Acta Neurologica Indonesia 2026; 04(02)

Acta Neurologica Indonesia
Department of Neurology, Faculty of Medicine Universitas Indonesia
ISSN 3032-6303

Case Report

PERSISTENT HICCUP IN SUPRATENTORIAL INFARCTION WITH GLOBAL APHASIA: A CASE REPORT

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ABSTRACT

Introduction A persistent hiccup is an involuntary contraction involving the diaphragm and intercostal muscle that lasts more than 2 days until a month. This condition is often associated with cerebrovascular infarction in the brainstem region. In contrast, persistent hiccup related to ischemic stroke in the supratentorial region was rarely reported. Here, we describe a patient with a persistent hiccup accompanied by global aphasia in acute supratentorial infarction.

Case Summary A male, 61 years old Balinese, right-handed, was admitted due to palpitation. In the emergency room, the patient developed sudden right-sided weakness and an inability to speak. Neurological examination showed right-sided hemiplegia with muscle strength of 1 and global aphasia. EKG showed Rapid Atrial Fibrillation. Non-Contrast CT-Scan showed acute cerebral infarction within the left middle cerebral artery territory. Afterward, the patient experienced hiccups from the time he was in the emergency unit until he was discharged, and the hiccups lasted for more than 7 days. The patient was treated with anticoagulant and neuroprotectant alongside supportive therapy. The hiccup was treated with oral chlorpromazine.

Discussion The central hiccup component is believed to lie in the medulla oblongata. Although the exact mechanism of persistent hiccups is still unclear, global aphasia, which involves a large part of the dominant hemisphere, may provide a new insight into the pathophysiology of hiccups.

Conclusion Persistent hiccups may occur in supratentorial lesions with global aphasia, where a large portion of the dominant hemisphere is affected.

Keywords: persistent hiccup; global aphasia; supratentorial infarction

INTRODUCTION

Hiccups, or singultus, are involuntary, spasmodic contractions of the diaphragm followed by abrupt glottic closure, typically benign and self-limited. However, persistent hiccups defined as hiccups lasting longer than 48 hours may indicate underlying pathological processes, including neurological disorders. While brainstem lesions, particularly in the medulla oblongata, are well-recognized causes of central hiccups due to their involvement in the hiccup reflex arc, supratentorial lesions are rarely implicated.

Supratentorial infarctions, especially those involving the dominant hemisphere, are more commonly associated with cognitive, motor,

and

language deficits such as aphasia. Global aphasia, which results from extensive damage to language-related cortical areas, typically presents with severe impairments in both expressive and receptive language functions. The association between supratentorial infarction and persistent hiccup is infrequently reported and not fully understood.

Here, we present a rare case of persistent hiccup occurring in the context of a supratentorial infarction with global aphasia, highlighting the possible pathophysiological mechanisms, diagnostic considerations, and clinical implications of this unusual presentation.

CASE REPORT

This systematic review and meta- A 61-year-old Balinese male with no significant past medical history presented to the emergency department with complaints of palpitations, persistent hiccups, and a brief loss of consciousness that occurred approximately one hour prior to arrival. According to his wife, the patient had been asleep before the episode and abruptly stood up upon waking. He then experienced transient visual dimming

followed by a sudden collapse, remaining unconscious for approximately five minutes. Upon regaining consciousness, the patient reported persistent hiccups, palpitations, and nausea. He denied experiencing chest pain, headache, cold sweats, limb weakness, or slurred speech at that time. He had no known comorbidities, was not on any chronic medication, and reported a positive history of smoking.

On initial evaluation in the emergency department, the patient was fully alert and oriented, with a Glasgow Coma Scale (GCS) score of E4V5M6. His vital signs included a blood pressure of 100/70 mmHg, pulse rate of 116 beats per minute, respiratory rate of 24 breaths per minute, oxygen saturation of 99% on room air, and an axillary temperature of 36.0°C. Physical examination revealed no abnormalities; pupillary reflexes were normal and symmetrical, thoracic examination showed symmetrical chest movement with normal breath and heart sounds, and no peripheral edema was noted. Neurological examination at this point did not

demonstrate any focal deficits; there was no facial nerve palsy, tongue deviation, or sensory disturbances. A random capillary blood glucose level was 127 mg/dL,

and ECG revealed
atrial fibrillation with a rapid ventricular response at 117 bpm. The patient was diagnosed with

observation syncope ec susp transient ischemic attack (Sheldon score: 0; ABCD2 score: low risk 1 point) with AF RVR.

