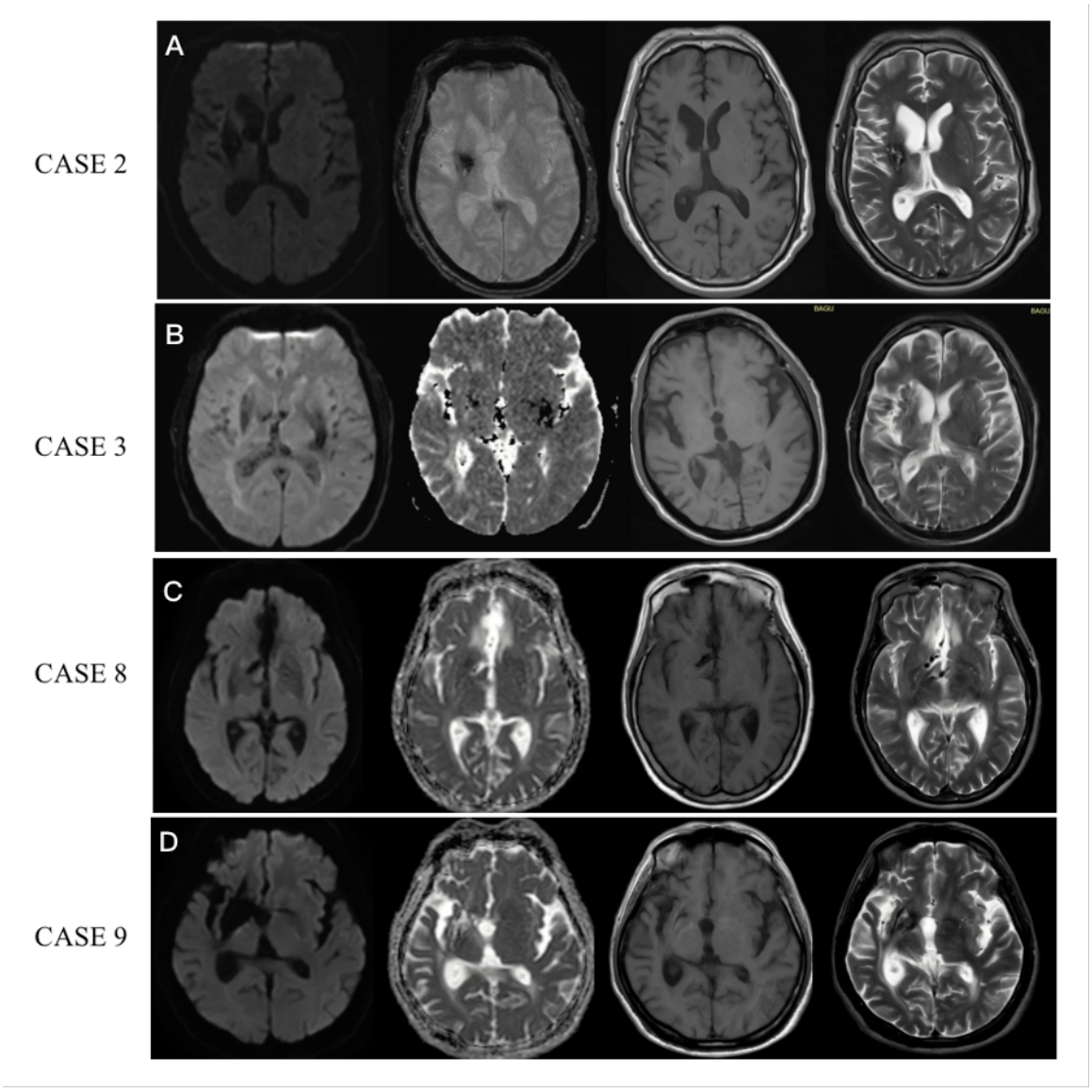


Figure 2. Magnetic Resonance Imaging (MRI) of Cerebrovascular Disease Intracerebral Hemorrhage patients presenting with Hyperkinetic movement Disorder on Day 1 of consult. The following MRI sequences as follows: DWI, ADC, T1 and T2.

A. Mixed hyperintense to hypointense on DWI and ADC with T1W isointense/T2W mixed hyper- to hypointense signal demonstrating rim and interspersed GRE susceptibilities located at the right putamen, surrounding T2 bright signals are seen. B. DWI and ADC showed hypointensity with T1 isointense/T2W hypointense signal at the left putamen C. Hypointense on DWI and ADC with T1/T2 hypointense revealing chronic hemorrhage at the right caudate nucleus D. Large area of CSF signal intensity is seen in the right corona radiata extending into the right basal ganglia with intralesional and perilesional GRE susceptibility signals. There is a tubular GRE susceptibility signal connecting the two basal ganglia adjacent to the anterior commissure.



