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CASE REPORT **OPEN ACCESS**

ISCHEMIC STROKE PRESENTING AS ISOLATED DIZZINESS IN A PATIENT WITH TYPE 2 DIABETES MELLITUS AND CONGESTIVE HEART FAILURE: A CASE REPORT

Elsa Lase[1*], Rio Tasti Surpa Rahmat Bintan[2], Kamila Auliya[2]

***Correspondence**: laseelsa@gmail.com*
[1]Dr Midiyato Suratani Naval Hospital, Tanjungpinang, Indonesia
[2]Airlangga University Hospital, Surabaya, Indonesia

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ABSTRACT

Background: Ischemic stroke may manifest with a wide range of clinical symptoms, including atypical presentations such as dizziness, especially in patients with complex cardiovascular and metabolic risk profiles. This case report describes the clinical course and management of ischemic stroke in a patient with pre-existing cardiovascular disease and type 2 diabetes mellitus.
Case: A 61-year-old woman with a history of cardiovascular disease and type 2 diabetes mellitus presented with sudden disorientation and persistent dizziness. On the second day of hospitalization, she developed right-sided weakness, facial asymmetry, and slurred speech. A head CT scan revealed infarctions in the left occipital lobe and left corona radiata. Due to the unavailability of thrombolytic therapy at the local hospital, the patient was managed conservatively with 80 mg aspirin, 75 mg clopidogrel, citicoline, and insulin.
Discussion: Dizziness/vertigo is recognized but is often an overlooked symptom of ischemic stroke, particularly in posterior circulation strokes. Approximately 20% of ischemic strokes involve the vertebrobasilar system, where vertigo is a predominant symptom. Type 2 diabetes mellitus and cardiovascular comorbidities significantly increase the risk of ischemic events. Patients presenting with dizziness or vertigo are at higher risk of subsequent stroke or cardiovascular events compared to those without these symptoms.
Conclusion: This case underscores the importance of recognizing dizziness as a potentially subtle and early manifestation of ischemic stroke, especially in patients with complex cardiovascular and metabolic risk profiles, such as type 2 diabetes mellitus. Prompt evaluation and appropriate management are crucial for preventing progression and improving outcomes.

Keywords: cardiovascular disease, diabetes mellitus, dizziness, ischemic stroke, vertigo

Introduction

In some cases of stroke, patients may present with symptoms such as dizziness or vertigo.[1,2] In cerebrovascular disorders, dizziness, or vertigo is often accompanied by other neurological signs and symptoms, including facial drooping, speech difficulties, unilateral or generalized weakness, confusion, altered mental status, decreased level of consciousness, and nausea or vomiting.[3,4] Due to the differences in management and prognosis, it is essential to distinguish dizziness or vertigo caused by vascular disorders from those of non-vascular origin.

Stroke remains the second leading cause of death worldwide, following ischemic heart disease.[6,7] Misdiagnosis of acute stroke may result in high morbidity and mortality; among survivors, many experience reduced mobility and persistent neurological deficits. According to the Centers for Disease Control and Prevention (CDC), in the United States in 2022, stroke accounted for 1 in 6 deaths (17.5%) related to cardiovascular disease. More than

795,000 people in the United States experience a stroke annually, with approximately 87% classified as ischemic.[8,9]

A comprehensive diagnostic approach is critical in cases of acute ischemic stroke. Standard investigations include blood tests such as a complete blood count, blood glucose, and metabolic profile, along with electrocardiography (ECG) and brain imaging, particularly a computed tomography (CT) scan.[10] Type 2 diabetes mellitus is a well-established risk factor for stroke.[11,12] Individuals without type 2 diabetes have a lower risk of stroke compared to those with the condition.[13] Glycemic control is therefore vital for reducing both the risk and severity of an initial stroke in diabetic patients.[14] Congestive heart failure (CHF) is another significant risk factor for stroke. The reduced ability of the heart to pump blood effectively may lead to stasis, particularly in the left atrium, facilitating thrombus formation and subsequent embolic events. Impaired cardiac function in CHF also predisposes patients to atrial fibrillation, further increasing stroke risk.[15] Additionally, CHF is associated with endothelial dysfunction and a chronic inflammatory state, both of which contribute to a prothrombotic environment, therefore increasing the likelihood of stroke.[16]

