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Atypical Presentation of Ischemic Stroke in A 64-Year-Old Female Patient with Multisystem Comorbidities: A Case Report

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ABSTRACT

Introduction: Cerebellar ischemic stroke is a rare subtype of acute stroke, accounting for approximately 3% of ischemic strokes, with an estimated 27,400 new cases annually. The initial clinical presentation often resembles peripheral vestibular disorders, such as vertigo, nausea, and vomiting, which may lead to delayed diagnosis and management. ***Case Presentation:*** Mrs. D, a 64-year-old Javanese woman, accompanied by her family to the emergency department with acute-onset vertigo since the morning of admission. She reported nausea, vomiting, left-sided weakness and paresthesia, and slurred speech. The patient had a history of type II diabetes mellitus, chronic kidney disease, congestive heart failure due to coronary artery disease, and hypertension. Neurological examination revealed dysarthria, hemiparesis flaccid, with muscle strength graded 3/5 in the upper extremity and 4/5 in the lower extremity. The HINTS examination (Head Impulse, Nystagmus, Test of Skew) indicated a central-type vestibular vertigo. A head CT scan demonstrated an ischemic infarct in the right cerebellar hemisphere. The patient was treated conservatively with antiplatelet therapy, strict glycemic control, and close fluid monitoring due to the risk of overhydration associated with cardiac and renal dysfunction. ***Discussion:*** Similarities with peripheral vertigo symptoms often cause cerebellar stroke misdiagnosis. Studies show 50% of cases involve nausea and vomiting, whereas 75% involve vertigo. Bedside tests, such as HINTS, are more sensitive than MRI in detecting acute cerebellar strokes. MRI is the imaging modality's gold standard, though the CT scan is more commonly used initially. ***Conclusion:*** A Multidisciplinary approach is crucial for patients with multiple comorbidities to optimize outcomes and prevent complications.

Keywords: cerebellar stroke; ischemic stroke; central vertigo; comorbidities; multidisciplinary care

INTRODUCTION

Cerebrovascular disease is a major cause of mortality and disability on a global scale. According to the findings of a survey conducted in 2010, the annual incidence of strokes among the population was 16.9 million [1]. Most of the cases are ischemic stroke, and around 3% of ischemic stroke cases occur in the cerebellum, with an estimated 27.400 new cases per annum in the United States of America. This condition can be caused by vascular flow occlusion or trauma to the three main arteries in the vertebrobasilar system: the posterior inferior cerebellar artery (PICA), the anterior inferior cerebellar artery (AICA), and the superior cerebellar artery (SCA) [2]. In addition, age, smoking, obesity, diabetes, hyperlipidemia, hypertension, atherosclerosis, arterial dissection, and arrhythmia have been identified as risk factors for cerebellar stroke [3]. The early symptoms of cerebellar stroke include dizziness, nystagmus, nausea, vomiting, slurred speech, coordination problems during walking, headache, and hearing disorder. The clinical manifestations associated with the location of the infarction include Wallenberg syndrome or lateral spinal cord syndrome associated with PICA, Foville syndrome associated with AICA, and Mill syndrome associated with SCA [2]. Nonspecific infarct presentation and overlapping with neurological symptoms, cardiovascular, gastrointestinal, and other systemic disorders can cause misdiagnosis that leads to morbidity and mortality. The most severe complication that can arise is obstructive hydrocephalus and herniation, which is associated with the obstruction of the cerebrospinal fluid (CSF) channel of the aqueduct of Sylvius. This obstruction can lead to edema due to ischemia and changes in tissue function, resulting in secondary cellular swelling [3].

CASE PRESENTATION

A 64-year-old Javanese woman, Muslim, married, and unemployed, was brought to the hospital by her family with a chief complaint of vertigo that began in the morning prior to hospital admission. The onset of symptoms was sudden. The patient reported that the vertigo occurred upon opening her eyes, and her condition did not improve after closing her eyes or changing her position. Additional symptoms included nausea and three episodes of vomiting. She also reported weakness on the left side of her body.

These symptoms had been present since day one prior to admission and had worsened that morning. Initially, the patient experienced weakness and tingling in the left arm and leg, but was still able to move them. However, progressive deterioration resulted in an inability to move the left upper and lower extremities. According to her family, the patient also exhibited slurred speech. She denied loss of consciousness, tinnitus, hearing loss, blurred vision, head trauma, and bowel or bladder disturbances.

