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CASE REPORT **OPEN ACCESS**

ACUTE HEMIBALLISMUS DUE TO VASCULAR OR DIABETIC STRIATOPATHY: A RARE CASE REPORT FROM KHZ MUSTHAFA GENERAL HOSPITAL, TASIKMALAYA

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ABSTRACT

Background: Hemiballismus is a hyperkinetic movement disorder characterized by sudden, involuntary, high-amplitude ballistic movements of the arm and leg on one side of the body, often due to contralateral central nervous system dysfunction. The most common causes are ischemic stroke and hyperosmolar hyperglycemic syndrome. Stroke-related movement disorders are rare, with an incidence of 0.4%–0.54%, while the prevalence of hyperglycemia-induced, known as diabetic striatopathy (DS), affects approximately 1 in 100,000 individuals, predominantly older women.
Case: A 63-year-old male presented with sudden, involuntary left arm movements for one week, starting with throwing-like motions, followed by stiffness, tremors, rapid arm drop, and facial grimacing on the left side. The condition was accompanied by left leg weakness. He had a history of uncontrolled type 2 diabetes mellitus. Physical examination showed left hemiballismus with motor strength 5/5/3/2 and a positive Babinski reflex on the left. Blood tests revealed a random glucose of 686 mg/dL, HbA1C of 15%, and 126 mmol/L sodium. A non-contrast CT scan showed multiple bilateral lacunar infarcts in the basal ganglia region.
Discussion: Hemiballismus occurs sporadically and is caused by multiple bilateral lacunar infarcts in the basal ganglia, with risk factors including type 2 diabetes mellitus and advanced age. Other causes, such as stroke and genetic factors, may still be considered.
Conclusion: Hemiballismus is a rare form of movement disorder. Vascular and metabolic disorders are the most common causes of hemiballismus. In this case, acute hemiballismus occurred in an elderly stroke patient with type 2 diabetes mellitus as a risk factor.

Keywords: diabetic striatopathy, hemiballismus, lacunar infarct stroke, type-2 diabetes mellitus

Introduction

Hemiballismus is a hyperkinetic movement disorder characterized by intermittent, sudden, coarse, involuntary, high-amplitude throwing or ballistic movements involving the arm and leg on the same side of the body, caused by dysfunction of the central nervous system on the opposite side.[1] The most common causes are ischemic stroke and hyperosmolar hyperglycemic syndrome.[2,3] The basal ganglia are a group of subcortical nuclei located within the cerebral hemispheres. There are direct pathways (cerebral cortex–striatum–internal globus pallidus/substantia nigra pars reticulata–ventral thalamus–cerebral cortex) and indirect pathways (cerebral cortex–striatum–external globus pallidus (GPe)–subthalamic nucleus–internal globus pallidus (Gpi)–ventral thalamus–cerebral cortex) that regulate motor excitation and inhibition.[4] The putamen is a common site of hemorrhage due to hypertension or ischemia.[5] Stroke is a syndrome of acute focal or global neurological deficits, clinically defined as a consequence of vascular injury (ischemic or hemorrhagic) to the central nervous system, with risk factors that are either non-modifiable, such as age, or modifiable, such as diabetes mellitus.[6]

As age increases, both the microcirculation and macrocirculation of the brain undergo structural and functional changes.[7] The incidence of movement disorders as a manifestation of stroke is infrequent, occurring in 0.4%–0.54% of cases, with hemichorea due to acute ischemic stroke specifically occurring in about 1% of cases.[1,8]

Diabetic striatopathy (DS) is a condition of hyperglycemia accompanied by one or both of the following manifestations: acute-onset chorea-ballism, striatal hyperdensity on computed tomography (CT), or striatal hyperintensity on T1-weighted magnetic resonance imaging (MRI).[9,10] According to several previous studies, patients with chorea-ballism movements do not have supportive neuroimaging findings in 5–45% of cases.[3,11,12] Chang et al. classified hemichorea-hemiballismus symptoms into two types: common and uncommon.[13] In non-ketotic hyperglycemia, brain metabolism shifts to an alternative anaerobic pathway in the Krebs cycle, leading to a rapid decrease in GABA (gamma-aminobutyric acid), which results in the disinhibition of the subthalamus and basal ganglia, causing hyperkinetic movements in SD patients.[3,14] The prevalence of SD is 1 per 100,000, primarily affecting elderly female patients.[1,3]

