

# Ischemic Stroke Presenting with Hemichorea: A Case Series

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## ABSTRACT

**Background:** Hemichorea is a rare hyperkinetic movement disorder that may occur as a complication of ischemic stroke or metabolic disturbances, particularly non-ketotic hyperglycemic hyperosmolar state (NKHHS).

**Case Series:** We present two female patients with uncontrolled type II diabetes mellitus who developed hemichorea associated with ischemic stroke. The first case showed severe hyperglycemia (>500 mg/dL) without radiological abnormalities on non-contrast CT scan. The second case presented with hyperglycemia (375 mg/dL) and a hyperdense lesion in the left caudate nucleus. Both patients received aspirin and insulin, while symptomatic management included haloperidol and, in the second case, additional valproic acid. Both patients demonstrated gradual clinical improvement.

**Discussion:** Post-stroke hemichorea is reported in only 1–4% of cases. The underlying mechanism involves disruption of the striato-thalamo-cortical pathway and, in hyperglycemia, impairment of GABA metabolism. Most cases respond well to glycemic control, although neuroleptics or antiepileptics may be necessary to control symptoms.

**Conclusion:** This case series highlights that hemichorea can be a rare manifestation of ischemic stroke, either with or without radiological evidence, and may be

aggravated by uncontrolled hyperglycemia. Early diagnosis, strict glycemic regulation, and appropriate symptomatic treatment are crucial for favorable outcomes.

**Keywords:** Ischemic stroke, hemichorea, hyperglycemia

## INTRODUCTION

Stroke according to the World Health Organization (WHO), is defined as rapidly developing clinical signs of focal (or global) disturbance of cerebral function, lasting for 24 hours or longer, or leading to death, with no apparent cause other than vascular origin. The Indonesian National Guidelines for Stroke Management (PNPK) 2019 define stroke as an acute clinical manifestation due to neurological dysfunction of the brain, spinal cord, or retina, either partial or total, persisting for  $\geq 24$  hours or resulting in death caused by vascular disorders.<sup>1</sup>

Focal brain lesions can cause various abnormal movement disorders, including hemichorea-hemiballismus, parkinsonism, myoclonus, dystonia, tremor, and asterixis. The most common cause is stroke, followed by trauma, neoplasm, anoxia, vascular malformation, metabolic disease, or multiple sclerosis.<sup>2</sup>

Hemichorea is a hyperkinetic movement disorder characterized by sudden, irregular, dance-like involuntary movements that can affect any part of the body.<sup>3</sup> This condition

may occur as a primary disorder or secondary to various medical conditions. Primary hemichorea is idiopathic or genetic, while secondary causes include structural lesions, metabolic or endocrine disorders, infections, drugs, and autoimmune processes. Metabolic etiologies include hyperglycemia, hypoglycemia, uremia, hyperthyroidism, and hyponatremia, with cerebrovascular disease and non-ketotic hyperglycemic hyperosmolar state (NKHHS) being the most common causes.<sup>3,4</sup>

We report two cases of ischemic stroke accompanied by hemichorea. This case series aims to describe patients with ischemic stroke presenting with hemichorea, with history diabetes mellitus.

### CASES

Case 1: A 57-year-old woman presented to the neurology clinic with involuntary, jerky movements of the right arm for the past 5 days, which subsided at rest and disappeared during sleep. Ten days earlier, she had developed right-sided weakness, facial asymmetry, and slurred speech. She

denied fever, headache, or vomiting. This was her first such episode. She had a 16-year history of type II diabetes mellitus but was not taking medication regularly. She denied a history of hypertension, thyroid disease, head trauma, or seizures.

On general examination, blood pressure was 100/67 mmHg, pulse 97/min, respiratory rate 20/min, and temperature 36.4°C. Neurological examination revealed alert consciousness, central-type facial and lingual palsy on the right side, right hemiparesis with motor strength grade 3, no sensory deficit, positive Babinski and Chaddock signs on the right, and rapid arrhythmic involuntary movements of the right arm. Laboratory findings showed very high blood glucose (>500 mg/dL) with negative urine ketones. Non-contrast CT scan of the head showed no abnormalities (Figure 1). The patient was hospitalized for 5 days and treated with aspirin 80 mg daily, haloperidol 2 × 2.5 mg, and insulin starting at 10 units. Clinical improvement was noted, and she was discharged on outpatient care.

