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A CASE OF LEFT-SIDED HEMIBALLISMUS WITH DYSARTHRIA: MULTIDISCIPLINARY APPROACHES FOR RECOVERY

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ABSTRACT

A rare hyperkinetic movement condition called hemiballismus is typified by powerful, involuntary, and ballistic limb motions. It is usually brought on by injuries in the basal ganglia on the opposite side. We report the case of a 65-year-old man who has dysarthria, poorly managed diabetic mellitus, and coronary artery disease. He also has left-sided hemiballismus. The left inferior cerebellar hemisphere had persistent ischemic alterations and gliosis on CT and MRI, most likely as a result of an earlier stroke. A multidisciplinary strategy was used to treat the patient, involving the use of insulin for glycemic control and drugs such as tetrabenazine, clonazepam, and haloperidol to control hyperkinetic movements. The patient showed notable improvement in his neurological condition during his stay in the hospital. This case study emphasizes to treat hemiballismus by controlling vascular risk factors and glycemic management, as well as by conducting regular follow-ups to ensure the best outcomes.

Keywords: hyperkinetic movement condition, hemiballismus, ballistic limb motions

INTRODUCTION:

Hemiballismus is a hyperkinetic involuntary movement condition that affects the ipsilateral arm and leg. It is characterized by sporadic, abrupt, forceful, involuntary, flinging, or ballistic high amplitude motions that are brought on by dysfunction in the contralateral side's central nervous system **[1]**. Lesions in the brainstem, corpus striatum, cerebral cortex, thalamus, and subthalamic nuclei can explain ballismus **[2]**. The etiology of hemiballismus is varied, with the most common cause being ischemic stroke. Other potential causes include hemorrhage, traumatic brain injury, tumors, infections, metabolic abnormalities (such as hyperglycemia), and rarely, demyelinating diseases or drug-induced cases **[3]**. Among these, non-ketotic hyperglycemia is an increasingly recognized metabolic cause, particularly in elderly patients with poorly controlled diabetes mellitus **[4]**. Although the exact incidence and frequency of ballismus and chorea syndromes are unknown, they are thought to be between 1 and 2 per 1,000,000 worldwide **[5]**. The pathophysiology of hemiballismus involves disruption of the indirect basal ganglia pathway, where damage to the subthalamic nucleus reduces excitatory input to the globus pallidus internus. This leads to excessive thalamic stimulation of the motor cortex, resulting in the characteristic hyperkinetic movements **[5]**. While hemiballismus was historically associated with poor prognosis, most cases are now known to resolve spontaneously or with medical treatment. In medical management, medications targeting D2 receptors (e.g., risperidone, haloperidol, perphenazine, pimozide, chlorpromazine) are used, along with benzodiazepines (clonazepam), anti-epileptics (topiramate), and tetrabenazine to relieve severe hyperkinetic movements **[6]**. Since hemiballismus is caused by disruption to the basal ganglia's inhibitory circuits and usually goes away on its own with symptomatic treatment, treatment-related complications are uncommon. In acute, life-threatening etiologies such as an ischemic stroke, treating the underlying etiology is essential to preventing additional complications and functional decline.

CASE REPORT:

A 65-year-old male presented to the hospital with a three-day history of involuntary movements in his left upper and lower limbs. He reported that these movements were associated with a reduced intensity of movement in the right upper limb, trunk, and right lower limb, exacerbating during exertion and alleviating during sleep.

Additionally, the patient experienced slurring of speech but denied any fever, loss of consciousness, vomiting, limb weakness, giddiness, or headache. He had diabetic mellitus (DM), systemic hypertension (SHT), and coronary artery disease (CAD) that was treated with percutaneous coronary intervention (PCI) but with irregular follow-up. Upon neurological examination, he was conscious, alert, and oriented, dysarthria was present. Motor examination revealed 5/5 strength in all four limbs, along with left hemiballismus, and no cranial nerve involvement. Left cerebellar signs were also present.

Investigations included a CT scan of the brain with contrast, which showed no acute infarcts or hemorrhages but indicated encephalomalacic changes with gliosis in the left inferior cerebellar hemisphere, likely due to a chronic infarct, along with chronic small vessel ischemic changes. Routine blood investigations revealed a HbA1c of 12.1%, urea of 41 mg/dL, creatinine of 1.5 mg/dL, and normal electrolytes and liver function tests. The patient's hemoglobin was 12 g/dL, packed cell volume was 34.9%, and his white blood cell count was 8.2 x 10^9/L. A consultation with an endocrinologist was requested, leading to the initiation of a tailored insulin regimen: Inj. Lupisulin M 30/70 at 16 units in the morning and 14 units in the evening, and Inj. HIR 7 units in the afternoon and adjusted based on blood sugar levels. Due to the persistence of left hemiballismus, MRI was performed, confirming chronic infarct with gliotic changes in the left inferior cerebral hemisphere, age-related cerebral atrophy, and chronic small vessel ischemic changes, with no signs of acute infarction.

