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Cognitive Impairment Related to Bilateral Lacunar Stroke in a 38-Year-Old Patient: A Case Report

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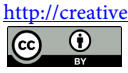

Abstract

Bilateral lacunar strokes are rare and can lead to significant cognitive impairment, even in young adults without classical risk factors. We report the case of a 38-year-old Guinean man who developed progressive memory and executive function deficits over a six-month period. Brain CT revealed bilateral lacunar infarcts involving the caudate nuclei and putamina. Vascular, metabolic, cardiac work-up, and EEG were normal. This case highlights the importance of considering bilateral lacunar stroke as a cause of unexplained cognitive decline in young adults and underscores the need for early diagnosis and multidisciplinary management, especially in resource-limited settings.

Subject Areas

Neurology

Keywords

Bilateral Lacunar Stroke, Cognitive Impairment, Young Adult, Brain Imaging, Sub-Saharan Africa

1. Introduction

Stroke remains a leading cause of morbidity and mortality worldwide. Lacunar strokes are small infarcts involving the territories of deep perforating arteries (Fisher, 1982) [1]. Although common in elderly hypertensive or diabetic patients, they can occur in young adults, often related to specific etiologies such as thrombophilic disorders, cardioembolic heart disease, or inflammatory angiopathies

(Wardlaw *et al.*, 2019) [2].

Bilateral lacunar involvement of the basal ganglia is exceptional and can cause marked cognitive impairment (Sachdev *et al.*, 2014) [3]. Diagnosis relies on brain imaging and appropriate neuropsychological assessment. In low-resource settings, recognition is essential to initiate preventive treatment and rehabilitation.

2. Case Report

Mr. M., a 38-year-old man with no known medical history, presented with progressive memory problems over approximately 6 months, associated with psychomotor slowing, poor concentration, and disorganization in daily activities. There was no history of epilepsy, head trauma, substance abuse, or chronic intoxication.

2.1. Timeline of Events

- **Month 0 (onset):**

The patient's family noticed subtle memory lapses, such as forgetting recent conversations, misplacing personal items, and repeating questions within a short time. He was still fully independent in daily activities and able to work, but his relatives observed an unusual slowness in responding to questions.

- **Month 3 (progression):**

Cognitive difficulties became more evident in the workplace. The patient began making calculation errors, missing scheduled meetings, and requiring repeated instructions to complete tasks he previously handled easily. At home, his wife reported that he sometimes forgot to turn off the stove and left the house without locking the door.

- **Month 5 (first medical evaluation):**

Due to increasing concerns from both family and colleagues, he was brought to a general practitioner. The examination did not reveal any motor or sensory deficit, but cognitive changes were noted, prompting referral to the neurology department for further assessment.

- **Month 6 (specialized assessment):**

At the neurology clinic, a detailed neurocognitive evaluation was conducted, revealing significant deficits in executive functions and working memory. A brain CT scan was performed, showing bilateral lacunar infarcts of the caudate nuclei and putamina. Additional vascular, metabolic, and cardiac work-ups were ordered.

2.2. Clinical Examination

- Consciousness: normal.
- Speech: fluent.
- No motor or sensory deficit.
- MoCA score: 20/30 with marked executive dysfunction, impaired working memory, and reduced sustained attention.
- No associated depressive symptoms.

2.3. Additional Investigations

- **Brain CT:** bilateral lacunar infarcts of the caudate nuclei and putamina.
- **EEG:** normal.
- **Vascular work-up:** normal fasting glucose, lipid profile, creatinine, urea, uric acid; blood pressure 120/80 mmHg.
- **Cardioembolic work-up:** normal ECG and transthoracic echocardiography.
- **Thrombophilia screen:** protein C, protein S, antithrombin III, factor V Leiden mutation, lupus anticoagulant, antiphospholipid antibodies—all negative.

2.4. Initial Management

- Aspirin 160 mg/day.
- Atorvastatin 40 mg/day.
- Lifestyle and dietary measures, cardiovascular risk monitoring.
- Weekly cognitive rehabilitation program adapted to local resources.

2.5. Follow-Up

At 3 months: mild improvement in executive functions (MoCA 23/30) but persistent moderate memory deficit.

3. Discussion

This case illustrates several points:

- **Rarity:** Bilateral lacunar stroke in a young adult without classical risk factors is uncommon.
- **Critical lesion location:** The caudate nuclei and putamina are involved in executive functions, behavioral initiation, and working memory (Gouw *et al.*, 2011) [4].
- **Comprehensive etiological approach:** Excluding rare conditions (thrombophilias, cardioembolic sources, angiopathies) increases the plausibility of idiopathic small-vessel disease.
- **Role of CT in resource-limited settings:** While MRI is more sensitive, CT often suffices to make a diagnosis and initiate treatment.

Differential diagnoses considered and excluded:

- **Inflammatory encephalopathy:** No clinical or laboratory evidence.
- **Toxic or deficiency-related encephalopathy:** Negative history and normal laboratory tests.

4. Limitations

The absence of MRI is a major limitation, reducing sensitivity for detecting white matter lesions or smaller lacunar infarcts and limiting the investigation of other diagnoses (angiopathies, multiple sclerosis).

5. Conclusion

Bilateral lacunar stroke is a rare cause of cognitive decline in young adults. This

case emphasizes the importance of early diagnosis, multidisciplinary management, and rehabilitation programs adapted to the local context to improve functional outcomes.

Conflicts of Interest

The authors declare no conflicts of interest.

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