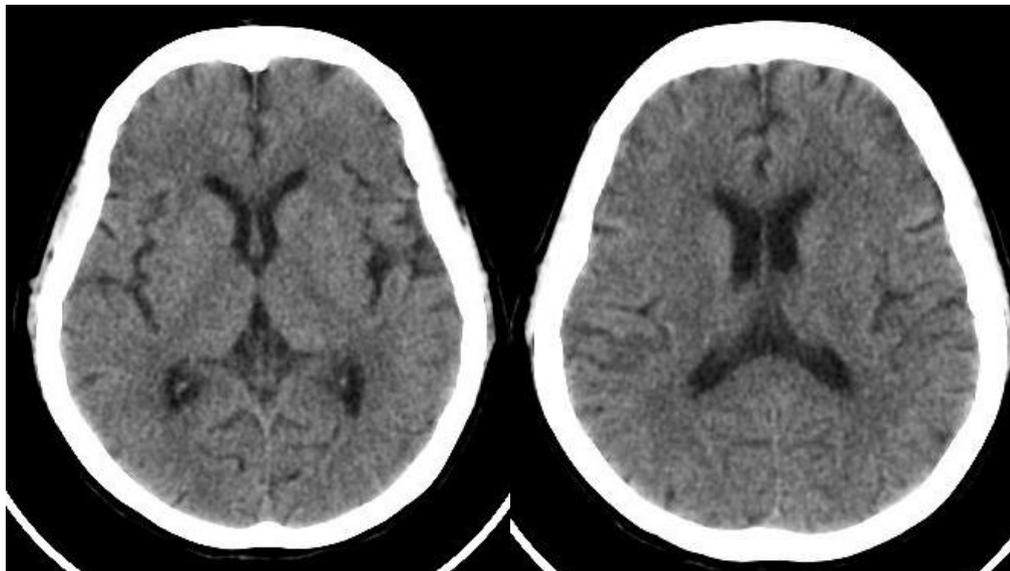


Figure 1: Non-contrast head CT-scan

Case 2: A 56-year-old woman presented to the emergency department with continuous, uncontrolled involuntary movements of the right arm and leg for the past week, subsiding only during sleep. These

movements were accompanied by slurred speech, facial asymmetry, and right-sided weakness. This was her first such episode. She had a 2-year history of poorly controlled type II diabetes mellitus. She

denied hypertension, thyroid disease, head trauma, or seizures. On general examination, blood pressure was 140/80 mmHg, pulse 108/min, respiratory rate 20/min, and temperature 36.6°C. Neurological examination showed alert consciousness, central-type facial and lingual palsy on the right side, positive Babinski sign on the right, and continuous rapid, arrhythmic involuntary movements of the right arm and leg. Muscle strength was

difficult to assess due to involuntary movements. Blood glucose was 375 mg/dL with negative urine ketones. Non-contrast CT scan revealed a hyperdense lesion in the left caudate nucleus (Figure 2). The patient was hospitalized for 7 days and treated with aspirin 80 mg daily, haloperidol 5 mg, valproic acid 2 × 250 mg, and insulin starting at 10 units. She improved clinically and was discharged on outpatient care.

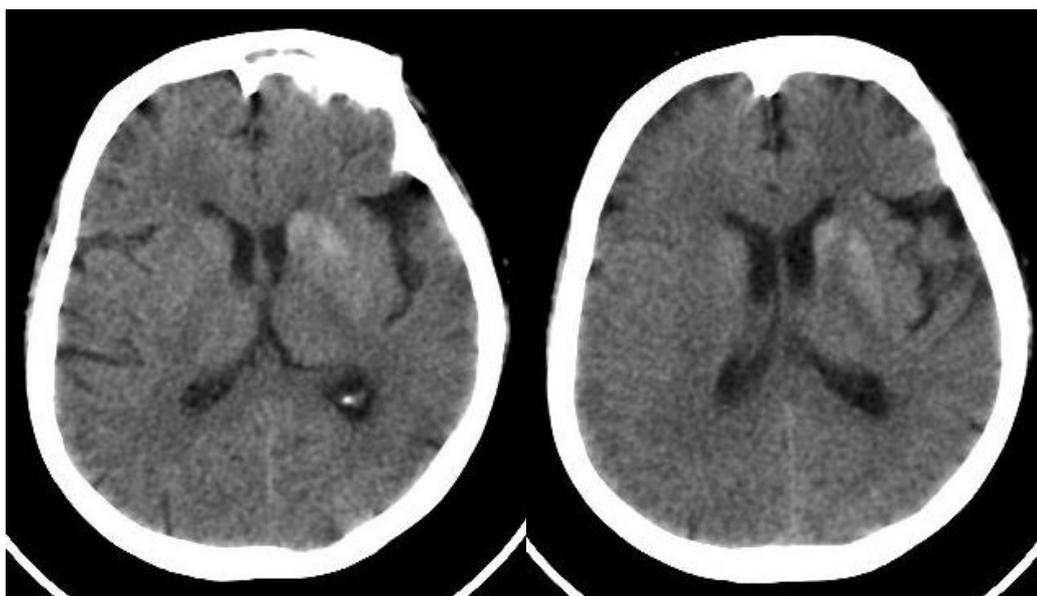


Figure 2: Non-contrast head CT-scan

## DISCUSSION

The incidence of hemichorea due to stroke is rare, accounting for about 1–4% of all strokes.<sup>3</sup> A study screening 1,500 stroke patients between 1990 and 1999 reported 56 patients with post-stroke movement disorders, of whom 20 developed chorea.<sup>3</sup> The female-to-male ratio was 1.8:1, with older age being a major risk factor; the average onset age was around 70 years (range 20–90 years).<sup>5</sup> Hemichorea has multiple etiologies, including metabolic disorders, hypoxic-ischemic injury, vascular disease, drugs, toxins, infections, and systemic inflammatory-immunologic conditions. Among metabolic causes, NKHHS is the most common.<sup>3</sup> The pathogenesis of hemichorea caused by contralateral striatal neuron lesions involves the striato-thalamo-cortical pathway, which

