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IDENTIFYING ACUTE ISCHEMIC STROKE WITH HEMICHOREA AS A CLINICAL MANIFESTATION: A CASE REPORT

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ABSTRACT

Focal brain lesions can induce abnormal involuntary movement disorders, including hemichorea, hemiballismus, parkinsonism, myoclonus, dystonia, tremor, and asterixis. The most common cause of focal brain lesions is stroke, followed by trauma, neoplasm, anoxia, vascular malformations, metabolic disease, and multiple sclerosis. Hemichorea-hemiballismus as an initial presentation of acute ischemic stroke is very rare, with an incidence rate of 0.54%. **Case Presentation:** A 65-year-old male with a past medical history of hypertension and diabetes experienced sudden, involuntary, non-rhythmic, and uncontrollable movements of the left extremity one day before being admitted to the hospital. A non-contrast CT scan of the head revealed acute cerebral infarction on the right basal ganglia. The symptoms of hemichorea improved with the initiation of haloperidol after three days of treatment. Regarding this case, acute ischemic stroke should be considered suppose a patient without hyperglycemia and with sudden onset of hemichorea visits to emergency room.

Keywords: *hemichorea, stroke, ischemic stroke, haloperidol, movement disorders*

BACKGROUND

Focal brain lesions can cause various abnormal movement disorders including hemichorea, hemiballismus, parkinsonism, myoclonus, dystonia, tremor, and asterixis. The most common of these lesions is stroke, followed by other causes such as trauma, neoplasm, anoxia, vascular malformations, metabolic diseases, or multiple sclerosis.

Movement disorders are often unilateral and contralateral to the focal brain lesion. Bilateral manifestations are more common in the metabolic causes including the hyperglycemic conditions. Movement disorder usually occurs because of lesions in the striatopallidal complex, mesencephalon, thalamus, and rarely in the cortex.[1,2]

Hemichorea consists of continuous, random, jerky movements involving proximal and distal muscles (sometimes more distal), while hemiballismus is characterized by irregular, violent flinging movements due to proximal muscle contractions.[2] Hemichorea-hemiballismus as an initial presentation of acute ischemic stroke is very rare, with an incidence rate of 0.54%.[3] Infarct lesions are usually subcortical with or without involvement of the basal ganglia. However, a pure cortical stroke causing hemichorea-hemiballismus is rare, likely due to disturbances in inter-cortical and/or cortical-ganglionic connections. Despite the lesion's location, the affected tissues remain consistent.[4] Despite the rarity of this occurrence, we encountered a rare manifestation of hemichorea in acute stroke patients, which piqued our interest in presenting this case.

CASE PRESENTATION

A 65-year-old man visited the emergency room (ER) with sudden abnormal movements in his left upper and lower extremities one day before admission. The patient exhibited involuntary, non-rhythmic, and uncontrollable movements. He was conscious, with normal vital signs and no other neurological deficits.

His medical history included hypertension and diabetes mellitus. At the time of admission, his blood pressure was 130/80 mmHg, and his random blood glucose was 178. A non-contrast CT scan of the head revealed acute cerebral infarction in the right basal ganglia, particularly the globus pallidus. Metabolic, infectious, and toxic etiologies of encephalopathy were ruled out based on the parameters and clinical condition including the normal blood glucose level, absence of signs of infection, and history of substance consumption.

The patient was initially treated with the clopidogrel 75mg per day, and the neuroleptic drug haloperidol 0.5mg twice daily. The patient's hemichorea improved with the initiation of haloperidol during three days of treatment, and he was discharged on the seventh day of hospitalization.

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DISCUSSION

Hemichorea can arise from several etiologies, with vascular chorea being the third most frequent cause, occurring in 9% of cases. Vascular etiology should be considered when patients visit with acute and unilateral clinical manifestations. Moreover, delayed manifestations (after months or years) can still result from a stroke.[5] In some patients, chorea becomes intermittent and often resolves spontaneously in more than 50% of cases. However, it can be moderate to severe and disabling in others.[2]

The pathogenesis of hemichorea involves the disruption of gamma-aminobutyric acid (GABA) transmission from the striatum to the external globus pallidus (GPE), resulting from a contralateral striatal lesion in the indirect striatal-thalamocortical pathway. This may lead to increased GPE neuronal activity and inhibition of the subthalamic nucleus, causing a loss of control by the internal globus pallidus (GPI) and subsequent disinhibition of motor thalamus movements. Similar dysfunction with motor thalamus disinhibition can be caused by subthalamic nucleus lesions.[5] Disruption of excitatory circuits from the frontal or parietal cortex (somatosensory cortex projecting to the caudate nucleus and putamen) is necessary to induce movement disorders in hemichorea-hemiballismus caused by cortical injury. Patients with cortical stroke typically have a significantly better functional prognosis than those with subthalamic lesions; for the former, movement disorders are likely due to transient hypoperfusion or functional "disconnection" rather than damage to basal ganglia circuitry.[2,6]

A large striatal infarction involving the pyramidal tract can cause motor deficits. Conversely, the transient nature of this movement disorder may be due to the regulation of accessory pathways (striatonigro-striatal, cortico-striato-nigro-thalamocortical, and cortico-subthalamic). Selective disruption of the indirect pathway is necessary to induce chorea, even though the indirect pathway represents only one-third of total motor function. Studies indicate that the prognosis of hemichorea caused by stroke depends on the lesion's location. About 50% of cases with basal ganglia circuit infarcts achieve complete resolution, while subthalamic lesions can result in persistent disorders.[2,6] In our patient, the CT scan showed an acute cerebral infarction in the right basal ganglia, which caused his movement disorder and significantly improved by the third day of hospital treatment. This supports the theory that basal ganglia infarcts have better outcomes. In our patient, the lesion occurred in the right globus pallidus, leading to the disruption of GABA transmission from the striatum.

Tetrabenazine may be beneficial for chorea management without the risk of tardive dyskinesia. Neuroleptic drugs (both old and new generations) can be administered short-term to inhibit dopamine receptors.[2,6] The patient's hemichorea significantly improved after three days of haloperidol treatment.

One differential diagnosis for hemichorea is non-ketotic hyperglycemia hemichorea, characterized by involuntary, random, continuous, and proximal movements on one side of the body contralateral to the affected basal ganglia during non-ketotic hyperglycemia. Rapid symptom resolution occurs after blood glucose normalization with insulin and hydration; however, our patient did not have elevated blood glucose.[1]

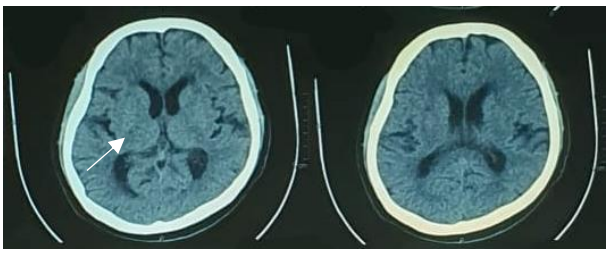

Figure 1. Head CT scan showed acute cerebral infarct at the right basal ganglia (white arrow).

CONCLUSION

Acute ischemic stroke should be considered for patients with sudden onset hemichorea. Rapid recognition of this movement disorder in the appropriate clinical setting can reduce diagnostic delays and minimize functional impairment with early treatment.

CONFLICT OF INTEREST

The authors stated there are no conflicts of interest.

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