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CASE SERIES

Diagnosis of Isolated Central Vertigo: Report for a Series Cases

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Abstract: Vertigo, including central and peripheral causes, is one of the common symptoms in patients who are admitted to neurological outpatient and emergency rooms. Despite the advancements in imaging techniques in recent years, central vertigo is difficult to identify and is often misdiagnosed in clinical practice. In this study, 4 patients were admitted to the hospital with complaints of dizziness or vertigo. Information about their symptoms, physical examinations and imaging were collected. Two patients were accurately diagnosed using diffusion-weighted imaging (DWI), a specific type of brain MRI. They received targeted treatments, which led to significant improvement, and were discharged nearly cured within a week. One patient with dorsolateral medullary infarction was misdiagnosed due to atypical symptoms, such as vertigo without the typical lateral medullary syndrome signs, and was discharged with a mild swallowing disorder after 2 weeks of treatment. One patient was diagnosed with both central and peripheral vertigo. It was observed that the symptoms of isolated vertigo caused by an acute lacunar infarction resolved more quickly than the accompanying physical symptoms. In summary, more attention should be paid to the diagnosis of isolated central vertigo, as early identification and intervention can improve a patient's prognosis and reduce medical expenses.

Keywords: cerebral infarction, isolated central vertigo, case series, central vestibular disorders, physical examination, brain MRI

Introduction

Vertigo, or dizziness, affects 20–30% of the general population and has been recognised as the primary complaint in approximately 3% of presentations to hospital emergency departments.[1,2] It is essential to determine whether the vertigo is centrally or peripherally sourced, as it is often misdiagnosed clinically. Many patients who report to the emergency department with acute vertigo or dizziness are diagnosed as having a benign peripheral vestibular illness without any evidence from neuroimaging.[3] Acute Vestibular Syndrome (AVS) is a clinical condition characterized by the acute onset of vertigo, nausea/vomiting, head-motion intolerance, and unsteady gait lasting days to weeks. Its significance lies in differentiating between central and peripheral causes during acute management. In the realm of benign peripheral disorders, three predominant conditions merit attention due to their frequent occurrence and diagnostic nuances. Benign Paroxysmal Positional Vertigo (BPPV) is characterized by brief episodes of vertigo related to head position changes, often linked to dislodged otoliths within the inner ear. Ménière's disease presents with episodic vertigo, hearing loss, tinnitus, and aural fullness, attributed to the abnormal fluid accumulation in the inner ear. Vestibular neuritis, presumed to be of viral origin, causes acute, persistent vertigo without auditory symptoms.[4]

Some of the instances, particularly vertebrobasilar acute ischemic stroke and vertebral artery dissection, are brought on by nonbenign central aetiologies. For central vertigo, especially acute lacunar infarcts occurring in the vestibular nucleus, choroid, parietal nucleus and their associated connected fibres, early recognition and diagnosis remain extremely

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difficult, although the imaging techniques have been greatly improved in recent years.[5,6] Timely and accurate diagnosis and treatment can shorten hospitalisation, improve patient prognosis, reduce disability and mortality rates, reduce social burden and save medical costs.

Nonbenign reasons have been frequently ignored when determining the aetiology of individuals who present with vertigo or dizziness, and symptoms have instead been assigned to benign causes.[7] Posterior circulation strokes, often implicated in central vertigo, predominantly affect the cerebellum and brainstem, including the medulla, pons, and midbrain. These regions are vital for balance and spatial orientation, and their ischemia can manifest as vertigo, underscoring the importance of precise neuroimaging in diagnosing vertigo's central causes. According to one study, 15–33% of posterior circulatory strokes diagnosed in emergency departments were actually severe peripheral vertigo.[8] If misdiagnosed, patients with cerebellar infarction have a significant risk of sequelae (approximately 40% fatality rate).[9] To lower morbidity and mortality, it is crucial to identify the central aetiologies in patients who report vertigo or dizziness.