Myocardial infarction (MI), commonly referred to as a heart attack, also markedly increases the risk of stroke.[17] Post-MI patients are more susceptible to clot formation within the cardiac chambers, particularly in areas with damaged myocardium. These clots may dislodge and travel to the cerebral circulation, resulting in an embolic stroke. Moreover, MI can induce arrhythmias such as atrial fibrillation, which promotes blood stasis and thrombus formation. The systemic inflammatory response and hypercoagulable state following MI further elevate stroke risk.[18] Traditional vascular risk factors such as dyslipidemia, diabetes mellitus, and hypertension are widely recognized. Additionally, lifestyle-related factors, including obesity, alcohol consumption, and smoking, also contribute significantly to stroke risk.[19]

Case Report

A 61-year-old female patient presented to the emergency department with a chief complaint of dizziness accompanied by shortness of breath that began approximately one hour before admission. The patient described the dizziness as a sensation of spinning and lightheadedness. She also experienced nausea without vomiting, a burning sensation in the epigastric area radiating to the left hemithorax, and fatigue. The symptoms occurred while the patient was having lunch. She denied any symptoms suggestive of neurological deficits, as well as urinary or bowel disturbances.

The patient had a known history of cardiovascular disease, having suffered a myocardial infarction and undergone percutaneous coronary intervention (PCI) in 2023. Since then, she has been diagnosed with CHF and typically sleeps with more than two pillows to alleviate orthopnea. She also had a history of type 2 diabetes mellitus for the past eight years. Her current medications included aspirin 80 mg, clopidogrel 75 mg, amlodipine 10 mg, spironolactone 25 mg, digoxin 0.25 mg, bisoprolol 5 mg, furosemide 40 mg, isosorbide dinitrate (ISDN) 5 mg, nitroglycerin 5 mg, candesartan 16 mg, glimepiride 4 mg, metformin 500 mg, atorvastatin 20 mg, and lansoprazole 30 mg. She had been on coronary medications for six years and diabetic medications for eight years.

On physical examination, the patient was fully conscious with a Glasgow Coma Scale score of 15. Her vital signs were as follows: blood pressure 131/81 mmHg, pulse rate 98 beats per minute with a strong and regular rhythm, respiratory rate 27 breaths per minute, body temperature 36 °C, and oxygen saturation of 93% on room air, improving to 98% with 3 liters per minute oxygen via nasal cannula. Neurological examination revealed no meningeal signs or cranial nerve abnormalities. However, there was mild bilateral lower limb weakness, while the upper limbs were normal. Physiological deep tendon reflexes were within physiological limits in all extremities. Sensation was intact, and no pathological reflexes were observed. Cardiac and pulmonary auscultation revealed normal heart and breath sounds, with no murmurs, gallops, rales, or wheezing. Abdominal, spinal, genital, and rectal examinations were unremarkable. Laboratory findings on the day of admission revealed a random blood glucose level of 216 mg/dL.

The patient was admitted under the care of a cardiologist, with input from an internist, due to her history of myocardial infarction, CHF, and type 2 diabetes mellitus. The primary concern at admission was her dyspnea and epigastric discomfort. The internist initiated insulin therapy to manage hyperglycemia. The mild lower limb weakness was initially suspected to be related to general frailty associated with her chronic medical conditions. Additional treatments included intravenous omeprazole 40 mg, ketorolac 30 mg, and diphenhydramine 10 mg, and 500 mL of normal saline for fluid maintenance. An ECG was performed, and the patient was admitted for observation and further evaluation.

On the second day of hospitalization, her fasting blood glucose was 146 mg/dL. The patient developed mild confusion, though her level of consciousness remained intact. There were no sensory disturbances.

She subsequently developed significant right-sided weakness in both upper and lower extremities, with muscle strength graded at 3/5 (mild bilateral lower limb weakness).[20]

This weakness extended to the right side of her face, resulting in facial asymmetry, especially noticeable when smiling or baring teeth, along with a diminished right nasolabial fold. Reflexes on the affected side were reduced, and pathological signs were positive for both Babinski and Chaddock reflexes on the right foot.

Additional neurological symptoms emerged, including dysarthria. Her speech became slow and effortful, although comprehension of spoken and written language remained intact. She did not experience difficulty with repetition. Ocular movements were preserved. In response to the new onset of hemiparesis, right facial weakness, and dysarthria, a neurologist was consulted. A head computed tomography scan was performed approximately 6 hours after the initial presentation of new onset symptoms to evaluate for potential cerebrovascular pathology.

