



Case Report: Ischaemic Stroke Presented with Hemichorea-Hemiballism

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ABSTRACT

Introduction: Movement disorders can be separated into hypokinetic disorders, which cause paucity or slowness (bradykinesia), and hyperkinetic disorders, which cause excessive, aberrant involuntary motions. Less than 5% of individuals with cerebrovascular diseases presented with involuntary movement. It might be difficult to identify and diagnose hyperkinetic disorders.

Case: We describe a 56-year-old man who arrived at the hospital with 5 hours of abrupt, uncontrollable movement in his right upper and lower limbs. A complete neurological evaluation revealed an uncontrolled, nonrhythmic, non-patterned, aimless, and frequently jerky movement of the right upper and lower limbs with a ballistic component that varies in amplitude and frequency. Higher psychic function and cranial nerves were normal. Chest radiography, electrocardiography were normal. Hemorrhage was ruled out by a brain non-contrast CT scan at admission. The patient was diagnosed with hemichorea-hemiballism caused by an ischemic stroke based on clinical evidence of a sudden neurological deficit of aberrant involuntary movement. After receiving medical treatment for five days, the involuntary motions stopped occurring without causing any more neurological abnormalities or weakening.

Discussion: Ischemic stroke diagnosis relied on skilled clinical assessment without explicit neuroimaging. While hemiballismus is characterized by violent irregular flinging movements of the limbs brought on by contractions of the proximal muscles, hemichorea consists of continuous random, anarchic, and jerking movements involving both the distal and proximal muscles (though it is occasionally localized more distally).

Conclusion : Hyperkinetic movement disorders are a rare presentation of stroke. The pathophysiology of these abnormal movements remains uncertain. Even though they are uncommon, following a stroke, aberrant motions can occur suddenly or develop gradually. Hemichorea-hemiballismus with abrupt onset should be treated as an acute stroke unless proven other causes.

Introduction

Movement disorders can be separated into hypokinetic disorders, which cause

paucity or slowness (bradykinesia), and hyperkinetic disorders, which cause excessive, aberrant involuntary motions

(Tater & Sanjay, 2021). Less than 5% of individuals with cerebrovascular illnesses have hyperkinetic and hypokinetic movement abnormalities (Caproni & Colosimo, 2017). It might be difficult to identify and diagnose hyperkinetic disorders (Alonso et al., 2015). Less than 4% of all strokes result in post-stroke movement problems (Caproni & Colosimo, 2017; Tater & Sanjay, 2021). The incidence peak occurs in the sixth and seventh decades of life, and there are no appreciable variations between males and females (Caproni & Colosimo, 2017). Between 0.4% to 0.54% of acute ischemic stroke patients had hemichorea-hemiballismus, with a frequency of 1% (Chen & Xu, 2020).

A stroke can cause a variety of movement abnormalities, each with a unique natural history, prognosis, and course of therapy from its idiopathic counterpart (Tater & Sanjay, 2021). One of the most striking diseases in neurology is hemichorea-hemiballismus. In the emergency room, it is not usually observed (Alonso et al., 2015; Carbayo et al., 2020). Because it is clinically uncommon, post-stroke hyperkinetic movement disorder can sometimes develop gradually over time rather than immediately after an acute stroke, which makes diagnosis more challenging and prone to mistake (Chen & Xu, 2020). This is an uncommon case of an

ischemic stroke patient who had an abrupt involuntary movement.

Case

A 56-year-old man presented to the emergency room with sudden involuntary movement in the right upper and lower limbs for 5 hours before he came to hospital. This movement disappeared only during sleep, and it was impairing his basic activities of daily living. There was no headache, language difficulty, visual disturbance, paraesthesia, or sign of infection. He had no family history of such disorders. There was no history of diabetes mellitus, hypertension, epilepsy, trauma and no prior exposure to neuroleptics. Patient is an active smoker.

On initial exam, patient is obese the blood pressure was 119/81 mmHg, the temperature was 36,5°C, the pulse was 98 beats per minute, the respiratory rate was 22 breaths per minute, and the oxygen saturation was 99% while breathing room air. He was alert and cooperative. The patient had no carotid bruits and no significant jugular venous distention. Cardiovascular exam revealed a regular rate and rhythm with no murmurs. A complete neurological evaluation revealed an uncontrolled, nonrhythmic, nonpatterned, aimless, and frequently jerky movement of the right upper and lower limbs with a ballistic component that varies in amplitude

and frequency. Higher psychic function and cranial nerves were normal. Sensations in all limbs were intact. Other systemic evaluations were unremarkable. There was no postural instability, ataxia, myoclonus, abnormalities on visual field evaluation, facial deviation, dysarthria, tremor or rigidity. Muscle power and reflexes were normal, and there was no clonus or positive Babinski sign.

