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# Post Stroke Chorea: A Rare Case Report

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**Abstract:**
Post-stroke chorea is a neurological condition characterized by rapid, irregular, and unpredictable movements typically affecting one side of the body. This disorder can manifest after ischemic or hemorrhagic strokes and presents diagnostic challenges due to varied initial symptoms. Common causes include Huntington's disease, infections, and metabolic disorders. Diagnosis relies on comprehensive history and neuroimaging. Treatment options encompass pharmacotherapy, including neuroleptics and antiepileptics, alongside non-pharmacological interventions such as physiotherapy. Rehabilitation aims to improve motor control and functional independence through personalized therapy. We present a case of a 40-year-old male with post-stroke chorea following TB meningitis, demonstrating dystonic tremors in the left upper limb. Treatment includes methylcobalamin, pantoprazole, haloperidol, clonazepam, aspirin, atorvastatin, propranolol, and supportive medications. The patient showed mild improvement with pharmacotherapy and physiotherapy. Laboratory findings revealed alterations in neutrophils, lymphocytes, MCV, and CRP levels. This case underscores the complexity of managing post-stroke chorea and highlights the importance of interdisciplinary care.

**Keywords:** post-stroke chorea, neurological disorder, ischemic stroke, pharmacotherapy, rehabilitation.

## Introduction:

Post-stroke chorea refers to a neurological disease characterized by rapid, rapid, irregular and unpredictable development after a stroke, such as an ischemic or hemorrhagic stroke. These movements usually involve one side of the body and may involve more than one muscle, which can cause coordination and control problems.[1] Dyskinesias are a known complication of stroke; both hyperkinetic and hypokinetic dyskinesias have been accounted after ischemic and haemorrhagic stroke. There are reports of Hemichorea-hemiballism, Dystonia, Tremor, Myoclonus, Parkinsonism after stroke as well as in disorders that were delayed or progressed.[2] Post stroke dyskinesia report up to 22% of all secondary movement disorders. However, post stroke dyskinesias are only observed in 1%-4% ofall strokes.[3]

## Terminology

A stroke refers to a neurological impairment resulting from either a localized blockage in the central nervous system or spontaneous bleeding within the brain, leading to dysfunction.[4] While focal neurological symptoms like hemiparesis typically indicate stroke, non-focal deficits or positive neurological signs like hemi dyskinesia

can sometimes be the first signs, leading to diagnostic challenges and delayed treatment.[4-5] The term Chorea refers to irregular, flowing, random, involuntary motions that frequently have a writhing quality known as choreoathetosis.[6]

Causes of post-stroke Chorea are Huntington's disease, Neuroacanthocytosis, Dentatorubral pallidoluysian atrophy, Benign hereditary chorea, Wilson's disease, Sydenham's chorea, Drug induced chorea, Infectious chorea, Vascular chorea, Hormonal disorders and other. Common causes include Huntington's disease, a genetic disorder affecting the brain, as well as certain infections, metabolic disorders, and side effects of medications. Neurological conditions such as Sydenham's chorea can also trigger chorea symptoms.[7-8]

Diagnosis of post-stroke dyskinesias relies on comprehensive history, identifying dominant abnormal movements, and neuroimaging to confirm stroke. Typical presentations include hemi choreiform dyskinesia or dystonia, often contralateral to the lesion. Tremor, myoclonus, and asterixis are less common.

**Types of movement disorder**

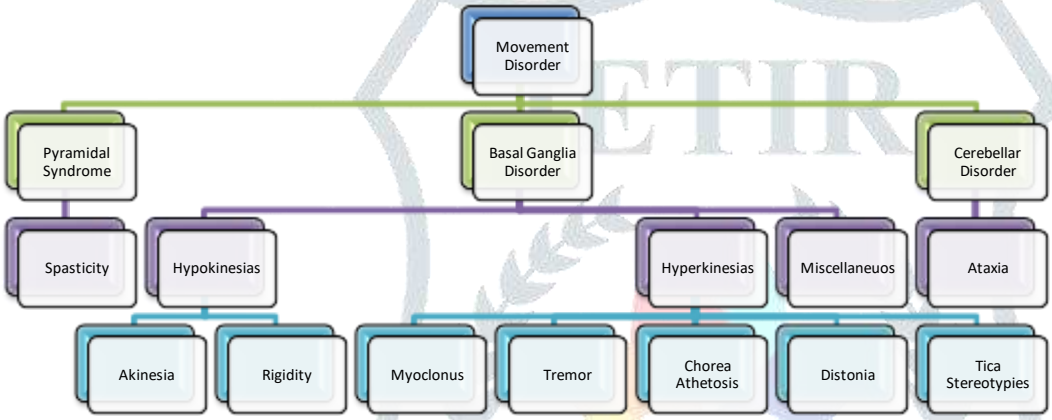


Chart: Types of movement disorder [9-10]

**Pathophysiology**

The generation of movement disorders involves intricate interactions within the basal ganglia and cerebellar circuitries. These systems work together with cortical input to modulate motor functions.