Although diffusion-weighted imaging MRI is particularly crucial in identifying central causes of vertigo, it has limitations. Brain MRI results are generally negative until 72 hours after symptom onset, making it less useful for diagnosing central causes of vertigo after two weeks. Therefore, physical examination remains essential in the acute phase of vertigo, and brain MRI must generally be performed within a two-week interval. In acute vertigo evaluation, the presence of hearing loss and tinnitus, typically linked to peripheral disorders, warrants careful consideration. Their occurrence alongside vertigo, without the classic symptoms of vestibular neuritis, might indicate a central pathology, such as posterior circulatory strokes. This highlights the critical role of the HINTS+ examination, which is highly adept at distinguishing between central and peripheral vestibular lesions. Studies underscore the absence of cochlear symptoms in vestibular neuritis, reinforcing the need for thorough assessment.[10] Furthermore, limitations in hearing testing for acute vertigo should be considered, as advanced tests like brainstem audiometry are often inaccessible in acute healthcare settings. Additionally, understanding central vestibular disorders is essential, emphasizing the diagnostic value of the vestibulospinal reflex in identifying central etiologies.[11]

This study presents four clinical cases of acute infarction in patients who presented with dizziness or vertigo. These cases illustrate the challenges of accurately diagnosing central causes of vertigo in the emergency setting. This study highlights the importance of early and accurate diagnosis in improving patient outcomes. The study was conducted with the approval of the institutional ethics committee.

Case Report

Case One

A 65-year-old man presented to the emergency room with sudden onset of acute vertigo. The acute phase of vertigo lasted for approximately 48 hours, after which the patient continued to experience a persistent sensation of dizziness and lightheadedness, distinct from the initial spinning sensation. Laboratory testing revealed the following notable findings: gamma-glutamyl transferase (GGT) was 61.7 U/L, total protein (TP) was 64.1 g/L, elevated triglycerides (TG) was 2.48 mmol/L, and total cholesterol (CHO) was 6.32 mmol/L. Low-density lipoprotein cholesterol (LDL-C) was 4.49 mmol/L, and apolipoprotein B (Apo-B) was 1.10 g/L. A white blood cell (WBC) count was 18.1 G/L, with a high neutrophil count (NEUT) at 16.15 G/L and a neutrophil percentage (NEUT%) at 89.00%. An emergency biochemical analysis revealed glucose (GLU) at 9.92 mmol/L. This prolonged vertigo posed challenges in distinguishing between peripheral and central causes. In everyday practice, the duration of vertigo attacks is crucial for accurate diagnosis and management. Special care is required to exclude potential central causes such as posterior circulatory disorders, although this information can be difficult to obtain in the acute phase of vertigo. Upon examination, a nystagmus with the fast component beating to the left was observed, which initially suggested a right-sided peripheral vestibular lesion. However, the head impulse test revealed abnormal results on the right side. These findings can be misleading, as they may indicate peripheral vestibular dysfunction but must be interpreted with caution, particularly in the context of potential central causes. The patient's clinical presentation and imaging findings were crucial in correctly diagnosing the underlying cause. Yet, in this intricate ballet of eyes and balance, no skew deviation stepped forward, leaving vertical alignment unchallenged. He was standing unstably, falling to the right and had difficulty sitting still;

a closed-eye step test showed deflection forward and right.[12] The patient had suffered hypertension for 5 years but could not be controlled by medical regularly. Blood pressure measurements taken during the emergency examinations revealed elevated levels of 142/91 mmHg, further corroborating the diagnosis of uncontrolled hypertension. No other chronic illnesses or conditions were present. In the initial presentation of the patient, detailed neuroimaging was conducted to assess the underlying cause of the reported vertigo symptoms. Figure 1 illustrates the diffusion-weighted MRI findings, highlighting two nodular infarctions near the vestibular nucleus, which are consistent with the patient's clinical presentation of acute vertigo (Figure 1). MRI image showcases a well-defined lesion in the right vestibular nucleus. The patient did not reveal any additional neurological abnormalities and did not report experiencing hearing loss or tinnitus during the assessment. The treatment regimen included intravenous administration of butylphthalide and urinary kallidinogenase to enhance cerebral circulation and metabolism. Additionally, the patient was prescribed oral aspirin and clopidogrel to prevent platelet aggregation, and atorvastatin calcium to manage lipid levels and stabilize atherosclerotic plaques. Lansoprazole was administered orally for gastroprotection, along with other symptomatic treatments as needed. Following the improvement of his symptoms, the patient was discharged from the hospital.