Blood count and chemistries were found to be in normal limit as follows; Hb 15,2 g/dl, Hct 47%, WBC 12.700/ μ l, Plt 237.000/ μ l, AST 20,5 U/L, ALT 29,7 U/L, BUN 22,4 mg/dl, Cr 1,24 mg/dl, T-Chol 149 mg/dl, LDL-Chol 115 mg/dl, Glu 113 mg/dl, uric acid 6,3 mg/dL, Na 144 mEq/L, K 3,6 mEq/L, Cl 107 mEq/L. Chest radiography, electrocardiography were normal. Brain non-contrast CT scan on admission excluded hemorrhage (Figure 1.).



Figure 1. Non contrast brain CT scan examination did not shown any abnormality

Based on clinical clinical of sudden neurological deficit of abnormal involuntary movement and radiological findings with limited perfusion CT or MRI

facilities for further evaluation the patients was diagnosed with hemichorea-hemiballism due to ischaemic stroke. Unfortunately, the patient did not get thrombolytic therapy due to excessive time since symptom onset upon arrival. The patient was treated with long term low dose aspirin, piracetam as a neuroprotectant and patient's involuntary movement was treated symptomatically with haloperidol. After 5 days of treatment in hospital, the involuntary movements disappeared without any further weakness or neurological abnormality.

Discussion

Movement disorders are characterized by a slowness or paucity of movement (bradykinesia and hypokinesia), or at the other hand of the spectrum, excessive movement caused by abnormal involuntary movements, such as tremor, dystonia, chorea, ballism, athetosis, and myoclonus (hyperkinetic movement disorders) (Caproni & Colosimo, 2017). Sudden cease of movements and appearance of new focal neurological signs require clinical reassessment and possibly neuroimaging studies to look for ischaemic or haemorrhagic insults (Carrion & Carrion, 2013). Hyperkinetic movement disorders are a rare presentation of stroke. The pathophysiology of these abnormal movements remains uncertain (Carbayo et

al., 2020). The prevalence of hyperkinetic diseases tends to exceed that of hypokinetic syndromes (Caproni & Colosimo, 2017). A range of hyperkinetic movement disorders called hemichorea-hemiballismus include choreic and/or ballistic motions of variable intensity (Alonso et al., 2015; Wei & Zhang, 2021). Hemichorea describes motions that impact both the proximal and distal limbs and are similar in nature but less in amplitude. Early in the presentation, there is a pronounced hemiballismus, and as the condition resolves, a lesser amplitude hemichorea emerges (Alonso et al., 2015). Movement disorders share common etiologies, prognoses, and treatments, hence there is probably little pathophysiologic variation between them. In actuality, they frequently coexist in the same patient (Alonso et al., 2015; Tater & Sanjay, 2021).

A hyperkinetic movement disease called hemichorea-hemiballismus causes unintentional, large-amplitude limb excursions that primarily affect distal body regions. Choreic motions are not frequently observed in emergency situations. Acquired hemichorea-hemiballismus syndrome can result from a number of disorders polycythemia rubra, chorea gravidarum, oestrogen or levodopa replacement therapy, infectious, autoimmune, structural damage to deep brain structures (cerebrovascular disease, infection, trauma, neoplasia), or neurodegenerative conditions (Chen & Xu,

2020; Guida et al., 2013; Ueta et al., 2021). Focused brain lesions have been linked to hemichorea, hemiballismus, dystonia, tremor, myoclonus, parkinsonism, and asterixis, among other abnormal involuntary movements (AIMs) (Defebvre & Krystkowiak, 2016). These disorders can be classified by their genesis as primary (caused by one of several neurodegenerative diseases) or secondary (caused by a specific cause). Cerebrovascular diseases cause roughly 22% of secondary movement abnormalities, but aberrant movements may be seen as a result of an acute stroke in 1 – 4% of patients (Alonso et al., 2015; Caproni & Colosimo, 2017; Tater & Sanjay, 2021).