In the cortex, excitatory signals project to the striatum, initiating the basal ganglia pathways: the direct and indirect pathways. In the direct pathway, the striatum inhibits the globus pallidus interna (GPi), relieving inhibition on the thalamic VpL nucleus, resulting in cortical excitation. Conversely, in the indirect pathway, the striatum inhibits the globus pallidus externa (GPe), which in turn fails to inhibit the subthalamic nucleus (STN). Consequently, the STN activates the GPi, leading to thalamic inhibition and subsequent cortical suppression.

Meanwhile, cerebellar circuitry involves cortical excitation of pontine nuclei, which then inhibit Purkinje cells in the cerebellum. This disinhibits the dentate nucleus, initiating the crossed dentatorubrothalamic pathway, ultimately facilitating cortical excitation.

Moreover, the STN enhances the excitatory cerebellar influence over the cortex by stimulating pontine nuclei.

The Guillain-Mollaret triangle (GMT) comprises the dentate nucleus, red nucleus (RN), and inferior olivary nucleus (ION). The RN regulates the ION via inhibitory signals, preventing excessive excitation of Purkinje cells. [11-12-13]

**POST STROKE CHOREA MEDICATION TREATMENT OPTION**

1. **Dystonia**

   Drug-Trihexyphenidyl, Baclofen, clonazepam, diazepam, tetrabenazine, Botulinum toxin injection, intrathecal baclofen, Motor cortex stimulation, functional neurosurgery

2. **Vascular parkinsonism**
   Drug-Dopaminergic drugs
3. **Chorea/ballism**
   Drug-Neuroleptics-haloperidol, perphenazine, fluphenazine, Atypical neuroleptics: olanzapine, quetiapine, clozapine, Dopamine-depleting agents: tetrabenazine, reserpine, GABA receptor agonist: clonazepam, valproic acid
4. **Antiepileptics**
   Drug- levetiracetam, topiramate, Reperfusion surgery
5. **Myoclonus**
   Drug -Valproic acid, levetiracetam, Clonazepam, acetazolamide
6. **Tic**
   Drug -Clonidine, neuroleptics, dopamine-depleting agents
7. **Restless legs syndrome**
   Drug-Dopaminergic drugs
8. **Tremor**
   Drug-Propranolol, primidone, trihexyphenidyl, Clonazepam, diazepam, Motor cortex stimulation, functional neurosurgery [14]

**Non-pharmacological interventions for post-stroke chorea**

Non-pharmacological interventions for post-stroke chorea include physiotherapy, occupational therapy, and motor and communication therapy. Tools and changing environments help with daily tasks. Psychosocial support and counseling helps individuals and families cope with emotional problems. These supports improve overall health and functional independence.[15]

**Rehabilitation for post-stroke chorea**

Personalized physical and occupational therapy is part of the rehabilitation process for post-stroke chorea, with the goal of improving motor control, coordination, and functional independence. Communication problems are addressed through speech therapy. Mobility is facilitated by assistive technology, and emotional difficulties are managed with the help of psychosocial assistance. Long-term well-being is promoted and healing is optimized through interdisciplinary treatment. (16-17-18-19)

**Case Presentation:**

A 40-year-old male presented with involuntary rhythmic dystonic coarse tremors which increased in postural change in left upper limb. Patient has history of TB meningitis in 2014 with multiple infarcts in left basal ganglia, right thalamus, right temporals, right pons and right superior cerebellar hemisphere. 3 months post recovery he developed tingling with involuntary spasmic movements of left upper limb since that time. He was admitted multiply for TB meningitis in 2014 and TBM with left hand P/O Epilepsia Partialis Continua in 2015, post TBM with Epilepsia Partialis Continua with chronic right thalamic infarct with erectile dysfunction in 2017. Patient is addicted to alcohol. On examination patients had left upper limb dystonia with tremorness V/S choreoathetoid movements in all postures which disappears on sleep and circumductive gail with 4/5 power in left extremities with spasticity brisk reference in left half. Patient was taking AKT for 9 months (2014); Clobazam (2015); Phenytoin, Clobazam, Eco-AV, PeroxCR, Levoca, Cisplatin and Sypradyn (2017). MRI Brain showed multiple T2 hyperintense areas involving right thalamus and right medial temporal lobe, suggestive of chronic infarct. MRA brain revealed fetal origin of right posterior cerebral artery and absence of flow related to enhancement in A1 segment of right