Imaging revealed infarctions in the left occipital lobe and the left corona radiata (Figure 1). Despite the occipital infarction, the patient did not exhibit visual field deficits or loss of vision.

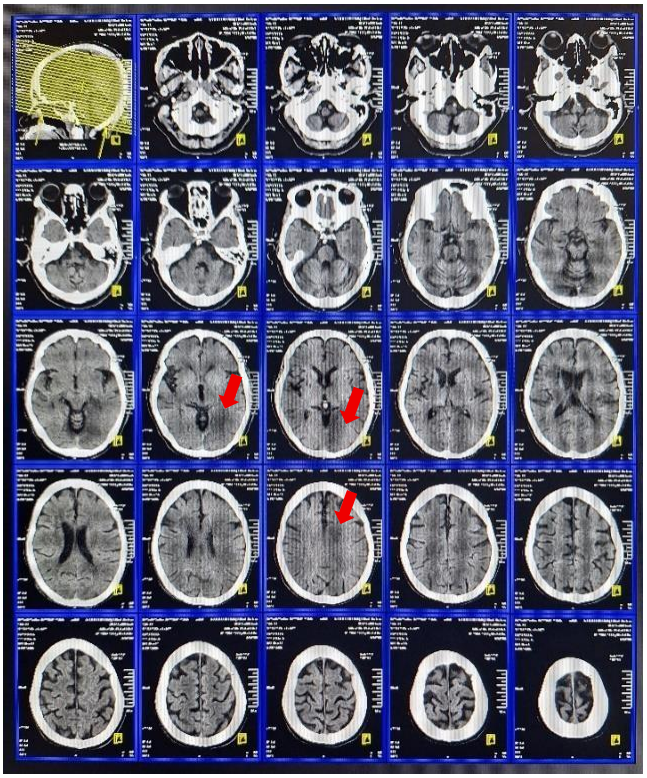

Figure 1. A head scan showed an infarction in the left occipital and left corona radiata region (red arrow)

The neurologist advised continuation of the patient's antiplatelet regimen, which included oral aspirin 80 mg and clopidogrel 75 mg once daily, along with her other routine medications. Citicoline 500 mg was prescribed three times daily to support neuroprotection. Due to the unavailability of thrombolytic therapy at the hospital, conservative management was implemented. The patient's vital signs were closely monitored, and staff were instructed to report any clinical deterioration.

Over the following 24 hours, the patient remained clinically stable, with no progression of neurological symptoms. She continued her regular medications and reported no new complaints. The dizziness improved, and the dyspnea was manageable with positional adjustments. This stable condition persisted through the fourth day of hospitalization.

By the fifth day, the patient demonstrated improvement in speech fluency, indicating partial recovery from dysarthria. However, the right-sided hemiparesis remained unchanged. Her vital signs remained stable. On the sixth day, the patient was discharged with a follow-up appointment at the neurology outpatient clinic. A post-discharge rehabilitation program was planned, and she was instructed to continue her daily antithrombotic medications.

Discussion

The infarction identified in this patient was primarily localized to the left occipital region. The occipital lobe is primarily responsible for visual processing, including perception of color, motion, and spatial relationships.[20] In addition to containing cortical regions related to sensory and language function at the subcortical level, the parietal lobe is a crossroad of white matter tracts related to motor, sensory, language, visuospatial, and visual function.[21] The temporal lobe is involved in auditory processing, memory formation, and aspects of language comprehension.[22]

Based on the Trial of ORG 10172 in Acute Stroke Treatment (TOAST) Classification system, it falls under the "stroke of undetermined etiology" criteria because of the multiple possible causes of stroke in these cases.[23] This patient's past medical history was significant for a MI one year prior and a subsequent diagnosis of CHF. Both conditions are well-established risk factors for ischemic stroke. Patients with heart failure have a two to five-fold increased risk of developing stroke.