Case Report

A 63-year-old male presented to the hospital with sudden, intermittent, involuntary movements of his left arm. The episode began with a throwing-like motion, followed by stiffness and tremors, then a rapid drop of the arm, accompanied by left-sided facial grimacing. These symptoms had been occurring for one week. The complaint was also accompanied by left leg weakness. The patient has a history of uncontrolled type 2 diabetes mellitus (DM). Physical Examination: Findings revealed left-sided hemiballismus with motor strength of 5/5/3/2, and a positive left Babinski reflex. Laboratory tests showed a random blood glucose level of 686 mg/dL, HbA1C of 15%, and sodium level of 126 mmol/L. A non-contrast head CT scan revealed multiple bilateral lacunar infarcts in the basal ganglia region (Figure 1).

Neurology treatment: Mecobalamin IV 500 mcg/8 hours, citicoline IV 1 gram/12 hours for 3 days, valproic acid 250 mg orally/12 hours, diazepam IV 2 mg/12 hours, Proton Pump Inhibitor IV 40 mg/24 hours, aspirin 80 mg orally/24 hours, nattokinase 10 mg orally/24 hours, and pregabalin IV 37.5 mg/12 hours for stroke and movement disorder management. Internal medicine treatment: Insulin glargine 16 units SC at night (0-0-0-16 U), and insulin aspart 12 units SC three times a day (3dd12 U) for blood glucose regulation.

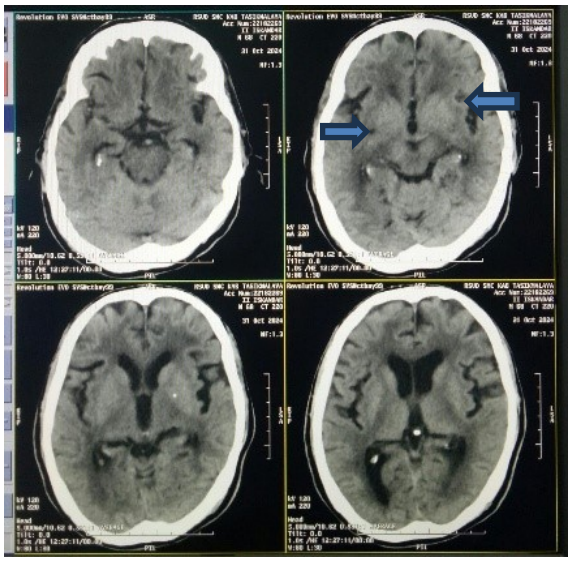

Figure 1. Non-contrast head CT scan showing multiple bilateral lacunar infarcts in the basal ganglia region (arrow)

Discussion

In this case, a 63-year-old male experienced movement disorders, with his left arm moving involuntarily, suddenly, in a throwing-like motion, followed by a rapid drop of the hand, which is characteristic of hemiballismus. Hemiballismus is a hyperkinetic movement disorder characterized by intermittent, sudden, coarse, involuntary throwing or ballistic movements with high amplitude involving the arm and leg on the same side of the body, caused by dysfunction of the central nervous system on the opposite side. Hemiballismus represents the most severe form within the chorea spectrum.[1] Chorea is an involuntary movement disorder characterized by brief, sudden, spontaneous, non-rhythmic, dance-like movements on one side of the body.[1] The causes of chorea can be classified based on the time of onset, age, and prevalence to facilitate diagnosis.[15] Acquired or sporadic causes of chorea typically have an acute or subacute temporal profile, including vascular (ischemic or hemorrhagic stroke), metabolic (non-ketotic hyperosmolar hyperglycemia, hypo/hypernatremia, uremia, hyperthyroidism), infectious (toxoplasmosis, HIV encephalopathy), and drug-induced causes (phenytoin, dopamine agonists used in Parkinson's disease), which are more common in the elderly. In contrast, Sydenham chorea occurs in children, while genetic causes tend to be chronic (lasting more than one year), as seen in patients with Huntington's disease.[1,15,16] Vascular and structural lesions in the contralateral subthalamic nucleus and basal ganglia are the most common causes of hemiballismus chorea.