anterior cerebral artery and hypoplastic right vertebral artery. Patient underwent physiotherapy for task specific functional exercises, one leg standing and gait reeducation from 06/08/22 to 08/08/22. After starting pharmacotherapy and physiotherapy patient had mild improvement in postural resting tremor on 2nd day.

| Lab Parameters | Results | Reference Range |
|---|---|---|
| Neutrophils | 55 % | 60-70% |
| Lymphocytes | 36% | 20-35% |
| MCV | 80.1 | 82-96fl |
| CRP | 1.58 | 0.6 mg/L |

**Table 1: Laboratory Investigations**

**Current theray treatment**

| Brand Name | Generic Name | Dose | Frequency | Indication |
|---|---|---|---|---|
| Optineuron | Methylcobalamin | 1500 mcg | Once daily | Neuropathy due to vitamin B12 deficiency |
| Pantoprazole | Pantoprazole | 40 mg | Once daily | Gastric acid suppression |
| Haloperidol | Haloperidol | 1 mg | Twice daily | Management of psychosis, including dystonia |
| Clonazepam | Clonazepam | 0.5 mg | Twice daily | Treatment of tremors and epilepsy |
| Aspirin + | Aspirin + Atorvastatin | Aspirin 75mg | Once daily | Prevention of cardiovascular events |
| Atorvastatin 10mg | Atorvastatin 10mg | 10 mg | Once daily | Lowering cholesterol levels |
| Propranolol | Propranolol | 10 mg | Twice daily | Management of tremors and anxiety |
| Liquid Paraffin | Liquid Paraffin | - | As needed | Relief of constipation |
| Ondansetron | Ondansetron | 4 mg | As needed | Management of nausea and vomiting |
| Paracetamol | Paracetamol | 500 mg | As needed | Relief of pain and fever |
| Bisacodyl | Bisacodyl | 5 mg | As needed | Treatment of constipation |
| Naproxen + | Naproxen + Domperidone | Naproxen 250mg | As needed | Relief of pain and inflammation, with added anti-nausea effect from Domperidone |

**Discussion:**

The case presented illustrates the complexity of post-stroke chorea management, particularly in patients with a history of multiple neurological insults, such as TB meningitis and recurrent strokes. Post-stroke chorea, characterized by involuntary movements, poses significant challenges in diagnosis and treatment due to its varied etiologies and manifestations.

In this case, the patient's history of TB meningitis and subsequent strokes highlights the multifactorial nature of post-stroke chorea. The involvement of various brain regions, including the basal ganglia and thalamus, contributes to the diverse motor symptoms observed, such as dystonia, tremors, and choreoathetoid movements.

Treatment strategies for post-stroke chorea typically involve a combination of pharmacotherapy and non-

pharmacological interventions. Pharmacological agents like haloperidol and clonazepam aim to alleviate symptoms and improve functional outcomes. Additionally, physiotherapy plays a crucial role in enhancing motor control and coordination, as demonstrated by the patient's mild improvement after starting pharmacotherapy and physiotherapy.

However, the patient's comorbidities, including alcohol addiction and cardiovascular risk factors, further complicate management and require a comprehensive approach. Addressing these underlying issues is essential for optimizing long-term outcomes and preventing recurrent strokes.

**Conclusion:**

Post-stroke chorea presents a complex clinical scenario necessitating a multidisciplinary approach for effective management. This case underscores the importance of thorough evaluation, considering both neurological and systemic factors contributing to the patient's condition. By integrating pharmacological treatment, rehabilitation, and addressing comorbidities, clinicians can strive to improve symptoms and enhance the patient's quality of life post-stroke. Continued research and clinical efforts are warranted to further refine treatment strategies and enhance outcomes in individuals with post-stroke chorea.

**Conflicts of interest**

The authors declare that there is no conflict of interest. There are no conflicts of interest

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