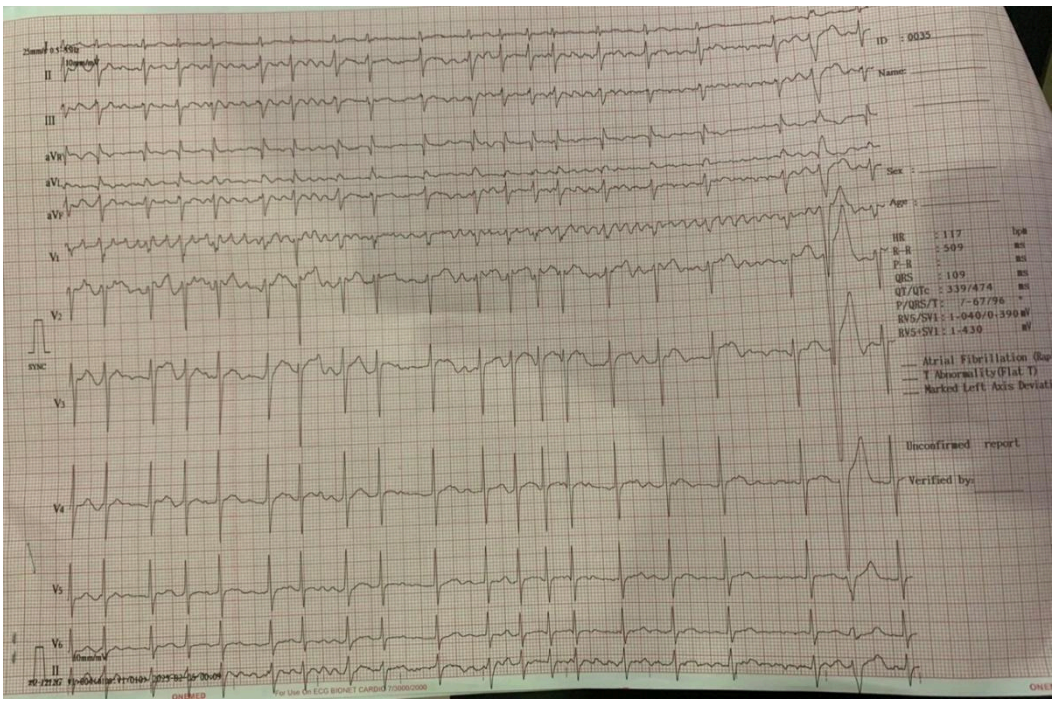

Figure 1. Twelve-lead electrocardiogram obtained on admission demonstrating atrial fibrillation (heart rate ~117 bpm). The tracing shows absence of distinct P waves, irregularly irregular R-R intervals, and narrow QRS complexes. Additional findings include flattened T waves.

However, during observation, the patient experienced an acute neurological deterioration. He became confused, his speech became unclear and slurred, and he failed to recognize family members. Importantly, there were no preceding complaints of headache or vomiting. Upon re-evaluation, his GCS dropped to E3V3M5, and neurological examination revealed right-sided flaccid hemiparesis with no signs of increased intracranial

pressure. Oxygen saturation remained stable at 99% with 2 L/min oxygen via nasal cannula. A non-contrast brain CT scan was performed, showed acute cerebral infarction within the left middle cerebral artery teritory. Chest X-ray was unremarkable with a cardio-thoracic ratio of 51%, and

routine laboratory parameters, including complete blood count, renal function, and electrolytes, were within normal limits.

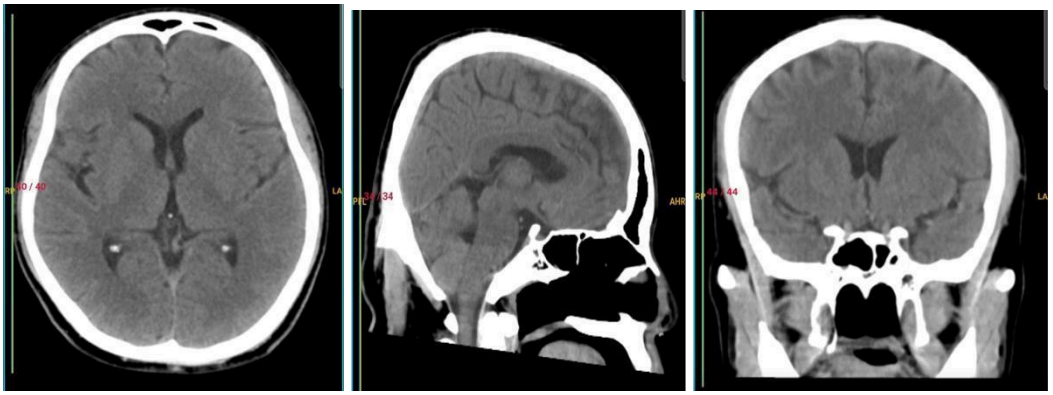

Figure 2. Non-contrast head CT scan (axial, sagittal, and coronal views) of a 61-year-old male presenting with persistent hiccups.

The patient was admitted with a working diagnosis of acute ischemic stroke with suspected cardioembolic etiology due to atrial fibrillation. He was started on IV citicoline and mecobalamin, oral atorvastatin, and anticoagulation therapy as advised by the cardiology team. Cardiovascular management included digoxin, spironolactone, bisoprolol, and gastric protection.