DISCUSSION

Post stroke movement disorders (PSMD) are relatively rare, with reported incidences of about 0.08% per year,[13] their occurrence highlights an important aspect of stroke outcomes. The origins of PSMDs are complex and multifaceted, involving several neurobiological mechanisms that disrupt normal motor control(8). Patients who later develop abnormal movements following a stroke generally present with motor dysfunction initially.[14] By the time these abnormal movements appear, the initial motor deficits often show some degree of improvement. Despite the established link between vascular lesions and these disorders, their incidence remains notably low, with a prevalence ranging between 0.4% and 0.54%[4] in stroke patients. This rarity underscores the complexity and specificity of post-stroke movement disorders compared to more common manifestations like weakness. This is more complex and specific due to several interconnected factors involving the motor circuity and maybe an etiology of other neurologic etiology (encephalopathy, seizure, autoimmune disorders or malignancy).

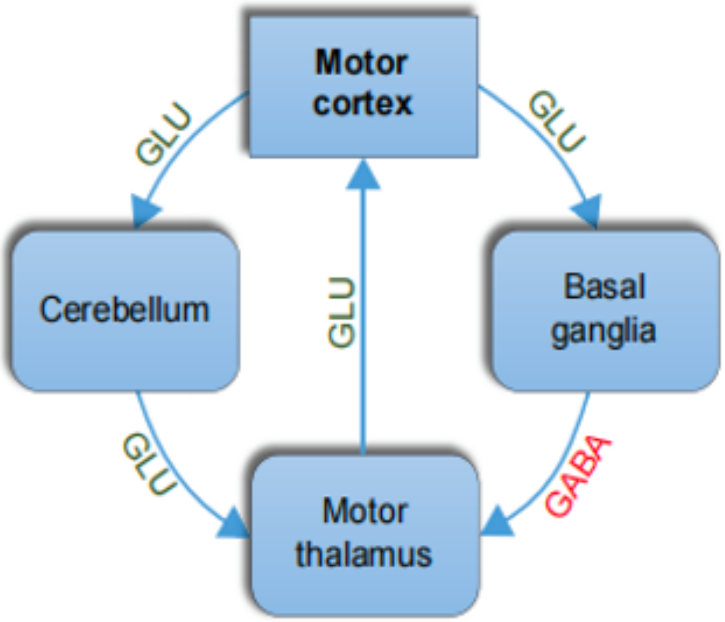
Chorea, ballismus, athethosis, myoclonus, asterixis, dystonia and tremors maybe a manifestation of post-stroke hyperkinetic movement disorders.[10] Typically, these involuntary movements vary widely and are influenced by factors such as stroke type, lesion location, and patient age.[1] In a case review of PSMD patients, parkinsonism, chorea, and dystonia are common after ischemic strokes, while dystonia, tremor, and myoclonus are more frequent after hemorrhagic strokes.[16] Lesions in the basal ganglia (44%), thalamus (37%), and cerebral cortex (12%) are frequently involved in PSMDs.[2,4,11]

Hemiballism and hemichorea are the most common hyperkinetic movement disorders after a stroke, seen in 40% of cases. About 72% of hemiballism cases are due to CVD and usually related in ischemic lesions in the contralateral subthalamic nucleus while hemichorea is associated with damage to the lentiform nucleus or thalamus[3] with an average onset age of 66.[13] Dystonia is the second most frequent movement disorder, accounting for around 20% of cases, often appearing as focal or hemidystonia. Stroke causes nearly 50% of hemidystonia cases, with most instances in individuals over 50 years old linked to stroke.[19] The usual involvement is in the putamen, caudate, pallidum, thalamus, or midbrain. Overall, subcortical strokes are three times more likely to result in movement abnormalities than cortical strokes, with basal ganglia and thalamic lesions being the most frequently involved.[7]

Hemorrhagic strokes have a higher likelihood of leading to PSMDs compared to ischemic strokes, without a clear preference for specific vascular regions or subtypes.[9] The frequency of movement disorder in hemorrhagic stroke may be attributed to deeper subcortical structures and larger lesion involvement.[20] Research indicates that patients with deep basal ganglia, thalamic, or brain stem lesions who develop abnormal movements often have a higher incidence of hemorrhages. Among patients with ischemic stroke, large vessel stenosis/occlusion is more commonly associated with PSMDs followed by multifocal small vessel stroke, lacunar and cardioembolic stroke.[9] Chronic small vessel disease, such as Binswanger's disease, can cause secondary parkinsonism with bilateral slowness and rigidity, particularly affecting the lower extremities.[12]