The impaired cardiac function in CHF predisposes patients to atrial fibrillation and blood stasis, both of which facilitate thrombus formation and subsequent embolic events.[24-27] The principal concept of thrombus in cardioembolism events applies when all three components of Virchow's triad are fulfilled, namely hypercoagulation, endothelial activation, and

a low-flow state with risk of blood stasis.[28] In addition, the patient had a history of type 2 diabetes mellitus, another independent and potent risk factor for ischemic stroke. Chronic hyperglycemia contributes to atherosclerosis, endothelial damage, and small vessel disease, all of which elevate cerebrovascular risk.11 Increased arterial stiffness in diabetes exposes small cerebral vessels to abnormal pulsatile flow, contributing to the pathogenesis of cerebral small vessel disease (SVD).[29]

Diabetes and arterial hypertension are two major risk factors for multiple lacunar strokes. Research shows that diabetes is present in about 28% to 43% of patients with cerebral lacunar infarction. In one study, the prevalence of diabetes was found to be similar in patients with lacunar infarction and other types of strokes.[29]

The most common lacunar syndromes include: (1) Pure motor hemiparesis, which involves the posterior limb of the internal capsule, corona radiata, or ventral pons. This type presents with contralateral hemiparesis of the face, arm, and leg. Mild dysarthria may be present, accompanied by the absence of sensory symptoms. Cortical signs like aphasia, cognitive deficit, or visual symptoms are always absent. (2) Ataxic-hemiparesis involves the internal capsule, pons, or corona radiata. This type of lacunar stroke typically causes hemiparesis of the contralateral face and leg, as well as ataxia of the contralateral limb. Pressure, vision, hearing, and taste. (3) Dysarthria-clumsy hand, which involves the pons or internal capsule. Dysarthria-clumsy hand syndrome presents with dysarthria and difficulties pronouncing words due to weakness in the muscles of the voice box, including the tongue, larynx, and other facial muscles. Contralateral clumsiness of the upper extremity is present with preserved motor strength.[30]

Another population-based registry revealed that 90% of isolated posterior circulation transient ischemic attacks (TIAs), with half presenting with isolated vertigo symptoms, were not recognized at the initial medical contact. Overall, dizziness and vertigo are among the symptoms most strongly associated with missed stroke diagnoses.[31] Posterior circulation (PC) stroke refers to an infarction in central nervous system structures supplied by the vertebrobasilar arterial distribution. The distribution of the PC includes the medulla, pons, midbrain, cerebellum, thalamus, and components of the temporal and occipital lobes. PC stroke commonly presents with dizziness. Literature suggests patients cannot easily distinguish between dizziness (e.g., feeling off balance), vertigo (classically described as “room spinning”), and lightheadedness (feeling as if one may pass out).[2,3,5]

However, the Face, Arm, and Speech test (FAST) is not sensitive to posterior circulation ischaemia, and the National Institutes of Health Stroke Scale (NIHSS) does not include dizziness or vertigo as a manifestation of stroke or transient ischemic attack (TIA). A systematic review found that the head-impulse, nystagmus, and test of skew (HINTS) test appears reliable and effective in differentiating acute PC stroke from peripheral vestibular causes in symptomatic patients. The head-impulse component examines the connections between vestibular pathways and the brainstem. The presence of direction-changing nystagmus suggests damage to gaze circuits in the brainstem and cerebellum; skew deviation of the eyes indicates injury to the central otolithic connections to the brainstem.[33] A positive Head Impulse–Nystagmus–Test of Skew (HINTS) examination indicates a possible posterior circulation stroke in patients with acute vestibular syndrome when at least one of the following findings is present: a normal horizontal head impulse test, direction-changing gaze-evoked nystagmus, or ocular skew deviation. In contrast, a 'negative HINTS examination is suggestive of a peripheral etiology. There was a 15-fold increased risk of PC Stroke in patients with a positive HINTS test compared to those who were negative.[32,33]

Several studies have reported similar prevalence of typical cardiovascular risk factors (i.e., diabetes, hypertension, smoking) in those with PC strokes.[2] Moreover, patients who experience symptoms such as dizziness or vertigo have been reported to have a twofold higher risk of future stroke or cardiovascular events compared to individuals without these symptoms.[5] This association underscores the importance of thorough clinical evaluation in patients with comorbidities such as diabetes and cardiovascular disease.[10,12,13]

