Case Report

Hemiballismus in Subcortical Lacunar Infarcts

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Abstract

Chorea-hemiballismus is the most common movement disorder, and it commonly results from vascular lesions in the subthalamic nucleus. Rarely, it can result from lacunar infarcts. Here is illustrated a 95-year-old male with sudden-onset left-sided chorea-hemiballismus of 2 weeks, without any other neurological deficit, who was not a diabetic patient and had no form of acidosis or electrolyte abnormality. Computerized tomogram, serial 32 slides pre- and post-contrast images showed small non-enhancing, hypodense foci within the right frontal subcortical region adjacent to the frontal horn of the right lateral ventricle as well as within the lentiform nucleus. There were no mass effects seen. A diagnosis of hemiballismus from subcortical lacunar infarct was made. The hemiballismus was controlled after 2 weeks of haloperidol and clonazepam therapy, among others, with a substantial reduction in the abnormal movements. Hemiballismus attributable to subcortical lacunar infarcts though rare was presented.

Keywords: Hemiballismus, lacunar infarcts, lentiform nucleus, Nigeria, subcortical infarcts, subthalamic nucleus

Introduction

Movement disorders or involuntary abnormal movements, are broadly categorized into hyperkinetic movement disorders and hypokinetic movement disorders. The former include chorea, ballism/hemiballismus, dystonia, tremor, myoclonus, athetosis, tics, restless leg syndrome, stereotypy, abdominal syslinesi, hemifacial spasm, akathisia, myokymia, painful moving toes and fingers, paroxysmal dyskinesia, myorhythmia, jumpy stumps hyperekplexia, and synkinesia. Both hyperkinetic movement disorders and hypokinetic movement disorders like Parkinsonism may occur after both ischemic and hemorrhagic stroke.[1,2]

The frequency of movement disorders after stroke is not clear, with varying reports of 1%–3% from two studies.[3,4]

Hemiballismus is a violent form of chorea.[1,5] Chorea and Hemiballismus may be part of the same spectrum of disease, and hence, the term hemichorea-hemiballismus is used to reflect this.[6] Ballism often evolves into the lower amplitude chorea over time, as it improves, after stroke.[7] Chorea was the most common post-stroke movement disorder.[3,4] Stroke patients with movement disorders show significantly greater involvement of deep brain lesions of the basal ganglia, thalamus, internal capsule, diencephalon, and mesencephalon; cortical areas most commonly associated with movement disorders include primary motor, supplementary motor, and premotor cortical areas.[8]

There was a paucity of reports on hemichorea-hemiballismus from lacunar infarcts in Nigeria, prompting this case report.

Case Report

This 95-year-old man presented in Daystar specialist Hospital, Awka, Nigeria, with sudden onset abnormal violent involuntary movement of the left upper and lower limbs of 2 weeks duration. The fingers, wrist, elbow, and shoulder were affected simultaneously and with the same intensity. The same was also said about the left foot and leg. Since the onset of the movement, the intensity and frequency remained the same. However, emotional irritation aggravated the movements while during sleep; it was low to absent. The eyes, mouth, head, and face were spared of the abnormal movement. There

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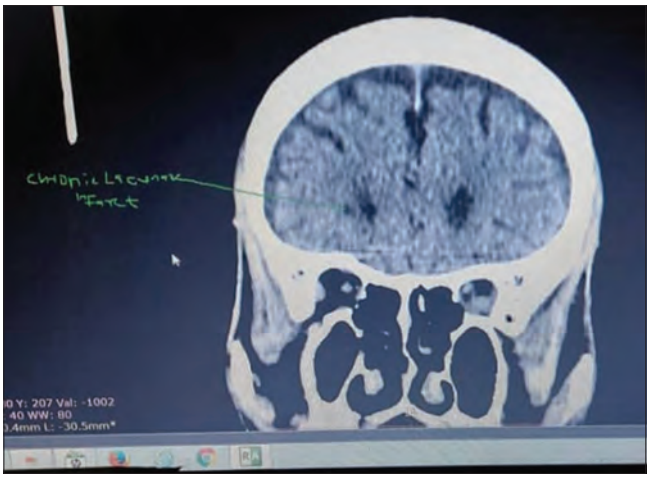

Figure 1: Brain computed tomography of the patient showing right frontal and left deep parietal subcortical lacunar infarcts