Her past medical history included type II diabetes mellitus, chronic kidney disease (suspected prerenal acute-on-CKD secondary to diabetic kidney disease), congestive heart failure due to coronary artery disease, and hypertension. The patient routinely took aspirin 80 mg, gliclazide 80 mg, candesartan 16 mg, furosemide 40 mg, spironolactone 25 mg, atorvastatin 20 mg, and bisoprolol 2.5 mg.

Based on the vital-sign assessment, the patient had a weak general condition with compos mentis, blood pressure 134/67 mmHg, heart rate 72 beats/min, respiratory rate 18 breaths/min, temperature 36.3°C, and oxygen saturation of 99% on room air. General physical examination of the head, neck, ear–nose–throat (ENT), thorax, abdomen, and extremities showed no abnormalities. Neurological examination demonstrated dysarthria and left-sided flaccid hemiparesis, with a muscle strength of 3/5 in the upper extremity and 4/5 in the lower extremity. The Head Impulse, Nystagmus, and Test of Skew (HINTS) examination result in central-type vestibular vertigo. Coordination examination could not be performed.

The complete blood test showed a WBC level of 939,000/µL with neutrophil predominance, hemoglobin 8.5 g/dL, hematocrit 26.2%, and platelet count 300,000/µL. Electrolyte analysis revealed mild hyponatremia (133.3 mmol/L). Random blood glucose was 258 mg/dL. A non-contrast computed tomography (CT) scan of the head demonstrated an ischemic infarct in the right cerebellar hemisphere, as shown in Figure 1.

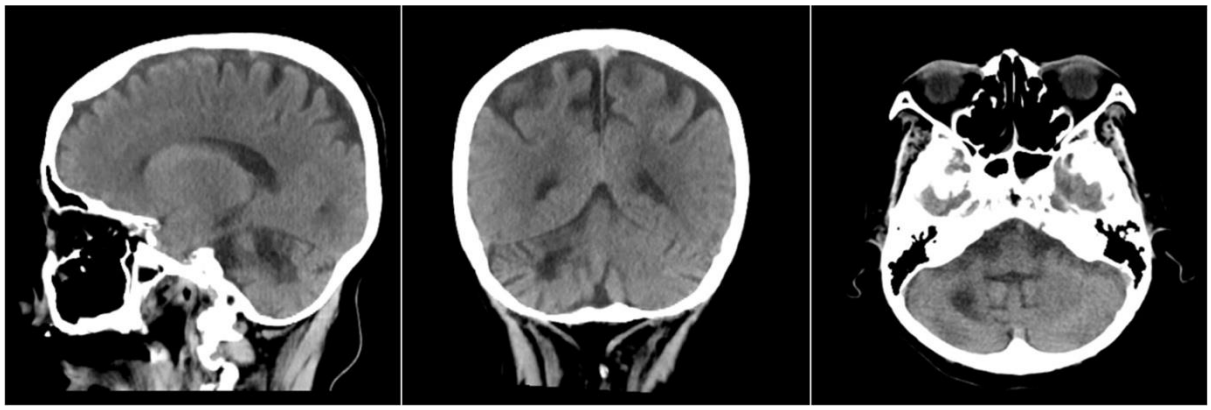

FIGURE 1: Non-Contrast Head CT Scan.

Initial management in the Emergency Department included intravenous (IV) pantoprazole 40 mg, ondansetron 8 mg IV, diphenhydramine 10 mg IV, and oral betahistine 12 mg. The patient was referred to the Neurology Department for inpatient care and further management. The clinical diagnosis was central vertigo, with a topographical diagnosis of posterior inferior cerebellar artery (PICA) involvement and an etiological diagnosis of right cerebellar infarction.

Non-pharmacological management included bed rest with the head elevated at 30° and supplemental oxygen at 4 L/min as needed. Pharmacological therapy consisted of IV normal saline at 20 drops/min, citicoline 500 mg IV twice daily, acetylsalicylic acid 100 mg orally once daily, betahistine mesylate 12 mg orally twice daily, bisoprolol 2.5 mg orally once daily, spironolactone 50 mg orally once daily, furosemide 40 mg orally once daily, atorvastatin 20 mg orally once daily, and gliclazide 80 mg orally once daily.