Transcranial Doppler (TCD) and carotid ultrasound (CU) are non-invasive ultrasonic detection techniques to diagnose and evaluate carotid artery disease in patients. TCD utilizes the weakest part of the human skull as the acoustic window for examination and employs the Doppler effect to investigate the hemodynamics of the cerebral low artery.[33] However, these investigations were not performed in this patient. The patient underwent a head CT scan approximately 6 hours after the initial presentation of new-onset symptoms. Non-contrast CT findings in ischemic stroke depend on the age of infarction: hyperacute (<12 hours), acute (12-24 hours), subacute (24 hours to 5 days), and old (within weeks after stroke). In the "hyperacute phase," the central role of non-contrast brain CT is to exclude intracranial hematoma. Sometimes an intra-arterial thrombus has high attenuation in non-contrast

computed tomography (NCCT) and can be detected. This phenomenon is known as the "hyperdense vessel sign." In "acute" infarction, NCCT can reveal a subtle loss of the gray-white matter interface (differentiation). In "subacute" infarction, NCCT shows vasogenic edema with mass effect and well-defined margins. In "old" findings, NCCT shows volume loss of brain parenchyma and hypoattenuation compatible with encephalomalacia.[34] The patient's CT scan revealed infarctions in the left occipital lobe and the left corona radiata. The involvement of the left corona radiata, which carries the corticospinal tract, directly explains the resulting right-sided hemiparesis. The corticospinal tract contains fibers from upper motor neurons that synapse on lower motor neurons. Although damage to both upper motor neuron (UMN) and lower motor neuron (LMN) results in muscle weakness, damage to upper motor neurons may result in increased reflexes, increased tone, positive Babinski, and spastic paresis. When the orolingual regions of the cortex are involved, the patient will present with dysarthria.[35]

The infarcted occipital lobe typically causes vision loss. Despite the occipital infarction, the patient did not exhibit visual field deficits or loss of vision. This finding may indicate that the infarct spared the primary visual cortex or the optic radiations, thus preserving visual function.[21] The persistence of dizziness without progression to more severe symptoms such as vertigo or visual field deficits supports the notion of a relatively localized infarction. The gradual resolution of dizziness during hospitalization suggests either partial recovery of the affected neural circuits or the successful activation of compensatory mechanisms from unaffected regions of the brain.[5]

Non-contrast CT is essential in the management of patients with ischemic stroke. In patients with a large infarction, thrombolysis is not helpful. Still, it can increase the risk of post-treatment intracranial hematoma, a severe complication with the risk of brain herniation and death.[36] Current international guidelines recommend that the optimal time for thrombolytic treatment of AIS is within 4.5 h of stroke onset to minimize risk of side effects such as hemorrhage.[37] Thrombolysis may be considered for a stroke patient experiencing dizziness if it's suspected of an acute ischemic stroke. However, presentations dominated by dizziness are frequently associated with delayed or missed stroke diagnosis. For this patient, it is not recommended to use IV thrombolysis, and dual antiplatelet therapy is given to the patient.

For patients with minor stroke or high-risk transient ischemic attack, dual antiplatelet therapy (DAPT) with clopidogrel and aspirin initiated within 24 hours after stroke onset significantly reduced the risk of recurrent stroke compared with aspirin alone. However, monotherapy with aspirin alone is still recommended by guidelines for patients with acute mild to moderate ischemic stroke who are at high risk of early neurologic deterioration (END), which is associated with poor prognosis. Patients with LAA (Large Artery Atherosclerosis) stroke benefited from DAPT within 48 hours of symptom onset, but patients with non-LAA stroke only within 24 hours.[38] In addition, Citicoline was prescribed for neuroprotection to inhibit neuronal death and halt or decelerate neuronal loss, thereby lowering mortality rates, decreasing disability, and improving the quality of life following an acute ischemic stroke.[39]

Conclusion

Dizziness may represent a subtle manifestation of ischemic stroke, particularly in patients with significant cardiovascular disease and type 2 diabetes mellitus. In this patient, dizziness initially perceived as a nonspecific complaint served as a subtle early indicator of an underlying cerebrovascular event. The progressive development of neurological symptoms, including motor and language deficits, underscores the importance of considering stroke in the differential diagnosis of patients presenting with unexplained balance disturbances, even in the absence of overt focal deficits.

The patient's medical history of myocardial infarction and CHF markedly increased her risk for cerebrovascular events. Despite adherence to antithrombotic therapy, these comorbidities contributed to a prothrombotic state, predisposing her to ischemic complications. The left-sided infarction was managed conservatively due to the unavailability of thrombolysis, with continuation of existing medications and the addition of citicoline as neuroprotective therapy. Comprehensive evaluation, including prompt neuroimaging (CT/MRI) and careful clinical assessment, remains important when managing high-risk patients with complex cardiovascular profiles who present with atypical or isolated symptoms such as dizziness.

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