Motor control is a complex process regulated by intricate motor circuitry that includes both pyramidal (cortical) and extrapyramidal (basal ganglionic and cerebellar) systems.[21] While motor commands originate in the motor cortex, the basal ganglia and cerebellum refine these signals through feedback loops, enabling smooth, accurate, and coordinated movements. The cortico-basal loop,

Figure 3. A simplified diagram of the motor control circuitry. The motor cortex projects to the cerebellum and basal ganglia and receives feedback signals from them. Although cerebellar output is tonically excitatory and basal ganglia output is tonically inhibitory, the balance between these two systems is of pivotal importance for motor control and coordination. GLU and GABA are the major excitatory and inhibitory neurotransmitters in this network, respectively.



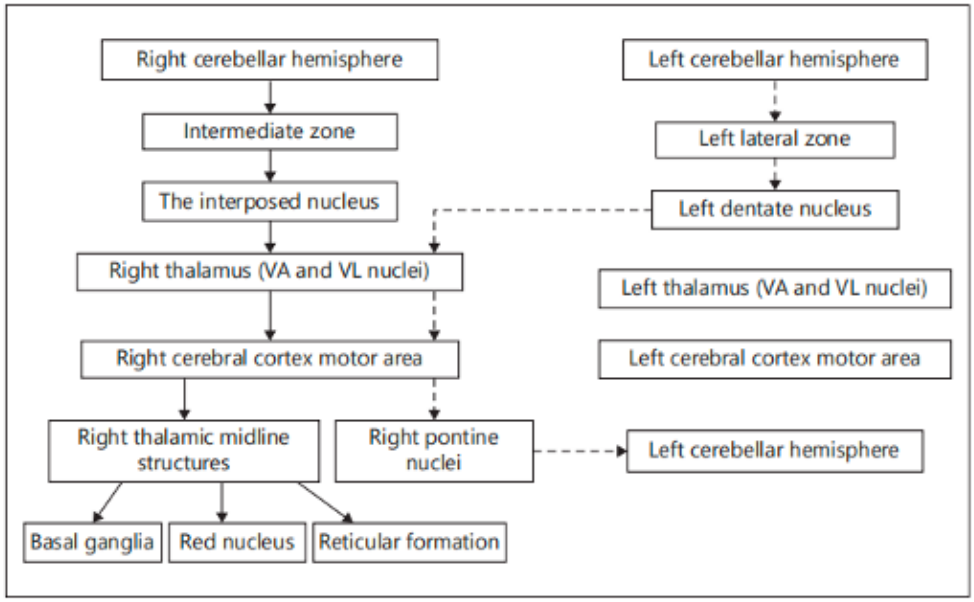
associated with the basal ganglia, provides tonic inhibitory feedback to the thalamus and, consequently, the motor cortex, whereas the cortico-cerebellar loop delivers tonic excitatory feedback (Figure 3). In this motor network, glutamate (Glu) functions as the primary excitatory neurotransmitter, while gamma-aminobutyric acid (GABA) serves as the main inhibitory neurotransmitter.

An acute onset movement disorder maybe a presentation of an acute stroke. In PSMD one hypothesis is the alteration in neuronal membrane excitability, which affects how neurons respond to stimuli.[8] Additionally, the loss of GABAergic inhibition around the perilesional area—caused by the stroke—can result in diminished inhibitory control, leading to the emergence of hyperkinetic movements.[7] Enhanced glutamatergic transmission, another post-stroke change, can contribute to this phenomenon by increasing excitatory signaling in the contralesional hemisphere, accompanied by reduced interhemispheric inhibition[12]. Such changes can enhance cortical excitability and facilitate motor recovery but may also lead to the emergence of abnormal movements as a side effect.

The delayed onset of involuntary movements could suggest the time needed for an unbalanced yet successful recovery of motor function, leading to the formation of pathological neuronal circuits, or it might reflect the time required for potential changes in synaptic activity within the neurons.[20] Postischemic synaptic plasticity also plays a crucial role, as the brain undergoes reorganization in response to stroke. This reorganization involves the activation of previously silent or less active synapses near the lesion, leading to the unmasking or disinhibition of these circuits and resulting in abnormal motor outputs.

