

Generalized Chorea Secondary to a Right Putaminal Infarct: A Rare Manifestation of a Unilateral Basal Ganglia Lesion

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Abstract

Background: Chorea is typically characterized by irregular, involuntary movements resulting from basal ganglia dysfunction. While most basal ganglia lesions cause hemichorea contralateral to the lesion, generalized chorea due to a unilateral infarct is rare.

Case Presentation: A 60-year-old hypertensive female presented with a three-day history of imbalance, transient loss of consciousness, and involuntary movements predominantly affecting the left upper and lower limbs, with milder involvement on the right. Examination revealed generalized chorea with left-side predominance and mild weakness (4+/5) in the left limbs. There were no other focal neurological deficits. MRI of the brain revealed a right subacute putaminal infarct. Other investigations including metabolic panel, ECG, echocardiography, and CT scan were unremarkable.

Conclusion: This case highlights the rare occurrence of generalized chorea resulting from a unilateral putaminal infarct, underscoring the complex network-level role of basal ganglia in motor control.

Keywords: Chorea, basal ganglia, putaminal infarct, stroke, movement disorder

Introduction

Chorea refers to involuntary, irregular, and unpredictable movements caused by dysfunction in the basal ganglia circuitry. While chorea due to vascular lesions is not uncommon, it typically presents as hemichorea contralateral to the lesion. Generalized chorea from a unilateral basal ganglia infarct is a rare phenomenon, likely reflecting broader network dysregulation. Here, we report a case of generalized chorea due to a right putaminal infarct in a hypertensive patient.

Case Report

A 60-year-old right-handed woman with a known history of hypertension presented to the Neurology outpatient department with a three-day history of imbalance predominantly to the left side, especially while eating. The event was followed by a transient loss of consciousness lasting approximately two minutes. Upon regaining consciousness, she noted giddiness and the onset of abnormal movements in the left upper and lower limbs, with milder involvement of the right limbs.

On neurological examination, she was alert and oriented. There were involuntary, irregular, semi-rhythmic, flowing movements predominantly affecting the fingers and distal limbs—characteristic of chorea—more prominent on the left side but also evident on the right. Muscle strength was slightly

reduced in the left upper and lower limbs (MRC grade 4+/5), but tone, reflexes, and plantar responses were normal. Sensory, cerebellar, cranial nerve, and autonomic examinations were unremarkable. Routine blood chemistry, metabolic panel, ECG, echocardiography, and urgent CT head were normal. MRI brain revealed a subacute infarct in the right putamen, with no other significant abnormalities. No structural abnormalities were found in the contralateral basal ganglia or cortex.

She was managed conservatively with symptomatic treatment and antiplatelets. Over the next few days, the chorea showed partial improvement. Given the rarity of this presentation, she was diagnosed with generalized chorea secondary to right putaminal infarct.

Discussion

The basal ganglia play a central role in motor control through the integration of excitatory and inhibitory signals from the cortex and thalamus. Lesions in these circuits typically produce contralateral hemichorea or hemiballismus. However, in rare instances, a unilateral lesion—particularly in the putamen—can disrupt the indirect pathway to such an extent that generalized chorea emerges. This phenomenon may be explained by: (1) Disinhibition of thalamocortical pathways due to impaired GABAergic output; (2) Transcallosal and interhemispheric dysregulation, leading to bilateral motor manifestations; (3) Diaschisis, where a focal lesion leads to functional disruption in remote but connected neural networks.