During hospitalization, the patient persistently complained of hiccups, which were only temporarily relieved by drinking water. Pharmacological management with IV omeprazole, oral domperidone, and chlorpromazine 25 mg once daily led to partial improvement. Despite persistent hiccups, the patient was discharged on day 8 with GCS E4VxM5, global aphasia, and right-sided hemiparesis (grade 1–2). At the follow-up visit on day 13, the patient remained unable to communicate effectively and continued to experience hiccups intermittently, which resolved completely by day 14 with continued pharmacologic therapy.

DISCUSSION

Persistent hiccups (singultus) are an uncommon but potentially important symptom in neurological practice. Defined as hiccups lasting more than 48 hours, they may be associated with central nervous system lesions, including stroke, neoplasms, or infections. While brainstem lesions are the most common neurological causes of hiccups, supratentorial lesions have also been implicated.[1,2,3]

In this case, a 61-year-old male developed persistent hiccups

following a left middle cerebral artery (MCA) infarction, likely cardioembolic in nature due to atrial fibrillation (AF). Although hiccups a are more typically reported in posterior circulation strokes, recent literature acknowledges that cortical lesions, particularly those involving the insula or opercular cortex, may influence hiccup generation through disruption of cortical inhibition over the brainstem hiccup center.[4] The involvement of cortical structures in modulating autonomic and visceral responses is increasingly recognized in neurogastroenterology.[5]

Atrial fibrillation remains a major risk factor for ischemic stroke, especially embolic strokes involving large vessels such as the MCA.[6] In this patient, the initial presentation of syncope and palpitations, coupled with ECG-confirmed AF, suggested a high likelihood of a cardioembolic event. Although the ABCD2 and Sheldon scores were initially low, the subsequent neurological deterioration highlights the dynamic nature of stroke progression in AF-related cases.

Pharmacological management of persistent hiccups often includes antipsychotics (e.g., chlorpromazine), prokinetic agents (e.g., metoclopramide), or GABAergic drugs (e.g., baclofen), with variable efficacy depending on the etiology.[7] In central hiccups, especially post-stroke, response to treatment may be incomplete or delayed. Our patient showed partial improvement with chlorpromazine and supportive therapy, with eventual resolution by day 14, likely due to gradual resolution of cortical dysfunction or neuroplastic compensation.

This case highlights the importance of considering central neurological causes, including supratentorial stroke, in patients with persistent hiccups, especially in those with risk factors such as AF. Early recognition and appropriate neuroimaging are essential for guiding both acute stroke management and symptom-directed therapy.

CONCLUSION

This case illustrates a rare presentation of persistent hiccups as an early symptom of acute ischemic stroke due to a cardioembolic event in a patient with atrial fibrillation.

While hiccups are classically linked to brainstem involvement, their occurrence in this patient with a left middle cerebral artery infarct and resultant global aphasia suggests that supratentorial lesions affecting the dominant hemisphere can also be implicated. Global aphasia, involving extensive cortical disruption, may influence central hiccup regulation pathways through mechanisms that are not yet fully understood. This highlights the need for clinicians to consider stroke in the differential diagnosis of persistent hiccups, especially when accompanied by other subtle neurological signs.

ACKNOWLEDGEMENT

The authors would like to acknowledge the contributions of the emergency department, neurology, and cardiology teams at Tabanan General Hospital for their clinical support and collaboration in the management of this case.

REFERENCES

1. Lee GW, Kim RB, Go SI, et al. Hiccup: a common problem with some unusual causes and cures. *Korean J Intern Med*. 2016;31(6):1037–1045. doi:10.3904/kjim.2016.015

2. Meletti S, Vaudano AE, Michelucci R. Central causes of hiccups: a new functional MRI finding. *Neurology*. 2020;94(6):e620–e622. doi:10.1212/WNL.0000000000008815

3. Itabashi R, Endo K, Saito T, Fukuma K, Yazawa Y. Supratentorial infarcts accompanying hiccup. *Brain Behav*. 2019;9(11):e01439. doi:10.1002/brb3.1439

4. Bektaş H, Karadaş Ö, Dursun Ş, et al. Hiccup as the presenting symptom of ischemic stroke: supratentorial involvement and literature review. *Neurol Sci*. 2020;41(10):2821–2824. doi:10.1007/s10072-020-04484-y

5. Farmer AD, Al Omari M, Aziz Q. Gut–brain axis and neurogastroenterology: bridging the mind–body gap. *Nat Rev Gastroenterol Hepatol*. 2020;17(2):77–86. doi:10.1038/s41575-019-0221-9

6. Hindricks G, Potpara T, Dagres N, et al. 2020 ESC Guidelines for the diagnosis and management of atrial fibrillation. *Eur Heart J*. 2021;42(5):373–498. doi:10.1093/eurheartj/ehaa612

7. Friedman NL. Hiccups: a treatment review. *Pharmacotherapy*. 2018;38(2):217–226. doi:10.1002/phar.2074