The cerebellar motor circuitry consisting of the two cerebellar feedback loops, the Guillain-Mollaret triangle (GMT) and the cortico-cerebello-cortical pathways,

Figure 4. A simplified diagram of the cerebellar motor circuitry. The cerebellar and thalamic connections: the pathway from the intermediate zone of the cerebellar hemisphere to thalamus helps coordinate reciprocal contractions of agonist and antagonist muscles in the peripheral musculature.



contribute to motor control and can be involved in movement disorders.[3] The interactions between the cerebellum and the motor cortex, through the dentate-rubro-olivary and dentate-rubro-thalamic circuits, are critical in coordinating motor signals.[10] The motor cortex sends projections to the cerebellar cortex through the pontine nuclei, which then primarily connect to the VL nuclei of the thalamus before relaying signals back to the cerebral cortex. The inferior olivary nucleus receives collateral inputs from all afferent pathways targeting the cerebellar cortex via mossy fibers (Figure 4).[21] It compares intended movements with those that are executed and transmits error signals to the cerebellar cortex via climbing fibers. Through alterations in these pathways, symptoms of dyskinesias can exacerbate.

The diagnosis of post stroke movement disorder is through clinical context and cranial imaging. Electroencephalomyogram (EEG) can be warranted in movement disorders, particularly when there is suspicion of seizures or non-epileptic events that may mimic movement disorders. It is particularly useful where the nature of episodic movement is unclear and for differentiating conditions such as epileptic seizures, were sudden, uncontrolled movements might indicate seizure activity. Additionally, EEG can help rule out psychogenic non-epileptic seizures (PNES), which are caused by psychological factors rather than neurological ones.

The management options for post-stroke movement disorders whether acute or chronic and ischemic or hemorrhagic, vary based on symptom severity and response to therapy.[1,2,5] Acute ischemic stroke patients are typically treated with thrombolytic therapy within 4.5 hours of symptom onset.[6] Short-term treatments may be necessary for controlling symptoms in secondary movement disorders, with an emphasis on addressing underlying causes.

For post-stroke choreiform dyskinesias, symptomatic treatment primarily involves anti-dopaminergic therapies using typical or atypical antipsychotics. The effectiveness of these

medications is attributed to their ability to block striatal dopamine D2 receptors, thus reducing involuntary movements.[15,21] However, there are risks associated with dopaminergic blockade, including acute dystonic reactions and tardive dyskinesia, making atypical antipsychotics a preferred option due to their lower side effect profile.

In addition to anti-dopaminergic drugs, various non-dopaminergic medications have been explored for managing vascular choreiform dyskinesias, with mixed results.[8] Case reports indicate that antiseizure medications such as Levetiracetam, Valproic Acid and Topiramate might offer some benefit. Newer selective vesicular monoamine transporter (VMAT) blockers have also been developed, showing promise with fewer side effects compared to traditional treatments.[3,6,9]

Post-stroke tremors present a significant challenge in pharmacotherapy, often proving resistant to conventional treatments. Medications with GABA-agonistic properties, like clonazepam and topiramate, may be beneficial in some cases.[21] For tremors associated with the dopaminergic system, such as Holmes tremor, treatment with levodopa or dopamine agonists may yield positive results. Dystonia management frequently relies on botulinum toxin injections, which are effective in alleviating muscular contractions responsible for dystonic posturing. Alongside botulinum toxin, other medical treatments include benzodiazepines[3], baclofen, and anticholinergic drugs, which can also contribute to symptom relief. While benzodiazepines are effective, caution is necessary due to potential side effects like drowsiness at higher doses.

In severe or drug-resistant cases, deep brain stimulation (DBS) targeting the globus pallidus internus has proven effective, as evidenced by significant symptom improvement in some patients.[3,8,9,12] Additionally, revascularization and motor cortex stimulation can offer substantial relief, particularly in cases of vascular hypoperfusion or chronic movement disorders. Spontaneous resolution of symptoms often occurs within weeks to months,[15] but persistent cases may necessitate ongoing treatment and advanced interventions.

CONCLUSION

Despite being uncommon, a range of abnormal movements, including hyperkinetic and hypokinetic types, can emerge during or post-stroke. The rarity of movement disorders following basal ganglia lesions suggests that stroke location alone is insufficient to predict these conditions. Instead, stroke type and individual patient factors play a crucial role. While many of these disorders are self-limiting, some may require intervention for effective symptom management. Treatment typically combines pharmacological and surgical approaches, tailored to symptom severity, with advanced options like deep brain stimulation (DBS) reserved for more resistant cases.

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