is part of the indirect pathway through disruption of GABAergic transmission from the striatum to the external globus pallidus (GPe). This disturbance increases dopaminergic activity, which enhances GPe neuronal activity and inhibits the subthalamic nucleus. Such inhibition leads to loss of control over the internal globus pallidus (GPi) neurons, ultimately resulting in the loss of motor inhibition by the thalamus. Lesions of the subthalamic nucleus may also produce the same dysfunction by reducing thalamic motor inhibition.<sup>4</sup> In cases of hemichorea caused by cortical lesions, the excitatory circuits from the frontal or parietal cortex are disrupted (the somatosensory cortex projects to the caudate nucleus and putamen), resulting in involuntary movements. These involuntary

movements may be caused by hypoperfusion in the basal ganglia circuits.<sup>4</sup> The pathophysiology of hemichorea associated with NKHHS remains unclear. The first hypothesis is depletion of gamma-aminobutyric acid (GABA) as an alternative energy source in hyperglycemia, leading to thalamic disinhibition and hyperkinetic movements in combination with GABA deficiency. Another hypothesis suggests that hyperglycemia directly affects brain metabolism, disrupts blood-brain barrier permeability, and that ischemic and microhemorrhagic changes related to hyperglycemia can be observed in the basal ganglia.<sup>3</sup>

Chang et al. concluded that the clinical manifestations of hemichorea-hemiballismus can be divided into two types. The common type occurs in patients with diabetes, elevated blood glucose levels, negative urine ketones, unilateral or bilateral chorea, and abnormalities detected on brain MRI or CT scans, which represents the most frequent presentation. The uncommon type also occurs in patients with diabetes, elevated blood glucose levels, negative urine ketones, and unilateral or bilateral chorea, but without any radiological abnormalities on MRI or CT scans, making it relatively rare in hemichorea-hemiballismus.<sup>6</sup>

In this case series, the patients experienced involuntary movements associated with ischemic stroke as well as hyperglycemia. The diagnosis of ischemic stroke was established based on clinical neurological findings. The most common cause of hemichorea is metabolic disorders, particularly NKHHS. After proper correction of hyperglycemia, gradual improvement of involuntary movements was observed. In the first case, a non-contrast head CT scan revealed no lesions, whereas in the second case, a non-contrast head CT scan demonstrated a hyperdense lesion in the caudate nucleus. This is consistent with the retrospective study by Guzey Aras et al., which reported no radiological abnormalities in some patients

with hemichorea.<sup>3</sup> Several studies have also noted that hemichorea-hemiballismus in NKHHS is associated with the incidence of ischemic stroke in the acute phase of the disease, which in such cases is confirmed by head CT scans.<sup>5</sup> The prevalence of hemichorea caused by stroke is estimated to be only 1–4%; however, due to limitations in diagnostic modalities, further investigations are required in these cases to determine a clear etiology.

In the administration of therapy, the underlying cause must first be identified and treated. In this case series, the patients were given aspirin for ischemic stroke, haloperidol to relieve movement disorder symptoms, and insulin to reduce blood glucose levels. The literature states that the first-line therapy for chorea is tetrabenazine; however, this drug class is not yet available in Indonesia. Other studies have reported that the use of neuroleptics such as haloperidol is commonly employed to accelerate the resolution of involuntary movements. In this case series, there was a difference in the doses of neuroleptics administered. In the second case, haloperidol with gradual dose titration up to the maximum daily dose of 6–8 mg did not reduce involuntary movements, whereas the addition of valproic acid was able to alleviate the movement disorder symptoms.<sup>5</sup> Hemichorea caused by NKHHS is often benign and responds rapidly to strict blood glucose control. However, until glycemic regulation is achieved, other medical treatments may be initiated to control the symptoms.<sup>3</sup>

## **CONCLUSION**

Hemichorea is a rare clinical manifestation. Movement disorders such as chorea may occur in ischemic stroke and metabolic disturbances, although the exact pathophysiology of this condition has not yet been clearly established. Hyperglycemia is not only a risk factor for stroke but may also directly cause movement disorders. Early recognition and appropriate management, including stroke therapy, strict

blood glucose control, administration of neuroleptic agents or combination neuroleptic and antiepileptics can lead to favorable clinical outcomes in patients.

**Declaration by Authors**

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