The basal ganglia are a group of subcortical nuclei located within the cerebral hemispheres. The most significant component of the basal ganglia is the corpus

striatum, which consists of the caudate nucleus and the lentiform nucleus (putamen, GPe, and GPi), STN, and the SN.[4] These structures are interconnected in a complex manner to facilitate or inhibit movement. Two pathways regulate the balance of basal ganglia function. The direct pathway involves the cerebral cortex sending excitatory signals (glutamate) to the striatum (caudate nucleus and putamen). The activated striatum then sends inhibitory GABA signals to the GPi/SNpr (substantia nigra pars reticulata). However, dopamine from the nigrostriatal pathway synapses on striatal neurons expressing D1-family receptors. Excited The dopamine D1 receptor (D1R) neurons send inhibitory projections that inhibit the GPi/SNpr, resulting in the disinhibition of the thalamus, which then sends excitatory signals to the motor cortex, promoting movement. The indirect pathway inhibits motor activity. Dorsal striatal neurons expressing D2-family receptors are inhibited by dopamine from the SN. The dopamine D2 receptor (D2R) neurons send inhibitory GABAergic connections to the GPe, inhibiting the STN. Normally, the STN excites the GPi/SNpr, which sends inhibitory signals to the thalamus, ultimately reducing excitation to the motor cortex.[4] The striatum is often involved in hyperkinetic movement disorders.[17] Lesions in the striatum disrupt GABA transport to the GPe, impairing STN inhibition.[17] These changes can lead to losing control over GPi inhibition of thalamic output.[17] The putamen is a common site for both hypertensive hemorrhage and ischemia.[5] The spiral-shaped pattern of lenticulostriate arteries (which increases intraluminal pressure) and the formation of Charcot-Bouchard aneurysms due to fibrinoid necrosis make it prone to rupture.[5] Conversely, lipohyalinosis and microatheroma formation are the primary causes of infarction.[18] The clinical manifestations of small strokes in the putamen can be categorized as follows) Mixed motor and sensory, 2) Pure motor, 3) Pure sensory, 4) Ataxic hemiparesis, 5) Dysarthria with a clumsy hand, and 5) Hemiballismus and hemichorea.[18]

In this case, the patient experienced multiple bilateral lacunar infarcts in the basal ganglia. Stroke is a syndrome of acute neurological deficits, either focal or global, clinically defined as a consequence of vascular injury (ischemic or hemorrhagic) in the central nervous system.[6] Stroke risk factors include non-modifiable factors (age, sex, ethnicity, genetics) and modifiable factors (hypertension, diabetes mellitus, heart disease, smoking, hyperlipidemia, obesity, and inflammation). In this case, the patient had uncontrolled type 2 diabetes mellitus for the past five years, along with advanced age, which are risk factors for ischemic stroke. As age increases, both microcirculation and macrocirculation in the brain undergo structural and functional changes. Age-related microcirculatory changes are mediated by endothelial dysfunction, impaired cerebral autoregulation, and neurovascular coupling dysfunction. Endothelial dysfunction leads to neuroinflammation, impaired cerebral autoregulation can result in microvascular injury, and neurovascular dysfunction contributes to cortical function decline.[6,7] The incidence of movement disorders as a manifestation of stroke is rare, occurring in 0.4%–0.54% of cases, with hemichorea due to acute ischemic stroke accounting for approximately 1%.[1,8] Hemichorea is associated with ischemia involving the middle cerebral artery and posterior cerebral artery, which supply blood to the basal ganglia (caudate nucleus, putamen, globus pallidus) and the subthalamic nucleus. This results in hypoperfusion-related dysfunction of the basal ganglia pathway and the frontal motor cortex area.[19–22]