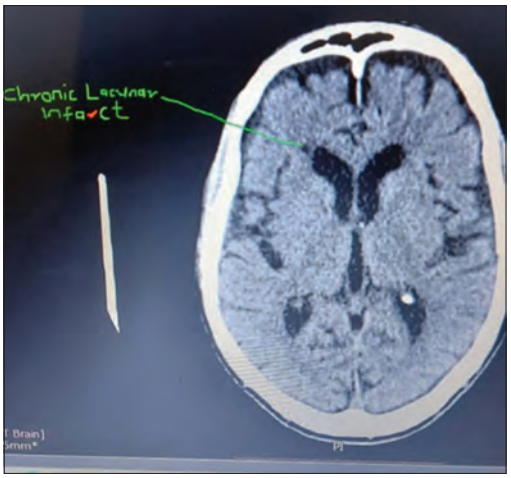

Figure 2: Brain computed tomography of the patient showing right frontal and left deep parietal subcortical lacunar infarcts

was no associated weakness of any part of the limbs or body; he also did not have any difficulty with speech, swallowing, vision. There was no irrational talk or change in behavior. He had no antecedent fever, seizure, trauma, ingestion of herbs, substance, or medications. He was not a known diabetes mellitus, dementia, or hypertensive patient. Neither had he any previous abnormal involuntary movement of any part of the body. He had no history of a previous stroke. There was no family history of abnormal involuntary movement. He had no history of tobacco, alcohol, or cigarette use.

Physical examination revealed an elderly man, conscious, cooperative, not in any distress, afebrile, not pale, not cyanosed, not icteric, but was mildly dehydrated. His pulse was 79 beats per minute, full volume and regular, and blood pressure 90/60 mmHg, in the supine position. The apex beat was at the fifth left intercostal space at the mid-clavicular line. Heart sound S1 and S2 were heard, and they were normal. His memory, both remote and recent, was intact. He had no meningeal irritation sign. Cranial nerves I–XII were intact. Muscle bulk was normal across all groups. There were involuntary, violent, jerky coarse and wide-amplitude movements of the left fingers, hand, wrist, arm, and the left leg, that waxed and waned in amplitude with apprehension and relaxation, respectively. The right side of the body was spared. Power was normal across all muscle groups. There was no sensory loss. Reflexes were normal and there was no sign of autonomic dysfunction. The cerebellar function was normal. The abdominal examination was unremarkable.

Full blood count showed hemoglobin of 10.7 g/dl. The white blood cell, both total and differential, were within the normal range. Erythrocyte sedimentation rate (ESR), and serum calcium were normal. Urinalysis and fasting serum lipid profile results, were within the normal range. Blood sugar was normal. Similarly, serum urea, creatinine, sodium, potassium, and chloride were normal. However, serum bicarbonate was elevated, 29.0 mmol/l, indicating mild alkalosis [Table 1].

Table 1: Laboratory results

Investigation	Results
Serum lipid profile (mg/dl)	
Total cholesterol	154
Low density lipoprotein cholesterol	107
High density lipoprotein cholesterol	54
Triglyceride	85
Very low density lipoprotein cholesterol	17
Full blood count (cells/ml)	
White blood count	6700
Lymphocytes	36
Neutrophils	59
Eosinophils	10
Monocytes	5
Basophil s	1
PCV	23
ESR	30 mm/ist h
Film	Microcytic hypochronic anaemia
Serum electrolytes urea and creatinine	
Sodium (mmol/l)	135
Potassium (mmol/l)	4.6
Chloride (mmol/l)	90
Bicarbonate (mmol/l)	29
Serum calcium (mg/dl)	9.2
Serum phosphate (mg/dl)	3.1

PCV: Packed cell volume, ESR: Erythrocyte sedimentation rate

Brain computed tomography scan report done 2 weeks and 3 days of illness

Serial 32 slides pre- and post-contrast (using iopamidol) were acquired at 0.5 mm cuts at the base and through the brain to the vertex, sagittal, and coronal formatting zone. Images showed small non-enhancing, hypodense foci within the right frontal subcortical region adjacent to the frontal horn of the right lateral ventricle as well as within the lentiform nucleus, no associated mass effect was appreciated. There was marked

prominence of the cerebral sulci, the Sylvian fissure, the basal cisterns and cerebella folia, associated fullness of the ventricular system noted all indicative of generalized cerebral and cerebella volume loss [Figures 1 and 2].