During his hospital stay, the patient received a comprehensive treatment regimen, including IV fluids, Inj. Pantoprazole 40 mg once daily for 5 days, Inj. Ondansetron 4 mg as needed for nausea, Tab. Ticagrelor 90 mg twice daily for 5 days, Tab. Aspirin 75 mg once daily for 5 days, Tab. Atorvastatin 40 mg at bedtime for 5 days, Tab. Clonazepam 0.25 mg at bedtime for 2 days, Inj. Sodium Valproate 200 mg twice daily for 4 days, and the previously mentioned insulin regimen for 5 days. Other medications included Tab. Glycomet SR 500 mg twice daily for 5 days, Tab. Haloperidol 0.25 mg half tablet three times daily for 2 days, Tab. Clonazepam 0.5 mg half tablet three times daily for 3 days, Injection was switched on to Tab. Sodium Valproate 250 mg twice daily for rest 3 days, Inj. Lorazepam 2 mg at bed time, and Tab. Tetrabenazine 25 mg half tablet at bedtime for 2 days, then once daily for another 2 days. The patient was also given Tab. Haloperdidol 0.25

mg three times daily for 5 days, Syrup Cremaffin Plus 30 ml at bedtime for 2 days, Tab. Itopride 50 mg twice daily for 3 days, Tab. Paracetamol 1 gm once daily for 3 days, and Actibis oil three times daily for 2 days.

Throughout his hospitalization, the patient demonstrated gradual neurological improvement, with a significant reduction in left hemiballismus. He was ultimately discharged with a detailed medication regimen and advice to maintain regular follow-ups with his healthcare providers, particularly a neurologist, to monitor his condition and adjust treatments as necessary. This case underscores the importance of a multidisciplinary approach in managing hemiballismus secondary to chronic ischemic changes, particularly in patients with multiple comorbidities.

DISCUSSION:

Hemiballismus is a hyperkinetic movement disorder that usually affects one side of the body and is characterized by tossing, forceful, and involuntary movements of the limbs. The disease is caused by disruption of the contralateral basal ganglia circuits, mainly as a result of damage to the subthalamic nucleus. The patient's left-sided hemiballismus in this instance is consistent with the origin of the movement condition and suggests underlying dysfunction in the right basal ganglia **[7]**.

Hemiballismus can have a wide range of etiologies, but the most prevalent cause is ischemic stroke, as this patient had a history of poorly controlled diabetes mellitus and cardiovascular disease. The CT and MRI results indicate that the patient's hemiballismus may have been caused by a chronic infarct in the left inferior cerebellar hemisphere, which resulted in gliotic abnormalities. These changes are indicative of chronic ischemic changes. This emphasizes how crucial it is to assess patients for stroke history and vascular risk factors when hemiballismus is suspected.

This patient's hemiballismus was accompanied by various neurological symptoms, including dysarthria and decreased motor function on the opposite side. This comprehensive presentation highlights the importance of a thorough neurological assessment because coexisting illnesses like dysarthria can make diagnosis and treatment more difficult.

The usual approach to controlling hemiballismus is to address the underlying cause; in this instance, managing the patient's vascular risk factors and achieving optimal glycemic control. In this patient, the start of a customized insulin regimen was critical,

underscoring the importance of endocrinology in the multidisciplinary treatment of such complicated situations. Furthermore, it was appropriate to employ medication to target hyperkinetic movements. Hemiballismus symptoms were treated with drugs such tetrabenazine, sodium valproate, clonazepam, and haloperidol. The selection of these drugs is in keeping with current recommendations, which suggest benzodiazepines and antipsychotics as first-line therapies for hyperkinetic movement disorders.

The patient's steady improvement throughout the course of his hospital stay supports the idea that hemiballismus can resolve with the right medical care and that, in most cases, hemiballismus has a good prognosis when the underlying reasons are successfully treated. This is consistent with the research, which indicates that even though hemiballismus has traditionally been linked to unfavorable outcomes, many instances show notable improvements after receiving therapy.

Furthermore, considering the possibility of recurrence and the danger of consequences from underlying comorbidities, it is imperative to continue following up with a neurologist. Frequent monitoring can improve overall patient outcomes and enable prompt modifications to the treatment plan.

CONCLUSION:

In summary, this case highlights the intricate relationship that exists between vascular illness and mobility difficulties in elderly patients who have a variety of comorbidities. To maximize therapy and improve functional outcomes for patients with hemiballismus, a multidisciplinary approach is essential. Future studies should concentrate on improving treatment approaches for this difficult condition and comprehending the pathophysiological principles behind hemiballismus.

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