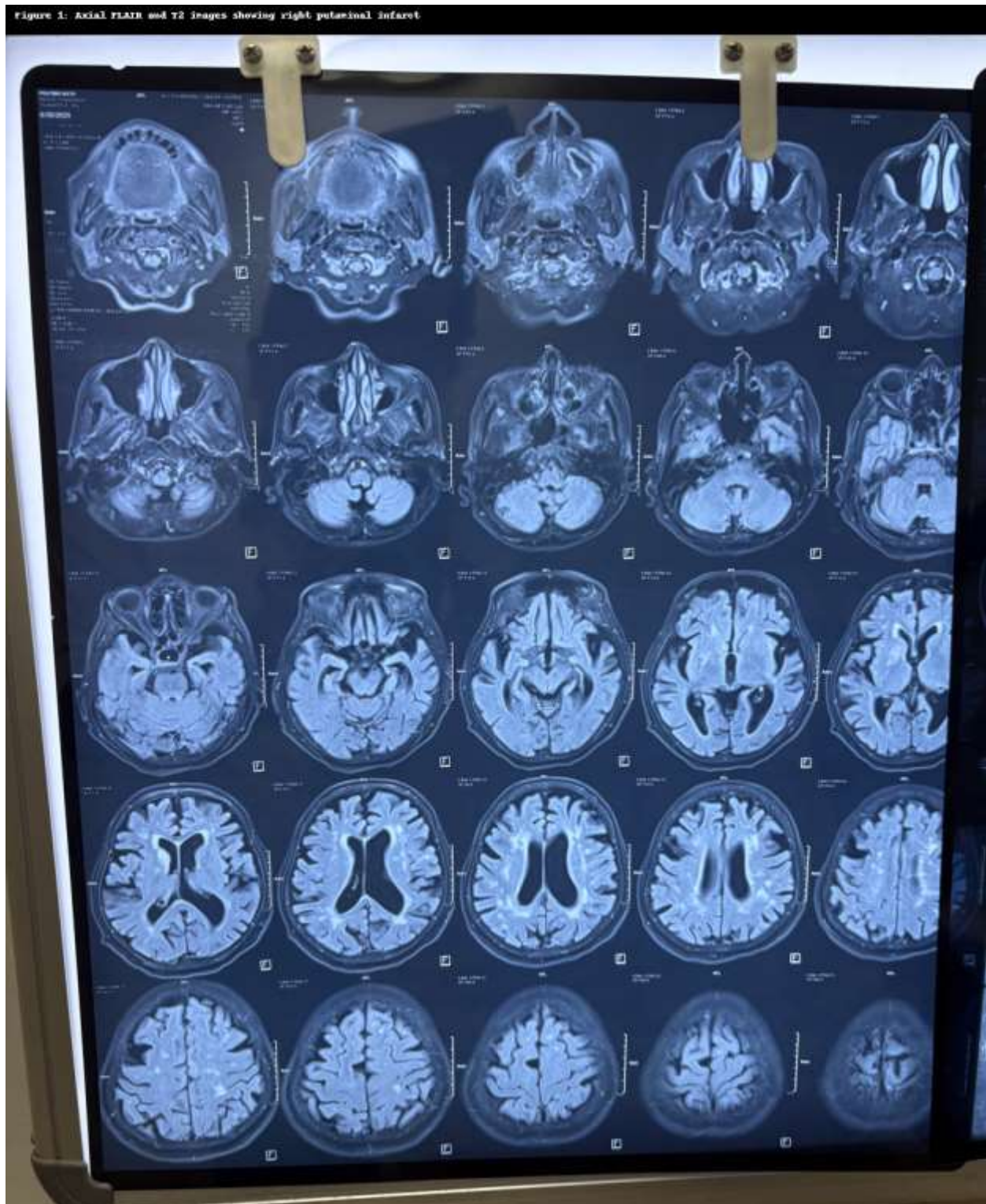
A few prior reports, such as those by Yeh et al. and Oh et al., have described similar presentations. Our case adds to this limited pool of evidence supporting the capacity of unilateral putaminal infarcts to cause generalized hyperkinetic states.

Conclusion

Although hemichorea is a well-known manifestation of basal ganglia infarction, generalized chorea from a unilateral putaminal lesion is rare. This case underscores the need for clinicians to consider focal structural lesions as a cause of generalized involuntary movements, especially when supported by neuroimaging.

References

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Link of Video showing Generalised chorea
<https://youtube.com/shorts/JSLoTLZ2zCs?si=Fvk3WYfb81MF2aEs>