Impression:

1. Right frontal and left deep parietal chronic lacunar infarct
2. Background generalized cerebral and cerebellar volume loss likely age-related.

Based on the compatible history of new-onset abnormal involuntary, violent, coarse, wide-amplitude movements of the left upper and lower limbs of 2 weeks, and computed tomography (CT) brain evidence of small non-enhancing, hypodense foci within the right frontal subcortical region adjacent to the frontal horn of the right lateral ventricle as well as within the lentiform nucleus, a diagnosis of hemiballismus due to subcortical lacunar infarct was made.

He was given PO Clonazepam 0.5 mg BID, PO Haloperidol 5 mg daily × 14 days, PO Atorvastatin 10 mg daily × 21 days, PO Zinc gluconate 10 mg daily × 14 days, PO aspirin 75 mg daily, intravenous fluids (0.9% normal saline) with fluid restricted to 1.5 L daily × 3 days. The fluid was restricted not because he had hemodynamic imbalance but to avert any such danger usually seen around this age, those >90 years. The hemiballismic movement markedly decreased within 24 h of the commencement of therapy. He was subsequently discharged after 3 weeks on admission. PO Clonazepam was reduced to 0.5 mg daily, with a substantial reduction in the frequency of the abnormal movement and to return for check-up after 2 weeks, a week after the writing of this report.

Discussion

Hemiballismus is a violent form of chorea comprised of wild, flinging, large-amplitude movements on one side of the body.[5] It describes violent swinging movements of one side caused usually by infarction or hemorrhage in the contralateral subthalamic nucleus.[2] Although it affects proximal limb muscles predominantly, ballistic movements may affect just one limb (monoballism) or, more exceptionally, both upper or lower limbs, or all limbs (paraballism).[6,7] Our index patient had the involvement of both the left upper and lower limbs. The most common cause is a partial lesion (infarct or hemorrhage) in the subthalamic nucleus, but cases can also be seen with lesions in the putamen, thalamus, and parietal cortex.[5] Our index patient had new-onset hemiballismus and subcortical lacunar infarcts, indicating he had hemiballismus from a rare cause. Ischemic stroke in the contralateral subthalamic nucleus was found to be the most common lesion in patients with hemiballismus in a study.[9] Suri *et al*. found out that chorea and Parkinsonism were the most common movement disorders after ischemic stroke, while dystonia and tremor were the most common movement disorders after a hemorrhagic stroke.[10] This reflected the picture in our patient as he had hemiballismus and lacunar infarcts.

Hemiballismus can occur in connective diseases like systemic lupus erythematosus (SLE) in which ESR will likely be elevated.[11] The normal value of ESR in our patient ruled out SLE as the cause of the hemiballismus. Similarly, the normal values of serum calcium and phosphate levels excluded the possibility of parathyroid disease as the cause of hemiballismus in this patient.[12] Diabetes mellitus is a known cause of hemiballismus, especially when it is complicated by ketoacidosis.[13] However, our patient was not observed to have diabetes mellitus, nor did he have acidosis, to explain the hemiballismus. Thus far, the sudden-onset hemiballismus observed in our index patients and the CT evidence of subcortical infarcts involving the subthalamic nucleus and the lentiform nucleus, on imaging conducted 2 weeks and 3 days supported the diagnosis of hemiballismus due to subthalamic, lentiform lacunar infarcts.

Ballism and chorea typically respond to the same drugs.[7] Typical dopamine receptor blockers such as haloperidol, pimozide, perphenazine, are the first-line treatment for hemiballismus.[6] Our index patient showed a remarkable response to haloperidol treatment. Surgical interventions such as stereotactic ventral intermediate thalamotomy and chronic thalamic stimulation have been found to be effective and may be considered in drug-resistant cases but are contraindicated in the very frail and those with hypertension.[6] Surgical intervention was not considered in our index patient as he was not followed enough; neither were the facilities available. Although hemiballismus is usually self-limiting and tends to resolve spontaneously after weeks or months[5] our patient was not followed up sufficiently to enable us to confirm this exertion in him.

Conclusion

Hemiballismus attributable to subcortical lacunar infarcts though rare was presented. The patient had sudden-onset hemiballismus and subcortical lacunar infarct.

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Conflicts of interest

There are no conflicts of interest.

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