Case Two

A 67-year-old man with a 10-year history of hypertension, who had been taking prolonged antihypertensive medication and had normal blood pressure control, had a sudden onset of vertigo with no apparent cause. He experienced severe vomiting, general weakness and unsteadiness in standing and walking, and his symptoms had lasted for 8 hours without relief before he came to the emergency room. He was unable to complete a standing and gait examination. The patient's eyes danced leftward with each gaze, painting a picture of fast left-beating nystagmus. However, the head impulse test, which is used to detect vestibular dysfunction, yielded normal results, indicating that there was no evidence of a significant vestibular deficit on the side tested. This patient, too, walked a straight line, with no skew deviation to tilt the tale. The patient initially presented with observable nystagmus. This nystagmus was characterized by rapid, involuntary eye movements, with the fast phase directed horizontally. Unfortunately, the complete HINTS (Head Impulse, Nystagmus, Test of Skew) examination, which is instrumental in distinguishing central from peripheral causes of vertigo, could not be fully administered due to the patient's condition at the time of assessment.[13] Blood pressure was measured and found to be within normal limits. A comprehensive suite of laboratory tests was performed, all of which returned results within normal parameters. On the second day, the patient's dizziness and vomiting improved significantly, and on physical examination, the finger-to-nose test was normal. On the third day, the patient's nystagmus disappeared, visual tracking was impaired, standing and walking were still unstable and acute *infarct foci* were seen on perfect cranial DWI. The patient's vestibular function report was negative for both spontaneous nystagmus and gaze-evoked nystagmus, and both the head impulse test and the test of skew deviation were normal. The patient's bilateral

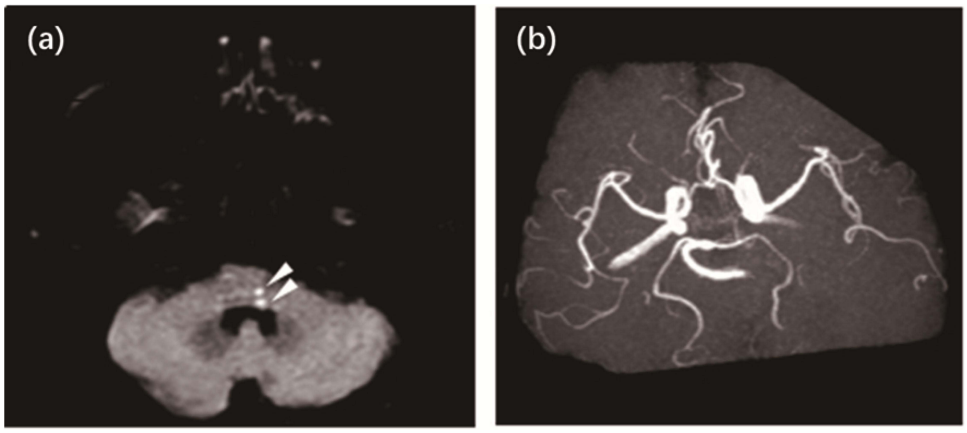

Figure 1 (**a**) The DWI show two nodular infarctions near the vestibular nucleus which cause patient dizziness and standing unstably but without nystagmus. (**b**) The MRA show the patient's cerebral artery overviews.