Cerebral small artery disease with tiny deep infarcts in the contralateral subthalamic nucleus or nearby structures is the most prevalent subtype of stroke that causes hemichorea-hemiballismus. The most common cause of vascular hemichorea, also known as hemiballismus, is an ischemic or hemorrhagic lesion of the basal ganglia and surrounding white matter in the region of the middle or posterior cerebral artery (Alonso et al., 2015; Caproni & Colosimo, 2017). However, reports of big vessel atherothrombotic stroke with cortical or watershed zone infarcts as well as cardiac cerebral embolism have also been made. A rare vasculopathy called isolated middle cerebral artery (MCA) dissection

can cause an ischemic stroke by mechanisms such as thromboembolism, hypoperfusion, or, very rarely, local branch blockage (Chen & Xu, 2020). Hemichorea, an uncommon manifestation of acute ischemic stroke, involves sudden, hyperkinetic movements and typically involves malfunctioning of the subthalamic nucleus and other basal ganglia structures. Either in the chronic phase of stroke involving the contralateral basal ganglia, particularly the subthalamic and lentiform nuclei, or in the acute phase of predominantly thalamic infarction as a result of deafferentation. Recently, areas causing chorea in strokes have included cortical ischemia lesions (Carbayo et al., 2020). A pertinent prevalence of hypertension, high cholesterol, obesity, diabetes, and smoking was noted in subjects without a history of vascular illness. Lower motor function and cognitive impairment were linked to the patient with two vascular risks (Caproni & Colosimo, 2017). Hemiballismus and hemichorea resulting from acute basal ganglia dysfunction are also frequently caused by poorly controlled diabetes because it raises the risk of cerebrovascular ischaemia and lowers cerebral blood flow (Cincotta & Walker, 2022).

The original theory that hemiballismus results from involvement of the subthalamic nucleus has been challenged by the

discovery of various different localizations, including the caudate nucleus, putamen, and thalamus. Most frequently, chorea is caused by vascular damage to the striatum, globus pallidus, or thalamus. Hemichorea is caused by regions in the thalamus and basal ganglia (Hao et al., 2015). A clinical-radiological correlation study revealed that the areas most frequently linked to hemichorea were the subthalamic nucleus, caudate, putamen, and cortical lesions on the side opposite the affected one. This localization is similar to that of hemiballismus, which is expected given the close phenomenological relationship between the two disorders. Ballismus and chorea brought on by vascular insults are often self-limited and gradually become better over time (Alonso et al., 2015). High-level motor control is mediated by a complex network involving the basal ganglia and subthalamic nucleus, which has reciprocal connections to a number of other brain areas. It is hypothesized that disruption of this functional link results in the recognizable hemichorea-hemiballismus movements (Chen & Xu, 2020). Lenticulo-striatal arteries carry the majority of the blood to the basal ganglia. These penetrating arteries have a modest diameter and branch out at a right angle from the middle cerebral artery's M1 segment. The basal ganglia are particularly susceptible to hypoxia or ischemia since

their distribution zone lacks a collateral blood supply and is functionally characterized as "terminal" (Guida et al., 2013). The regular basal ganglia circuitry transmission pattern would be interrupted by increased dopamine release brought on by ischemia. Acute circuitry dysfunction would cause excessive motor facilitation and a lack of thalamic movement control (Guida et al., 2013; Laganier et al., 2016). Less frequently, the cortex in the superficial area of the middle cerebral artery may also be affected by the stroke lesion (parietal, insular and temporal cortex) (Laganier et al., 2016).

Gamma-aminobutyric acid (GABA) transmission disruption from the striatum to the external globus pallidus (GPE) may be the pathogenesis of hemichorea caused by contralateral lesions of the striatal neurons of the indirect striato-thalamocortical pathways, which can then increase GPE neuronal activity and inhibit the subthalamic nucleus (Cincotta & Walker, 2022; Defebvre & Krystkowiak, 2016). Such inhibition would cause the internal globus pallidus (GPI) neurons to lose control, which could ultimately result in the motor thalamus's ability to inhibit movement. The same dysfunction, with a lack of motor thalamic inhibition, may be brought on by lesions of the subthalamic nucleus. An excitatory neural circuit from a portion of the frontal or parietal cortex (the

somatosensory cortex projecting into the caudate nucleus and putamen) must be disrupted in order to create AIMs in cases of hemichorea-hemiballism brought on by a cortical injury. Patients with cortical strokes have a functional prognosis that is significantly better than patients with subthalamic lesions; in the former group of patients, AIMs are likely brought on by transient hypoperfusion or a functional "disconnection" rather than by the breakdown of basal ganglia circuitry (Defebvre & Krystkowiak, 2016).