After seven days of hospitalization, the patient showed clinical improvement. Neurological examination revealed persistent dysarthria and left-sided flaccid hemiparesis with a muscle strength of grade 4/4, and her central-type vestibular vertigo had improved. She was discharged with recommendations for gradual mobilization, beginning with a semi-sitting position progressing to full sitting, as well as gradual oral intake from liquid to solid food.

DISCUSSION

Based on the case described above, the patient's primary complaint upon presentation to the hospital was vertigo. Vertigo is caused by disturbances of the vestibular system within the central nervous system (CNS). Central vertigo arises from disorder in the central nervous system, such as the brainstem, cerebellum, or cerebrum, and may be attributed to stroke, malignancy, or trauma. In contrast, peripheral vertigo results from disorders of the labyrinth or nervus vestibularis [4]. Misdiagnoses are reported to be three times more common in cases of posterior circulation stroke due to their atypical clinical presentation. A study demonstrated that posterior circulation infarction presenting with isolated vertigo is frequently misdiagnosed as peripheral vertigo, leading to delays in appropriate management [3].

Studies have shown that approximately 50% of cerebellar infarction cases present with nausea and vomiting, and around 75% experience dizziness [5]. Focal neurological deficits in stroke occur due to reduced cerebral blood flow secondary to occlusion by thrombus, embolus, or thromboembolism, which may lead to hypoxia or even anoxia in the affected vascular territories [4].

Magnetic Resonance Imaging (MRI) is the preferred imaging modality for diagnosing cerebellar infarction. However, a CT scan is more commonly used for the early evaluation of acute stroke. In certain cases of acute ischemic stroke, a CT scan may yield negative results, particularly in posterior fossa strokes with atypical manifestations, due to the relatively lower sensitivity of the CT scan compared to MRI. It is estimated that brain edema may develop around three days after stroke onset, and the infarct becomes isodense after approximately ten days [3]. The HINTS examination can be utilized to diagnose central vertigo and peripheral vertigo, which are associated with stroke and acute vestibular syndrome, respectively [4]. A study reported that among patients with acute vestibular syndrome and initially negative MRI findings, the HINTS examination demonstrated a sensitivity of 100% and a specificity of 96% for diagnosing stroke [3]. Additionally, in six other cases, a positive HINTS examination result increased the likelihood of posterior circulation stroke by fifteen-fold, with a sensitivity of 95.5% and specificity of 71.2%. The HINTS examination has therefore proven to be an effective tool for distinguishing posterior circulation stroke from peripheral causes of acute vestibular syndrome. A combined approach using clinical examination and appropriate imaging modalities remains an efficient strategy for the detection and management of cerebellar ischemic stroke [3].

The therapeutic management provided to the patient included antihypertensive, antiplatelet, and diabetes treatment. Citicoline is a cholinergic agent that influences the cellular oxygenation cycle and the formation of Adenosine Triphosphate (ATP). ATP functions to optimize the activity of the Sodium–Potassium (Na^+/K^+) ATPase, thereby maintaining cellular membrane integrity and preventing extracellular fluid accumulation that may lead to edema and lipid peroxidation. The administration of aspilet aims to reduce vascular occlusion within the cerebral arteries [4]. Aspilet therapy is given within the first 24–48 hours and may be continued for 21–90 days following the event for secondary stroke prevention [2]. The use of NaCl infusion and diuretics may assist in reducing cerebral edema [5]. A study conducted in Mexico reported that all of the patients who were admitted into their hospital due to cerebellar stroke had a history of hypertension, and 60% had diabetes mellitus. The high prevalence of hypertension is a significant factor in the formation of atherosclerotic thrombus. A multidisciplinary approach is essential during the acute phase to enable early detection of complications such as cerebellar edema, hydrocephalus, and consciousness impairment. Interprofessional rehabilitation and physiotherapy play a critical role in restoring balance, mobility, coordination, as well as speech and swallowing function. Simultaneous management of chronic comorbidities (hypertension, diabetes mellitus, chronic kidney disease, and coronary artery disease), patient education on secondary stroke prevention, and coordinated multidisciplinary care can reduce morbidity and mortality while improving functional recovery and long-term quality of life[6].

CONCLUSION

This case highlights the importance of combining clinical examination and imaging modality to detect cerebral ischemic stroke. The HINTS examination can be used in conjunction with a CT scan or MRI to prevent misdiagnosis of stroke and acute vestibular syndrome. A multidisciplinary approach is crucial for patients with multiple comorbidities to optimize outcomes and prevent complications.

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