horizontal semicircular canals were assessed using the video Head Impulse Test (vHIT) with a high-speed camera system designed for evaluating horizontal canal function specifically. This approach was chosen based on the clinical presentation, which primarily suggested horizontal vestibular dysfunction. Given the nature of the symptoms and initial findings, focusing on the horizontal canals was deemed appropriate. The patient was also found to have type 2 diabetes mellitus, hyperlipidaemia and homocysteinemia during admission. He had a history of hypertension for >10 years and was on long-term antihypertensive medication with normal blood pressure control. The patient was treated with intravenous butylphthalide and edaravone to improve cerebral circulation, brain metabolism, and scavenge oxygen free radicals. Oral aspirin and clopidogrel were administered to prevent platelet aggregation, while atorvastatin calcium was prescribed for lipid regulation and plaque stabilization. For glycemic control, the patient received oral glimepiride and metformin. Additionally, esomeprazole was given orally for gastric protection, and symptomatic treatments were provided. Methylcobalamin was injected at acupuncture points, and the patient was also given oral vitamin B6 and folic acid to reduce homocysteine levels and support nerve function. The patient recovered and was discharged 1 week later. The image captures subtle changes in the left cerebellar region (Figure 2).

Case Three

A 43-year-old male patient presented with no history of hypertension or diabetes mellitus. There was no obvious cause for his symptoms, which were dizziness, unsteadiness, pain in the throat, difficulty in swallowing and choking and coughing with water; he had no consciousness disorder and no numbness or weakness of the limbs. He came to the emergency department 1 hour after the onset, where the emergency physician misdiagnosed the symptoms as acute pharyngitis and admitted him to the ENT department. However, it should be noted that dysphagia, a critical symptom often underestimated in everyday practice, is one of the "deadly D's" and can indicate a more severe neurological cause rather than an ENT issue. Approximately 10 hours after admission, the patient's symptoms worsened; the right side of his limbs experienced numbness, a physical examination showed signs of left Horner's syndrome, such as ptosis (drooping eyelid), miosis (constricted pupil), and anhidrosis (lack of sweating) on one side of the face, the right-hand finger-to-nose test was not stable and the patient's right side was hyperalgesic. The patient exhibited horizontal nystagmus that was perplexing due to its unclear directional pattern. The nystagmus was a mix of spontaneous and gaze-evoked types, with the fast component showing an inconsistent direction. This bidirectional nature made it challenging to determine a clear pattern. This ambiguity extended to the head impulse test, which was positive on the left side, indicating a vestibular abnormality. The presence of horizontal nystagmus and a positive head impulse test on the left side suggests a peripheral vestibular lesion. However, the overall clinical picture, including the ambiguity in nystagmus direction, necessitates further evaluation to rule out a central origin. No spontaneous nystagmus was observed at rest, suggesting a lack of

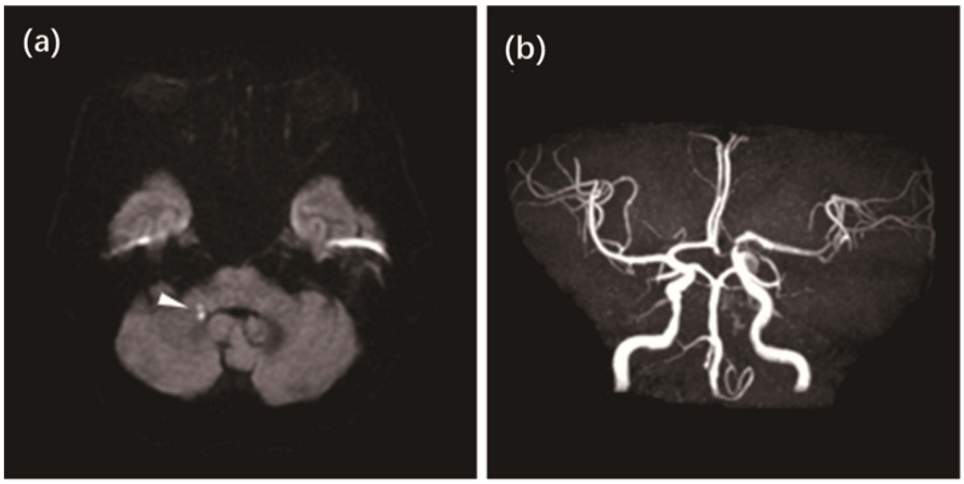

Figure 2 (**a**) A patient had a sudden onset of vertigo, severe vomiting, general weakness, unsteadiness in standing and walking, with left nystagmus, whose DWI showed acute infarctions near the vestibular nucleus. (**b**) MRA of the patient.