There have been numerous theories put forth: Although the indirect pathway only accounts for one-third of the overall population of motor striatal neurons, specific disruption of the indirect pathway is required to cause AIM. The striatal infarct is very large and also involves the pyramidal tract, which causes a motor deficit. Alternatively, the transient nature of these AIMs may be caused by regulation of the accessory striato-nigro-striatal, cortico-striato-nigro-thalamocortical, and cortical pathways, which are thought to regulate the indirect pathway (compensatory mechanisms) (Defebvre & Krystkowiak, 2016). Recent studies has been published detailing instances of ischemic strokes solely affecting the cerebral cortex and not the basal ganglia, leading to aberrant movements like chorea or ballism (Carbayo et al., 2020).

Post-stroke movement disorders can manifest with lesions affecting any segment of the motor circuitry; be it cortical which includes the primary motor, supplementary motor, and premotor cortical areas; or subcortical affecting the basal ganglia, thalamus, internal capsule, diencephalon, and mesencephalon; or cerebellar circuitry. The motor circuitry transmits information from the brain to the thalamus and back through its primary subcortical component, the basal ganglia. Two circuits make up the cerebellar circuitry: the GMT (Guillain-Mollaret triangle), also known as the dentate-rubro-olivary pathway, and the cortico-cerebello-cortical, also known as the dentate-rubro-thalamic pathway. Strokes that impact the subcortical regions are known to be more likely than cortical strokes to cause aberrant motions (Tater & Sanjay, 2021). Movement abnormalities are three times more common after subcortical strokes than after cortical strokes, with the basal ganglia (44%) and thalamus (33%) being most frequently affected. Movement difficulties following a stroke are uncommonly associated with cerebellar abnormalities that affect it alone (Hao et al., 2015; Tater & Sanjay, 2021).

Ischemic stroke diagnosis relied on skilled clinical assessment without explicit neuroimaging (Chen & Xu, 2020). While hemiballismus is characterized by violent irregular flinging movements of the limbs

brought on by contractions of the proximal muscles, hemichorea consists of continuous random, anarchic, and jerking movements involving both the distal and proximal muscles (though it is occasionally localized more distally) (Defebvre & Krystkowiak, 2016). Chorea is a symptom that is distinguished by sudden, uncontrollable movements brought on by an ongoing stream of erratically contracted muscles (Ueta et al., 2021). Ballism is a type of high-amplitude flinging chorea, which is characterized by short, arrhythmic movements that seem to flow from one muscle to the next (Caproni & Colosimo, 2017). While chorea frequently affects the entire body and is widespread, it can also be noticeably asymmetrical or even unilateral in a considerable number of circumstances. While stroke is the most prevalent cause of unilateral involvement and is traditionally associated with a contralateral anatomical lesion, such as one of the putamen or the subthalamic nucleus, systemic disease can also result in unilateral or noticeably asymmetric presentations (Chen & Xu, 2020).

The involvement of several extra-striatal cortical areas and subcortical regions in movement disorders has been demonstrated by a number of functional neuroimaging studies based on positron emission tomography and resting state functional magnetic resonance imaging, emphasizing

a role of network dysfunction and abnormal functional connectivity in these conditions (Caproni & Colosimo, 2017). The mean age of onset was lower (35 years) in cases of other major causes of hemichorea-hemiballism (abscesses, metastatic lesions, AIDS, levodopa medication, Sydenham's chorea, neonatal anoxic brain injury, multiple sclerosis, and central nervous system lupus) than in the stroke subgroup (61 years) (Defebvre & Krystkowiak, 2016). This type of AIM should be treated as a neurological emergency and treated as soon as feasible in a stroke center because any delay in diagnosis may have detrimental effects on the patient's clinical and therapeutic management. Patients with ischemic or hemorrhagic stroke, as well as those with cerebrovascular abnormalities and dural arteriovenous fistulas, can exhibit AIMs (Defebvre & Krystkowiak, 2016). Any anatomo clinical correlations will be easier to make if the vascular lesion is confined and has clearly defined limits. Numerous reports of clinicopathological correlations based on both single instances and short series of patients with lesions in the aforementioned structures have been generated by computed tomography (CT) and magnetic resonance imaging (MRI) examinations (Defebvre & Krystkowiak, 2016).

In 85% of cases, AIM started immediately the day of the stroke, but they

can sometimes deteriorate progressively over many weeks or come back after a latent period of several months. They are noticed on the side opposite the stroke; only when bilateral basal ganglia lesions are seen on brain MRI do bilateral symptoms become apparent. Usually, both the upper and lower limbs are affected, but occasionally, only one body portion is (neck, arm or leg) (Defebvre & Krystkowiak, 2016). Hemichorea typically appears within a few hours of the start of the stroke (as in our patient's case), although delays of up to five days have also been documented. The idea of pathogenesis mediated by restorative neuroplasticity has been brought forward as a result of this delay (Guida et al., 2013; Ueta et al., 2021). Hemichorea appears to happen a few days after stroke, whereas hemiballism is typically seen to happen promptly with the onset of stroke. However, there have been cases where a delay of up to five months was reported (Tater & Sanjay, 2021).