Diabetic striatopathy is a condition of hyperglycemia accompanied by one or both manifestations: acute-onset chorea-ballism; striatal hyperdensity on CT; or striatal hyperintensity on T1-weighted MRI.[9,10] According to several previous studies, patients with chorea-ballism movements do not have supportive neuroimaging findings in 5–45% of cases.[3,11,12] 2% of patients may show radiological striatal lesions without clinically manifested movement disorders, even leading to clinical-radiological discrepancies in DS. Thus, striatopathy with clinically manifested movement disorders (symptomatic DS) can be categorized into two groups: 1) Concordant: Neuroimaging lesions are contralateral to the movement disorder 2) Discordant: Neuroimaging lesions are ipsilateral to the movement disorder, neuroimaging shows unilateral lesions with bilateral movement disorders, or neuroimaging shows bilateral lesions with unilateral movement disorders.[9] Classified hemichorea-hemiballismus symptoms into common and uncommon. The standard type is the most frequently observed. It is described in diabetic patients with elevated blood sugar levels, negative urine ketones, unilateral or bilateral chorea, and abnormalities detected on MRI and CT scans of the brain. The uncommon type occurs in diabetic patients with elevated blood sugar levels and negative urine ketones, presenting with unilateral or bilateral chorea but without abnormalities on MRI or CT scans of the brain.[13] In this case, the patient's blood sugar level increased to 686 mg/dL, accompanied by an HbA1C of 15%, indicating uncontrolled hyperglycemia, which can be one of the criteria for DS. However, the patient did not show characteristic lesions consistent with DS criteria, making it still possible for this case to be classified as an uncommon type of DS. Based on the medical record history, the patient has consistently experienced hyperglycemia during internal medicine outpatient visits in the past year. Still, the hemiballismus condition occurred for the first time when the patient had a stroke.

Diabetic striatopathy is caused by vascular insufficiency, blood-brain barrier dysfunction, neurotransmitter changes, such as GABA, and increased dopaminergic activity.[23] In non-ketotic hyperglycemia, brain metabolism shifts to an alternative anaerobic pathway in the Krebs cycle, leading to a rapid decrease in GABA, which results in the disinhibition of the subthalamus and basal ganglia, causing hyperkinetic movements in DS patients.[3,14] In ketosis, GABA can be resynthesized using acetoacetate produced in the liver, making DS rare in cases of diabetic ketoacidosis.[24] Hyperglycemia induces hyperosmolarity and hyperviscosity, leading to decreased cerebral blood flow, which causes damage to striatal astrocytes that are highly sensitive to ischemia. These swollen reactive astrocytes, known as gemistocytes, are the most consistent findings observed in a limited number of biopsy studies.[3,14]
DS is a rare condition, with a prevalence of 1 per 100,000 cases in elderly female diabetic patients.[3] A systematic review conducted by Chua et al. in 2020 on 176 patients showed that most reported cases of DS come from Asian countries.[3] Acute-onset movement disorders typically occur in patients within the sixth to seventh decade of life.[3,11] Various studies have recorded that the incidence of hemichorea-hemiballism due to hyperglycemia is twice as common in women, likely due to increased dopaminergic receptor sensitivity caused by estrogen deficiency in the striatum of postmenopausal women.[3,12,14] Based on the affected body areas, the highest frequency, in order, is arm-leg, arm-leg-face, and arm alone.[14,25]

Movement disorder symptoms can be managed medically using first- and second-generation antidopaminergic therapy targeting D2 receptors (risperidone, haloperidol, perphenazine, pimozide, chlorpromazine), benzodiazepines (clonazepam), antiepileptics (topiramate), and tetrabenazine to alleviate severe hyperkinetic movements. Other anti-chorea medications include tiapride, quetiapine, pimozide, diazepam, and valproate.[3,26] The use of benzodiazepines has been reported in previous studies for patients with chorea and ballism associated with non-ketotic hyperglycemia. In this case, the patient was given benzodiazepines, specifically diazepam and valproic acid, for movement disorder therapy. Hyperglycemia treatment involves insulin administration and ensuring blood glucose levels are well controlled. In DS, complete symptom resolution with euglycemia is achieved in approximately 25% of patients, and the duration of symptoms is often brief, sometimes lasting only a few hours to days, with an average recovery time of 2 days.[3,26] If the patient is resistant to medical management, surgical procedures such as posteroventral stereotactic pallidotomy and intrathecal baclofen administration may be considered.[14]

Conclusion

Hemiballismus is a rare form of movement disorder. Vascular and metabolic disorders are the most common causes of hemiballismus. In this case, acute hemiballismus occurred in an elderly stroke patient with type 2 diabetes mellitus as a risk factor.

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