continuous vestibular irritation. However, the patient exhibited slight disorientation and imbalance during the Romberg test, pointing towards a possible central or peripheral vestibular deficit. Gait assessment also revealed a tendency to veer to one side, further supporting the presence of vestibular dysfunction. The neurological examination revealed no facial asymmetry or weakness, indicating intact cranial nerve function. Motor strength and coordination were normal across all limbs, with no signs of dysmetria or ataxia. Sensory pathways were intact, reflexes were symmetrical, and no pathological reflexes were elicited, suggesting no overt focal neurological deficits. The patient was discharged after 2 weeks of specialist treatment, which included targeted interventions to improve cerebral circulation and metabolism, along with swallowing rehabilitation in neurology. At the time of discharge, the numbness and ataxia of the right limb recovered, and most of the swallowing function was restored. Figure 3 illustrates the MRI findings for the patient, showcasing a distinct area of hyperintensity in the right cerebellar hemisphere, indicative of a cerebellar infarct. This image displayed an area of hyperintensity in the left medulla. This imaging evidence, combined with the patient's clinical presentation of dizziness, absence of significant neurological deficits, and vestibular symptoms, led to the diagnosis of cerebellar stroke. In summary, vestibular examinations played a crucial role in the final diagnosis of this case. The combination of the vHIT, nystagmus evaluation, and neuroimaging helped distinguish between peripheral and central causes of vertigo, ultimately leading to the identification of a cerebellar infarct. These findings emphasized the importance of comprehensive vestibular assessment in diagnosing complex vertigo cases, particularly when symptoms and imaging results may initially appear contradictory.

Case Four

A 65-year-old man was admitted to the hospital for sudden dizziness and vomiting for 4 hours, having previously been in good health. He had dizziness with no obvious cause, a sense of rotation, nausea and vomiting of gastric contents, accompanied by generalised weakness. In the initial description, the patient's symptoms were broadly referred to as "dizziness", a term encompassing various types of equilibrium disturbances. Upon closer examination, the patient specifically reported a "sense of rotation", distinguishing the symptom as vertigo—characterized by the sensation of spinning or moving surroundings. Upon reviewing the patient's medical history in light of the current presentation, no aberrations or exacerbations related to previously diagnosed illnesses were identified. The dizziness was aggravated by

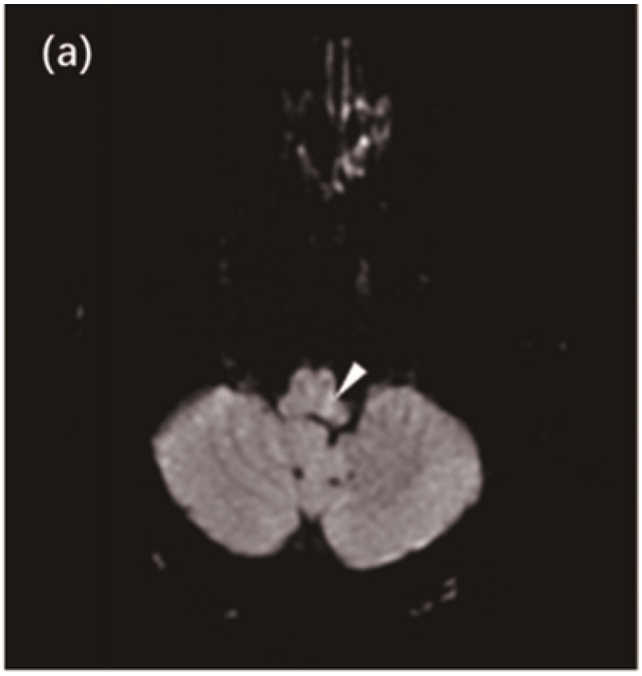

Figure 3 A patient with dizziness was misdiagnosed as acute pharyngitis and admitted to the ENT department, about 10 hours after admission, the patient's symptoms worsened, and the DWI was showed acute infarction on medulla.