Some treatment options are shared by vascular movement disorders and idiopathic variants of these illnesses (Caproni & Colosimo, 2017; Carbayo et al., 2020). All patients with acute ischemic stroke who meet the requirements for this treatment within 4.5 hours of stroke start are advised to receive thrombolytic therapy (Bembenek et al., 2015). In rats with ischemic stroke, piracetam has been shown to improve brain

penetration and drastically reduce infarct volume (Paliwal et al., 2018). Animal experiments showed that piracetam could help humans who are suffering from an acute stroke (Tortiglione et al., 2002). A meta-analysis of research using rat models of cerebral ischemia and stroke provided more evidence for piracetam's potential value (Wheble et al., 2008).

Hemichorea/hemiballism following a stroke frequently goes away on its own, thus a wait-and-see approach is recommended. The majority of instances resolve on their own, but when movement is significant and harmful enough to cause injury, pharmaceutical therapy is required (Tater & Sanjay, 2021). A symptomatic approach may be helpful for some individuals with hemichorea/ hemiballism whose involuntary movements have a major impact on their quality of life, increase their risk of falling, and reduce their independence in daily tasks (Caproni & Colosimo, 2017; Tater & Sanjay, 2021). The most popular treatment option is typical and atypical neuroleptics, which block dopamine receptors using typical and atypical neuroleptics and catecholamine-depleting drugs (Caproni & Colosimo, 2017; Tater & Sanjay, 2021).

Haloperidol, pimozide, perphenazine, and fluphenazine are examples of common neuroleptic medications that function by inhibiting dopamine receptors (D1 and D2)

(Tater & Sanjay, 2021). Because they inhibit the D3 and D4 dopamine receptors, the atypical neuroleptic medications olanzapine, quetiapine, and sulpiride are less likely to result in tardive dyskinesia and parkinsonism. Clozapine has been used in refractory instances, however it produces agranulocytosis. Tetrabenazine depletes presynaptic dopamine by blocking postsynaptic dopamine receptors and inhibits brain synaptic vesicular monoamine transporter type 2 (VMAT2). Reserpine has a presynaptic impact as well, but it also has negative side effects such severe depression, suicidal thoughts, hypotension, and parkinsonism. Clonazepam and sodium valproate are two more GABAergic medications that have been tried (Caproni & Colosimo, 2017; Chen & Xu, 2020; Tater & Sanjay, 2021). There have been reports of antiepileptic medications including clonazepam, sodium valproate, and topiramate being successful in tiny uncontrolled numbers of patients (Caproni & Colosimo, 2017). Surgery may be an alternate form of treatment for rare, severe, and persistent instances when pharmaceutical medicines are ineffective or when a patient is unable to tolerate medical therapy (Caproni & Colosimo, 2017; Tater & Sanjay, 2021). Surgery such as stereotactic ventral intermediate thalamotomy and continuous thalamic stimulation can be used to effectively treat

refractory hemichorea and hemiballism (Caproni & Colosimo, 2017; Tater & Sanjay, 2021).

Immediately after a stroke, abnormal involuntary motions start to appear and may eventually go away on their own. This usually happens within a few hours or days (Siniscalchi et al., 2012). In six months, up to 90% of these movement abnormalities with abrupt onset may go away (Caproni & Colosimo, 2017). Minimal functional impairment may linger in post-ischemic hemichorea-hemiballismus. The prognosis for syndromes connected to cortical ischemia localizations is believed to be better (Guida et al., 2013).

Conclusion

Even though they are uncommon, following a stroke, abnormal involuntary movement can occur suddenly or develop gradually. It can be either hypokinetic or hyperkinetic (most frequently hemichorea-hemiballismus) (most commonly vascular parkinsonism). Strokes can happen anywhere throughout the motor circuit, although the majority are brought on by lesions in the basal ganglia or thalamus. Although many are self-limiting, symptom management may necessitate therapy. The growing accessibility of sophisticated structural and functional neuroimaging methods may be crucial in advancing knowledge of vascular movement

disorders. Hemichorea-hemiballismus with abrupt onset should be treated as an acute stroke unless proven differently.

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