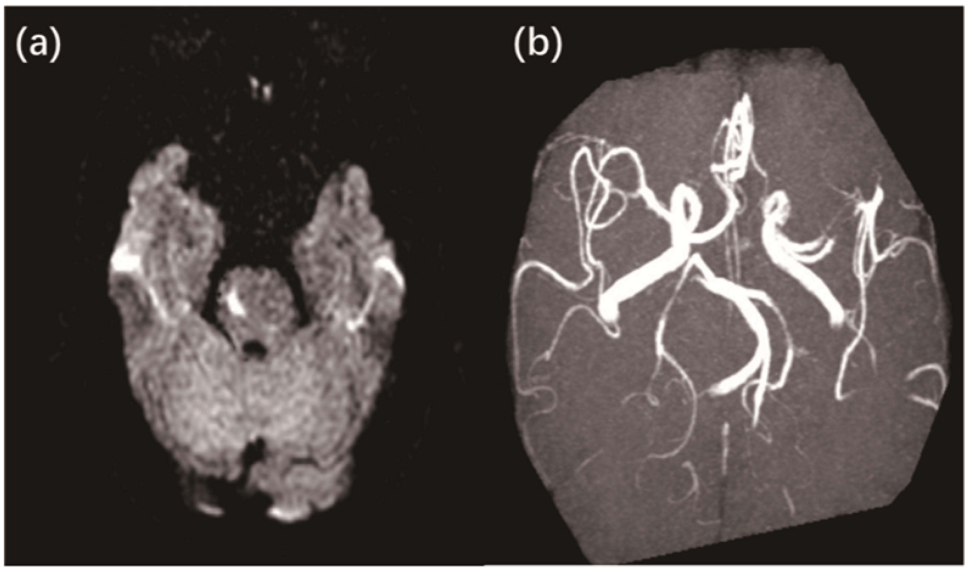

Figure 4 (**a**) A patient with sudden dizziness and vomiting for 4 hours was admitted to the ENT department. The patient's dizziness and vomiting improved the next day, and he walked steadily, but he still had persistent externally down-beating nystagmus on the right side. (**b**) MRA of the patient.

a change in body position. He had no headache, tinnitus, hearing loss or speech disorder; there was no improvement in the symptoms after the emergency clinic, and he was thus admitted to the department of medicine. The patient exhibited vertical nystagmus, which suggested a potential involvement of the central brainstem or vestibulocerebellum regions. The head impulse test showed no corrective saccades to either side, which aligns with the observed vertical nystagmus and supports a central aetiology of the vertigo. The test of skew deviation was negative in this patient, which, while not ruling out central vertigo, does not provide additional support for a central origin. However, given the vertical nystagmus and negative head impulse test, the overall clinical picture suggests a central vestibular cause. The patient's admission symptoms included dizziness, a sense of rotation related to a change of position, nausea, vomiting and generalised fatigue; he had no speech disorders, and his appetite, sleep patterns and bowel movements were normal. An examination showed that both pupils were round and equal in size with a diameter of approximately 3 mm, light reflex existed, the double eyeball activity was not limited, the right side of the externally rotated nystagmus was persistent, bilateral nasolabial folds were symmetrical, the tongue was centred, the extremities had normal muscle tone and the limb muscle strength was level 5. Moreover, the bilateral tendon reflexes were symmetrical and present, the varus test was normal, and there was symmetry of palpation sensation in limbs and neck tenderness. The tongue was dark red with petechiae, the tongue coating was white and greasy and the pulse was astringent. The patient was admitted to the hospital to check for hypertension and type 2 diabetes mellitus, and was given aspirin, clopidogrel, butylphthalide, edaravone and symptomatic treatment; the patient's dizziness and vomiting improved the next day and he walked steadily, but he still had persistent externally down-beating nystagmus on the right side. The MRI image revealed structural lesions that are consistent with central pathology, which was associated with the observed down-beating nystagmus (Figure 4).

Discussion

Vertigo is a common clinical manifestation of posterior circulation ischemia and it is the only symptom in most cases. It includes posterior circulation infarction and posterior circulation transient ischemic attack. Posterior circulation ischemic stroke accounts for 20–25% of all ischemic strokes and is an important cause of disability and death in patients.[14] Posterior circulation ischemia with isolated vertigo is often misdiagnosed and missed due to the lack of specific neurological symptoms, signs and auxiliary examination methods. Studies[15] have found that 3.7% of the patients with isolated vertigo admitted to the emergency department had posterior circulation acute cerebral infarction with lesions identified by DWI.

In terms of diagnostic ideas, first, it is relatively simple for neurologists to diagnose symptoms of dizziness or vertigo combined with related neurological localisation signs as cerebral infarction. However, for infarction or transient ischaemic attacks (TIAs) similar to vestibular neuronitis caused by the vestibular nucleus and the corresponding associated fibres, the differentiation is relatively difficult and requires a certain degree of clinical experience and professional knowledge, as well as a detailed physical examination at the time of consultation. For such patients, the receiving physician needs to be alert to two types of patients: those who do not fully conform to the classical symptoms of vestibular neuronitis and those with typical vestibular neuronitis symptoms with atypical neurological signs and at high risk of cerebrovascular disease. In the context of vestibular neuritis, it is crucial to consider cochlear symptoms such as hearing loss and tinnitus. These symptoms are especially significant if they are acute or if there is a sudden worsening of pre-existing chronic hearing loss or tinnitus. Given that audiometric examinations are often unavailable in acute healthcare settings, the importance of a thorough neurological examination and timely imaging is emphasized to aid in accurate diagnosis and differential diagnosis. These symptoms play a crucial role in the differential diagnosis, as their presence or absence can help distinguish between vestibular neuritis, which typically does not involve cochlear symptoms, and other vestibular disorders where these symptoms are prominent.[16] In patients with acute cerebral infarction, refinement of DWI can further clarify the diagnosis; however, the diagnosis of posterior circulation TIA is not clearly determined by cranial DWI. It can, nonetheless, be based on patients whose symptoms are consistent with isolated vertigo at the time of reception and whose symptoms disappear at 24 hours or remain with mild head drowsiness, combined with the patient's high-risk factors for cerebrovascular disease, and this is recommended for the prevention of TIA.

In everyday clinical practice, the overuse of brain CT scans for acute vertigo is a notable concern. Although CT scans are readily available and quick, they often lack the sensitivity required to detect central causes of vertigo, such as brainstem or cerebellar strokes. This reliance on CT not only exposes patients to unnecessary radiation but also increases healthcare costs without improving outcomes. MRI, particularly diffusion-weighted imaging, offers a more effective alternative for identifying central pathologies. Addressing this overuse requires emphasizing the value of clinical evaluation and the judicious application of imaging techniques in vertigo diagnosis.[17]

One of the challenges with utilizing brain MRI in the acute assessment of vertigo is its timing sensitivity. Specifically, brain MRI may not always show positive results immediately; in some cases, it can take up to 72 hours for ischemic changes, particularly those related to small infarcts in the posterior circulation, to become apparent on imaging. This delay can pose diagnostic challenges, especially in the early management of patients presenting with acute vertigo, where timely intervention is crucial.[18]

The significance of the HINTS (Head Impulse, Nystagmus, Test of Skew) examination cannot be overstated in the evaluation of patients presenting with acute vertigo. This diagnostic tool is invaluable for distinguishing between central and peripheral causes of vertigo, thereby guiding clinicians towards the most appropriate management and intervention strategies. Highlighting the HINTS examination underscores its critical role in the accurate and timely diagnosis of vestibular disorders.

Clarifying the characteristics of nystagmus observed in patients is essential for accurate diagnosis. Specifically, while detailing the direction of the fast component of nystagmus, such as noting that it beats to the left, it is important to recognize that this can indicate a peripheral lesion if the issue is in the left inner ear. However, nystagmus characteristics, such as vertical eye movements or bidirectional nystagmus, should also be considered. It is important to note that in many cases, distinguishing between central and peripheral vertigo based solely on nystagmus is challenging. Comprehensive evaluation, including clinical presentation and additional diagnostic tests, is essential for accurate differentiation. This level of detail enhances the diagnostic accuracy and is integral to the comprehensive evaluation of patients with vertigo.

Beyond risk factors and neurological symptoms, which are pivotal in distinguishing vestibular neuritis from posterior circulation strokes, the significance of conducting additional vestibular tests must be emphasized. These tests, including videonystagmography, vestibular evoked myogenic potentials, caloric testing, and vHIT, are crucial for the objective assessment of vestibular function. vHIT, in particular, is particularly valuable in acute vertigo cases, as demonstrated in one of our case presentations. Moreover, the foundational role of a thorough physical examination cannot be overstated; it remains a critical component of the initial assessment, guiding subsequent diagnostic and therapeutic decisions in the management of vertigo.

Right-sided vestibular neuritis is characterized by a left-beating fast component of the nystagmus, leaning to the right on standing, and a rightward deviation on the closed-eye step test. In contrast, a left-sided dorsal medial brainstem infarction also presents with a left-beating nystagmus but with persistent characteristics, leaning to the right on standing and showing a leftward deviation on the closed-eye step test. The head impulse test is crucial in distinguishing between these conditions, as it provides essential information about the presence and location of vestibular dysfunction. In conclusion, to distinguish central vertigo from peripheral vertigo, central lesions can be identified by symptoms that cannot be explained by vestibular neuronitis, in addition to risk factors and limb ataxia problems. This is of great significance for the timely use of thrombolytic drugs or antiplatelet and other drugs to prevent progression and improve the prognosis in middle-aged and elderly patients with initial stroke detection.

In fact, Lateral body movements may result from disruption of the output of the fastigial nucleus to the vestibulospinal and reticulospinal tracts.[19,20] Likewise, Subjective visual vertical contralateral tilt and ocular tilt responses may result from increased tonic resting activity in the ipsilateral vestibular nuclear complex.[21] Since the clinical symptoms (dizziness, vertigo, balance disturbance, etc) caused by different vestibular lesions are broadly similar, the diagnosis often needs to rely on other accompanying symptoms of the involvement of adjacent structures of the vestibular system and a detailed neurological examination. This can help in differential diagnoses.[22]

Vertigo may be the only manifestation of posterior circulation ischemia or infarction, which may develop into malignant vertigo and may even endanger the patient's life.[23] This study discussed the clinical manifestations, diagnostic ideas and differential diagnosis of isolated vertigo, reminding us to pay close attention to it. It provides valuable suggestions for the timely and accurate diagnosis and treatment of vertigo, improving the prognosis of patients, and reducing the morbidity and mortality rates.

Data Sharing Statement

All data generated or analyzed during this study are included in this published article.

Ethical Approval and Consent to Participate

Research experiments conducted in this article with humans were approved by the Ethics Committee of The First Affiliated Hospital of Hunan University of Chinese Medicine (HN-LL-KYSB-201827), following all guidelines, regulations, legal, and ethical standards as required for humans. Written informed consent was obtained from all participants.

Consent for Publication

Written informed consent was obtained from all the patients for publication of this case report.

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Disclosure

All of the authors had no any personal, financial, commercial, or academic conflicts of interest separately.

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